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CDFD

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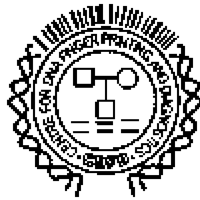

डी एन ए फिंगरप्रिंटिंग एवं निदान केन्द्र
उप्पल, हैदराबाद - 500 001
Centre for DNA Fingerprinting and Diagnostics
Uppal, Hyderabad - 500 001

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अधिदेश
Mandate

अधिदेश

सीडीएफडी सोसाइटी के संगम ज्ञापन तथा नियम एवं विनियमों में बताए गए अनुसार डीएनए फिंगरप्रिंटिंग एवं निदान केंद्र की स्थापना जिन उद्देश्यों के लिए हुई वे निम्न प्रकार हैं :

i. पितृत्व विवाद, आप्रवास और अस्पतालों में नवजात शिशुओं की अदला-बदली जैसे मामलों में निजी पक्षों सहित विविध अभिकरणों के लिए पर्याप्त अदायगी पर डीएनए प्रोफाइलिंग और उससे संबंधित विश्लेषण का वैज्ञानिक अनुसंधान करना।

ii. अपराध अन्वेषण अभिकरणों को डीएनए फिंगरप्रिंटिंग और उससे संबंधित विश्लेषण तथा सुविधाएं प्रदान करना।

iii. अपराध अन्वेषण और परिवार मामलों में डीएनए प्रोफाइल विश्लेषण और उससे संबंधित तकनीकों के साक्ष्य संबंधी मूल्य को समझने में पुलिस कर्मियों, न्यायिक वैज्ञानिकों, वकीलों तथा न्यायपालिका की सहायता करना।

iv. आनुवंशिक अव्यवस्थाओं को संसूचित करने हेतु डीएनए नैदानिक विधियां सिद्ध करना और इस प्रकार के संसूचन के लिए संपरीक्षाएं विकसित करना।

v. पादप और जंतु कोशिका माल, कोशिका लाइनों के प्रमाणीकरण के लिए डीएनए फिंगरप्रिंटिंग तकनीकों का उपयोग करना और ऐसे प्रयोजनों के लिए आवश्यकतानुसार नई संपरीक्षाएं विकसित करना।

vi. डीएनए फिंगरप्रिंटिंग तकनीकों पर प्रशिक्षण प्रदान करना।

vii. मूलभूत, अनुप्रयुक्त अनुसंधान एवं विकास कार्य करना।

viii. देश में चिकित्सा संस्थाओं, जन-स्वास्थ्य अभिकरणों और उद्योग को परामर्शी सेवाएं प्रदान करना।

ix. केंद्र के उद्देश्यों से संगत क्षेत्रों में विदेशी अनुसंधान संस्थाओं एवं प्रयोगशालाओं और अन्य अंतरराष्ट्रीय संगठनों के साथ सहयोग करना।

x. अनुसंधान छात्रों को स्नातकोत्तर उपाधियों के लिए पंजीकृत कर सकने के प्रयोजन हेतु उच्चतर अधिगम के मान्यता प्राप्त विश्वविद्यालयों एवं संस्थाओं के साथ संबंध स्थापित करना।

xi. भारत सरकार, राज्य सरकारों, देश में स्थित पूर्व संस्थाओं / न्यासों, व्यक्तियों और अन्य गतिविधियों के लिए अंतरराष्ट्रीय संगठनों सहित विदेशी स्रोतों से आर्थिक सहायता प्राप्त करना।

xii. केंद्र सरकार के पूर्व अनुमोदन से प्रशिक्षण कार्यक्रमों, वैज्ञानिक अनुसंधान और अन्य गतिविधियों के लिए अंतरराष्ट्रीय संगठनों सहित विदेशी स्रोतों से आर्थिक सहायता प्राप्त करना।

xiii. केंद्र की गतिविधियों को चलाने के लिए जैसा आवश्यक या सुविधाजनक हो, कोई भी संपत्ति चल या अचल या भवनों एवं निर्माणों को निर्मित करने, सुधार करने, परिवर्तित करने, गिरा देने या मरम्मत करने हेतु उपहार, क्रय, विनियम, पट्टा, भाडे पर लेने द्वारा या अन्यथा किसी भी तरह अर्जित करना।

xiv. केंद्र के प्रयोजन हेतु, भारत सरकार और अन्य प्रोनोटों, विनियम पत्रों या अन्य परक्राम्य लिखतों को आहरित करना और स्वीकार करना, तैयार करना और पृष्ठांकित करना, बट्टा करना और परक्रामण करना।

xv. केंद्र को सौंपी गई निधियों या धन को निवेश करने के लिए, ऐसी प्रतिभूतियों को खोलने या ऐसे तरीके से, जो कि समय-समय पर शासी परिषद द्वारा निर्धारित किए जाते हैं, इस प्रकार के निवेश को विक्रय या पक्षांतरण करना।

xvi. उक्त सभी उद्देश्यों या उनमें से किसी उद्देश्य की प्राप्ति के लिए सभी ऐसे अन्य विधिसम्मत कार्य, जैसा आवश्यक, प्रासंगिक या सहायक हो, करना।

xvii. केंद्र के उद्देश्यों को वास्तविक बनाने के लिए प्रोफेसरों, अन्य संकाय पदों, अभ्यागत अध्येतावृत्तियों सहित अध्येतावृत्तियों, अनुसंधान एवं संवर्ग पदों, छात्रवृत्तियों आदि को संस्थापित करना।

xviii. केंद्र के वैज्ञानिक एवं प्रौद्योगिकीय कार्य के लिए प्रयोगशाालाओं, कार्यशालाओं, भंडार, पुस्तकालय, कार्यालय और अन्य सुविधाओं को स्थापित करना।

xix. तकनीकी जानकारी को उद्यमकर्ताओं और उद्योगों से प्राप्त या उनको अंतरण करना, और

xx. पेटेंटों, डिजाइनों एवं तकनीकी जानकारी जो कि केंद्र द्वारा विकसित की गई हो, को पंजीकृत करना और केंद्र के हित में ऐसे पेटेंटों / डिजाइनों / तकनीकी जानकारी के किसी भाग को अंतरण करना।

MANDATE

The objectives for which the Centre for DNA Fingerprinting and Diagnostics (CDFD) was established, as enumerated in Memorandum of Association and Rules and Regulations of CDFD Society, are as follows:

i. To carry out scientific research pertaining to DNA profiling and related analysis in civil cases like paternity disputes, immigration, and exchange of newborns in hospitals, for various agencies including private parties, on appropriate payment;

ii. To provide DNA fingerprinting and related analysis and facilities to crime investigation agencies;

iii. To assist police personnel, forensic scientists, lawyers and the judiciary in understanding the evidential value of the DNA profile analysis and related techniques in crime investigation and family matters;

iv. To establish DNA diagnostic methods for detecting genetic disorders and to develop probes for such detection;

v. To use DNA fingerprinting techniques for the authentication of plant and animal cell material, cell lines and to develop new probes where necessary for such purposes;

vi. To provide training in DNA fingerprinting techniques;

vii. To undertake basic, applied and developmental R & D work;

viii. To provide consultancy services to medical institutions, public health agencies and industry in the country;

ix. To collaborate with foreign research institutions and laboratories and other international organizations in fields relevant to the objectives of the Centre;

x. To establish affiliation with recognized universities and institutions of higher learning for the purpose of enabling research scholars to register for post-graduate degrees;

xi. To receive grants, donations and contributions in cash or in other forms from the Government of India, State Governments, Charitable Institutions/Trusts, individuals and industry within the country;

xii. To receive, with the prior approval of the Central Government, monetary assistance from foreign sources including international organizations for training programmes, scientific research and other activities;

xiii. To acquire by gift, purchase, exchange, lease, hire or otherwise howsoever, any property movable or immovable or to construct, improve, alter, demolish or repair buildings and structures as may be necessary or convenient for carrying on the activities of the Centre;

xiv. For the purpose of the Centre, to draw and accept, make and endorse, discount and negotiate Government of India and other Promissory Notes, Bills of Exchange, Cheques or other Negotiable Instruments;

निदेशक का संदेश
From the Director's Desk

निदेशक का संदेश

मुझे वर्ष 2016 - 17 के लिए डीएनए फिंगर प्रिंटिंग एवं निदान केंद्र (सीडीएफडी) का वार्षिक प्रतिवेदन प्रस्तुत करते हुए प्रसन्नता है। यह बायोटेक्नोलॉजी विभाग, भारत सरकार के तहत एक स्वायत्त संस्थान है। संस्थान की प्रमुख गतिविधियां इस प्रकार हैं : 1. मानव और पादप डीएनए फिंगरप्रिंटिंग और नैदानिकी के क्षेत्रों में आनुवंशिक विकारों के लिए उच्च गुणवत्ता की सेवाएं प्रदान करना एवं 2. आधुनिक जीव विज्ञान के विभिन्न क्षेत्रों में बुनियादी अनुसंधान करना। वर्ष 2016 - 17 के दौरान केंद्र की कुछ प्रमुख उपलब्धियां और अनुसंधान के निष्कर्ष नीचे दिए गए हैं, जिनके विवरण प्रत्येक प्रयोगशाला के तहत रिपोर्ट में प्रदान किए गए हैं।

2016-17 की अवधि के दौरान डीएनए फिंगरप्रिंटिंग सेवा प्रयोगशाला में लगभग 140 मामले प्राप्त किए गए, जिन्हें न्यायपालिका और कानून प्रवर्तन एजेंसियों द्वारा अग्रेषित किया गया था और डीएनए जांच करने वालों ने पूरे देश की कानूनी अदालतों में अपनी रिपोर्ट से बचाव किए हैं।

नैदानिकी प्रभाग द्वारा विभिन्न आनुवंशिक रोगों के लिए लगभग 5000 रोगियों को आनुवंशिक सेवाएं प्रदान की गई हैं। केंद्र ने निजाम चिकित्सा विज्ञान संस्थान, हैदराबाद के चिकित्सा आनुवंशिकी विभाग के सहयोग से आनुवंशिक नैदानिक सेवाएं प्रदान की है और यह चिकित्सा आनुवंशिकी में डीएनबी कार्यक्रम चलाता है।

बासमती अपमिश्रण परीक्षण में जटिलताओं को विचार में लेकर पादप डीएनए फिंगरप्रिंटिंग प्रभाग द्वारा मार्करों की संख्या बढ़ाकर प्रोटोकोल में और अधिक सुधार लाने के प्रयास किए जा रहे हैं।

कोशिका चक्र नियमन प्रयोगशाला द्वारा आरबीपी 2 की प्रक्रिया को समझा गया है कि यह किस प्रकार एच 3 के 4 डिमेथिलेशन में प्रोटीन पी 130 का चयन करता है और ई 2 एफ प्रतिक्रिया शील जीनों का जीन रिप्रेशन होता है। पुन:, कोशिका विभाजन में एमएलएल एच 3 के 4 हिस्टोन मेथिल ट्रांसफरेज की भूमिका को समझने के लिए उन्होंने दर्शाया है कि एम एल एल/डब्ल्यूडीआर 5 कॉम्प्लेक्स द्वारा तर्कु निर्माण और गुणसूत्र का कंग्रेशन नियमित होता है।

आण्विक ओंकोलॉजी प्रयोगशाला ने स्वैमस कार्सिनोमा के लिए उत्पिरिवर्ती पी 53 के नए संगत अनुलेखन लक्ष्यों की पहचान की है और इनका सत्यापन किया है। इनके

कार्य में डब्ल्यूएनटी- लाशय के कैंसर में कैल्शियम आयन/एनएफएटी सिग्नलिंग का सुझाव मिलता है।

कोशिका सिग्नलिंग प्रयोगशाला द्वारा प्रदर्शित किया गया है कि आईपी 6 के 1 द्वारा कोशिका सतह के अतिरिक्त कोशिकीय मेट्रिक्स का नियमन इस प्रकार होता है आईपी 6 के 1 में कैंसर कोशिका की कमी का भेदन घट जाता है और आईपी 6 के 1 की कमी वाले चूहों में भेदक कार्सिनोमा के विकास का प्रतिरोध होता है। इस समूह द्वारा यह भी देखा गया है कि आई पी 6 के 1 नॉक आउट चूहों में लंबे स्पर्मेटिड में डीएनए का संघनन विफल रहता है और सोमेटिक हिस्टोइन की उपस्थिति प्रदर्शित होती है। वर्तमान में वे इन विविध कोशिकीय और शरीर क्रियात्मक कार्यों में आईपी 6 के 1 और आईपी 7 की भूमिका की विस्तृत आण्विक समझ की दिशा में कार्यरत हैं।

क्रोमेटिन जीव विज्ञान और एपिजेनेटिक्स प्रयोगशाला द्वारा खोजा गया है कि एस. पोम्बे के सिरटुइन एचएसटी 4 का नियमन यूबिक्विटिन लाइगेज द्वारा डीएनए क्षति की प्रतिक्रिया स्वरूप एससीएफ माध्यित प्रोटियोलाइसिस में होता है। यह प्रयोगशाला वर्तमान में डीएनए क्षति पर एचएसटी 4 के विखण्डन के सिग्नलिंग में जांच बिंदु काइनेस की भूमिका का अध्ययन और इस विखण्डन के महत्व का निर्धारण कर रही है।

अभिकलनात्मक जीव विज्ञान प्रयोगशाला में क्रम के बिगड़ने वाले हिस्सों को व्यावस्थित करने के लिए एक नए प्रतिस्थापन स्कोरिंग मेट्रिक्स की गणना की गई है और इसके निष्पादन का मूल्यांकन किया जा रहा है। एक नई एसवीएम आधारित विधि का विकास किया गया और प्रोटीनों के बिगडे हुए हिस्सों में मिससेंस उत्परिवर्तनों के कार्यात्मक प्रभाव का अनुमान लगाने के लिए इसे परखा गया। डोमेन पर एमडी सिमुलेशन के साथ बिगड़े हुए क्रम

के हिस्सों सहित आगे बढ़ने वाले रोग में उत्परिवर्तन होने से पता चला कि इस हिस्से में उत्परिवर्तन के कारण इनकी आंतरिक अभिविन्यास विषम जनकता नष्ट हो गई है।

कवक रोगाणु जनन प्रयोगशाला द्वारा पहली बार यह प्रदर्शित किया गया है कि रोग जनक कवक कैंडिडा ग्लाब्रेडा में फॉस्फोकइनोसिटाइड 3 - काइनेस (पी आई 3 के) कोशिका के आयरन होमियोस्टेसिस और रेट्रोग्रेड ट्रैफिकिंग का रखरखाव उच्च आयरन पर्यावरण परिस्थितियों में करने के लिए महत्वपूर्ण है जिसमें प्लाज्मा झिल्ली से आयरन द्वारा सीजीएफटीआर 1 निकलता है। परिणामों से सुझाव मिलता है कि आयरन की कमी और आयरन की पर्याप्त स्थिति दोनों में सी. ग्लाब्रेटा कोशिकाओं की उत्तर जीविता को सीजीवीपीएस 34 माध्यित आयरन होमियोस्टेसिस द्वारा बढ़ावा दिया जाता है।

स्तनधारी आनुवंशिकी प्रयोगशाला द्वारा प्रदर्शित किया गया है कि नाभिकीय रिप्रोग्रोमिंग के इफेक्टर जैसे डीएनए और मेथिल ट्रांसफरेज और हिस्टोन मोडिफायर पर्यावरण तथा आनुवंशिक सूचना के बीच एक महत्वपूर्ण स्थान रखते हैं। इनके कार्य से कार्सिनो जेनेसिस और विकास में डीएनए मेथिल ट्रांसफरेस डीएनएमटी 31 और डीएनएमटी 2 की भूमिका को समझा गया है।

आण्विक कोशिका जीव विज्ञान प्रयोगशाला द्वारा रिपोर्ट किया गया है कि पीपीई 2 नाभिक में यूकेरियोटिक अनुलेखन कारक को ट्रांसलोकेट करता है और आईएनओएस के अपस्ट्रीम विनियामक क्रम से जुड़कर आईएनओएस की अभिव्यक्ति का संदमन करता है। यह जानकारी *एम. ट्यूबरकुलोसिस* की मेजबान - रोगाणु अंत: क्रिया और रोग जनक प्रक्रिया को समझने में सहायता दे सकती है। साथ ही ईएसएटी-6 : बीटा 2 एम कम्प्लेक्सेशन के व्यापक लाक्षणीकरण को भी अध्ययन में समझा गया है।

रेशम कीट आनुवंशिकी और जीनोमिकी उत्कृष्टता केंद्र द्वारा *बॉम्बिक्स मोरी* के लिंग वाले भ्रूण चरणों के ट्रांसक्रिप्टोम विश्लेषण पर कार्य किया गया और इसमें लिंग निर्धारण तथा अवकलन में शामिल जीनों की पहचान के लिए लार्वा के शीर्ष का अध्ययन किया गया।

न्यूरोस्पोरा आनुवंशिकी प्रयोगशाला में बिना जोड़े वाले डीएनए की कोशिका विभाजन की साइलेंसिंग पर नई प्राप्तियां हुई है जो न्यूरोस्पोरा में एस्कोस्पोर के विभाजन पर है।

अनुलेखन प्रयोगशाला में विभिन्न रोगाणुओं से आरएचओ प्रोटीनों के खिलाफ पीएसयू की एंटागोनेस्टिक गतिविधियों को समझने में बड़ी प्रगति हुई है, जिसे डीएनए की मरम्मत और एंटीबायोटिक संवेदनशीलता में आरएचओ की भूमिकाओं को स्थापित किया गया तथा माइकोबैक्टीरियल क्षमताओं के साथ प्रोटीन के नए अणुओं की पहचान की गई।

पादप सूक्ष्म जीव अंत: क्रिया प्रयोगशाला में प्रदर्शित किया गया है कि जेंथोमोनोस कैमपेस्ट्रिस पीवी कैमपेस्ट्रिस (एक्ससीसी; क्रूसिफेरस पौधों का रोगाणु) जेंथोफेरीन, एक अल्फा - हाइड्रोक्सी कार्बोक्सीलेट - प्रकार साइडेरोर है जो वाइब्रियोफेरीन के समान होता है, जो अल्प-आयरन परिस्थितियों तथा रोग जनकता के तहत वृद्धि के लिए आवश्यक है। यह पहली रिपोर्ट है जिसमें प्रदर्शित किया गया है कि एक्ससीसी द्वारा जेंथोफेरीन साइडेरोफोर का उत्पादन होता है और साइडेरोफोर उत्पादन पौधों के रोगाणुओं के इस महत्वपूर्ण समूह में पौधों की वृद्धि और रोग जनकता के लिए आवश्यक है।

प्रतिरक्षा विज्ञान प्रयोगशाला में दर्शाया गया है कि रेसवरेट्रोल द्वारा संभावित मेलेनोमा कोशिका मृत्यु तुलनात्मक रूप से अन्य कैंसरों और अन्य कीमोथेरेपियूटिक एजेंटों का उद्दीपन करता है। जबकि इससे एनएफ-केबी का संदमन होता है और एमआईटीएफ डाउनरेगुलेट होता है, दूसरा मेलेनोमा कोशिका मौत में सबसे महत्वपूर्ण अंशदान कारक है।

जीवाणु आनुवंशिकी प्रयोगशाला में दर्शाया गया है कि ई. कोलाई में एंटीसेंस अनुलेखन की संभाव्यता बहुत अधिक है और इसे आरएचओ पर आधारित अनुलेखन समापन तथा आरएनए - डीएनए हाइब्रिड (आर-लूप) के निर्माण में कम आंका गया है। इसके अलावा, तीन प्रोटीन फोस्फोररिले और एक क्रिप्टिक पोटेशियम एफ्लक्स मार्ग के बीच, ई. *कोलाई* में शरीर क्रियात्मक संबंध का पता लगाया गया है और इसके नियमन में अतिरिक्ति कारकों का मॉड्यूलेशन पहचाना गया है। एक अन्य अध्ययन में दर्शाया गया है कि कठोर प्रतिक्रिया अणुओं पीपीपीजीपीपी और पीपीजीपीपी का अनुपात एसपीओटी गतिविधि के *ई. कोलाई* में घट जाने से विक्षुब्ध होता है और कोशिका व्यवहार्यता के लिए एसपीओटी का कार्य पीपीपीजीपीपी के विखण्डन में अनिवार्य है किंतु पीपीजीपीपी के लिए नहीं है।

ड्रोसोफिला विकास प्रयोगशाला द्वारा जीव विज्ञान की केंद्रीय समस्याओं में से एक का प्रदर्शन किया गया है कि जीव विज्ञान में एक ऊतक की स्थान में पहचान एक कोशिका द्वारा किस प्रकार प्राप्त की जाती है। उन्होंने इस

घटना का आण्विक आधार इस संदर्भ में अध्ययन किया है कि अनुलेखन कारकों का एचओएक्स परिवार किस प्रकार कोशिकाओं को केंद्रीय तंत्रिका तंत्र के अगले पिछले अक्ष के साथ उनकी विशेष पहचान देता है।

कोशिका मृत्यु और कोशिका उत्तर जीविता प्रयोगशाला में प्रोटीयोमिक मार्गों का उपयोग करते हुए 143 मानव फोस्फेटेज़ के एक अंत: क्रियात्मक नेटवर्क का मानचित्रण किया गया, जो 6595 उच्च विश्वास की अंत: क्रियाओं पर आधारित था, जिनमें से 85 प्रतिशत की रिपोर्ट नहीं की गई थी। इनके विश्लेषण नए कोशिकीय प्रक्रमों के साथ अनेक फॉस्फोटेज के साथ जुड़े है और इनसे कैंसर सहित विभिन्न मानव रोगों के साथ आनुवंशिक तौर पर जुड़े हुए प्रोटीनों की अंत:क्रिया को समझा गया है।

अभिकलनात्मक और कार्यात्मक जीनोमिकी प्रयोगशाला द्वारा इसे समझने के लिए एक्सरे क्रिस्टेलोग्राफी का सफलतापूर्वक उपयोग किया गया कि रोगाणु जनक ई. कोलाई एचओएसए किस प्रकार कोग्नेट डीएनए तथा इसके इफेक्टर लाइगैंड 4-हाइड्रोक्सी बेंजोइक एसिड (पीएचबीए) के साथ अंत: क्रिया करता है। इसमें एक्टोनपिक रूप से अभिव्यक्त) अनुलेखन विनियामकों के नए फिनोटाइपिक प्रभाग दर्शाए गए हैं जैसे आईसी1आर और एफएडीआर। पुन: यह सिद्ध किया गया था कि मानव हंटिंगटन प्रोटीन पॉली नेडीलेटिड होता है और इसमें विभिन्न लाइसिन अवशेषों के साथ इससे ऑटोफेगी हो सकती है।

सीडीएफडी के अनेक संकाय सदस्यों और अध्येताओं को प्रतिष्ठित राष्ट्रीय और अंतरराष्ट्रीय समान प्रदान किए गए हैं। इस अवधि के दौरान मणिपाल और हैदराबाद केंद्रीय विश्व विद्यालयों द्वारा हमारे 15 अध्येताओं को पीएचडी की उपाधि प्रदान की गई है। अनेक पोस्ट डॉक्टरल अध्येताओं, परियोजना सहयोगियों और ग्रीष्म कालीन प्रशिक्षुओं ने सीडीएफडी में कार्य किया और केंद्र के विकास में उल्लेखनीय भूमिकाएं निभाई।

केन्द्र के स्थायी परिसर निर्माण गतिविधियां लगभग पूरी हो चुकी हैं और हम जल्द ही हमारे नए परिसर में जा रहे हैं।

मैं शासी परिषद्, अनुसंधान क्षेत्र पैनल-वैज्ञानिक सलाहकार समिति, शैक्षिक/वित्त/भवन निर्माण समिति और बेशक सीडीएफडी की गतिविधियों के लिए बायोटेक्नोलॉजी विभाग द्वारा दिए गए सभी सहयोगों के प्रति आभार व्यक्त करता हूं। मैं सीडीएफडी परिवार के सभी सदस्यों और अधिकारियों को हमारी गतिविधियों तथा उपलब्धियों में समर्थन देने के लिए उनके समय और प्रयासों के लिए धन्यवाद प्रेषित करता हूं।

रंजन सेन
प्रभारी निदेशक

31 मार्च, 2017

Director’s Message

I have great pleasure in presenting the Annual Report of the Centre for DNA Fingerprinting and Diagnostics (CDFD), Hyderabad of the year 2016-17. It is an autonomous institute under the Department of Biotechnology, Govt. of India. The major activities of the institute are as follows. i) Providing high quality services in the areas of Human and Plant DNA Fingerprinting and Diagnostics of genetic disorders and ii) undertake basic research in different areas of modern biology. A few of the major achievements and research findings from the Centre during 2016-17 are mentioned below, the details of which are given under the reports of each laboratory.

During the period 2016-17, the Laboratory of DNA Fingerprinting Services received ~140 cases that were forwarded by the judiciary and the law enforcing agencies and the DNA Examiners have defended their reports in various Courts of law throughout the country.

The Diagnostics division provided genetic services to around 5000 patients for various genetic diseases. The Centre in collaboration with the Medical Genetics department of the Nizam’s Institute of Medical Sciences, Hyderabad provided genetic diagnostics services and runs the DNB program in Medical Genetics.

In view of the complexities in Basmati adulteration testing, efforts are being made by the Plant DNA Fingerprinting division to further improve the protocol by increasing the number of markers.

The Laboratory of Cell Cycle Regulation has delineated the mechanism of how RBP2 is recruited by pocket protein p130 to bring about H3K4 demethylation and gene repression of E2F-responsive genes. Further, in order to understand the role of MLL H3K4 histone methyltransferases in mitosis, they show that MLL/WDR5 complex regulates spindle formation and chromosome congression.

The Laboratory of Molecular Oncology has identified and validated novel transcriptional targets of mutant p53 relevant for squamous carcinomas. Their work suggests Ca2+/NFAT signaling to be enriched in Wnt- rectal cancer.

The Laboratory of Cell Signalling demonstrated that IP6K1 regulates cell surface-extracellular matrix signalling so that the cancer cells deficient in IP6K1 display reduced invasion, and mice lacking IP6K1 are resistant to the development of invasive carcinoma. The group also observed that elongating spermatids in Ip6k1 knockout mice fail to undergo DNA condensation and display the persistence of somatic histones. They are currently working towards a detailed molecular understanding of the role of IP6K1 and IP7 in these diverse cellular and physiological functions.

The Laboratory of Chromatin Biology and Epigenetics have discovered that sirtuin Hst4 of *S. pombe* is regulated by ubiquitin ligase SCF mediated proteolysis in response to DNA damage. The laboratory is currently investigating the role of checkpoint kinase in the signalling of degradation of Hst4 on DNA damage and determining the significance of this degradation.

In the Laboratory of Computational Biology, a new substitution-scoring matrix suitable for aligning disordered regions has been calculated and its performance evaluation is underway. A new SVM based method was developed and tested for prediction of functional impact missense mutations in the disordered regions of proteins. MD simulations on domains containing disordered regions harbouring disease causing mutations revealed that such regions lose their intrinsic conformational heterogeneity due to the mutations.

The Laboratory of Fungal Pathogenesis demonstrated for the first time that the phosphoinositide 3-kinase (PI3K) in the pathogenic yeast *Candida glabrata* is pivotal to maintenance of the cellular iron homoeostasis and retrograde trafficking, under high-iron environmental conditions, of the iron permease CgFtr1 from the plasma membrane. The results suggest that CgVps34-mediated iron homeostasis promotes survival of *C. glabrata* cells in both iron-deficient and iron-sufficient conditions.

The Laboratory of Mammalian Genetics demonstrated that effectors of nuclear reprogramming like DNA methyltransferases and histone modifiers play an important interphase between environment and the genetic

information. Their work has dissected out the role of DNA methyltransferases Dnmt3l and Dnmt2 in carcinogenesis and development.

The Laboratory of Molecular Cell Biology reported that PPE2 as a eukaryotic transcription factor translocates into the nucleus and binds to upstream regulatory sequences of iNOS, inhibiting the expression of the inos. This information may be helpful to understand host-pathogen interaction and virulence mechanism of *M. tuberculosis*. Also, their study elucidate comprehensive characterization of ESAT-6:β2M complexation.

The Centre of Excellence in Silkmoth Genetics and Genomics worked on the transcriptome analysis of sexed embryonic stages and larval heads of *Bombyx mori* to identify genes involved in the sex determination and differentiation.

The Laboratory of Neurospora Genetics have the novel findings on meiotic silencing by unpaired DNA, and on the ascospore partitioning in Neurospora.

The Laboratory of Transcription made major progresses in understanding the antagonistic activities of Psu against Rho proteins from different pathogens, established roles of Rho in DNA repair and antibiotic sensitivity and identified new protein molecules with myco-bacteriocidal abilities.

The Laboratory of Plant Microbe Interaction have demonstrated that *Xanthomonas campestris pv. campestris* (Xcc; a pathogen of cruciferous plants) produces xanthoferrin, a α-hydroxy carboxylate-type siderophore similar to vibrioferrin, which is required for growth under low-iron conditions and virulence. This is the first report which demonstrates that Xcc produce xanthoferrin siderophore and siderophore production is required for in planta growth and virulence in this important group of plant pathogens.

The Laboratory of Immunology showed that resveratrol induces potent melanoma cell deathcompared to other cancers and other chemotherapeutic agents. Though it inhibits NF-κB and downregulates MITF, latter is the most important contributing factor for melanoma cell death.

The Laboratory of Bacterial Genetics has shown that the potential for antisense transcription in *E. coli* is quite large and that it has been underestimated in past on account of Rho-dependent transcription termination and formation of RNA-DNA hybrids (R-loops). Additionally, a physiological connection between the three protein phosphorelay and a cryptic potassium efflux pathway, in *E. coli* has been delineated and additional factors modulating its regulation have been identified. Another study has shown that the ratio of the stringent response molecules, pppGpp and ppGpp is perturbed by the lowering of SpoT activity in E. coli and that the SpoT function essential for cell viability is the degradation of pppGpp, but not ppGpp.

The Laboratory of Drosophila Development demonstrated one of the central problems in biology is to understand how a cell obtains its positional identity in a tissue. They studied the molecular basis of this phenomenon in context of how Hox family of transcription factors give cells their specific identity along the anterior posterior axis of the central nervous system.

Laboratory of Cell Death & Cell Survival, by utilizing proteomic approaches, mapped an interaction network of 143 human phosphatases built on 6596 high-confidence interactions of which 85% were unreported. Their analysis has linked several phosphatases with new cellular processes and unveiled protein interactions genetically linked to various human diseases including cancer.

Laboratory of Computational and Functional Genomics have successfully used X-ray crystallography to understand how pathogenic *E. coli* HosA interacts with its cognate DNA and its effector-ligand 4-hydroxy benzoic acid (PHBA). They have shown novel phenotypic effects of ectopically expressed transcription regulators like IclR and FadR. Further, it was established that human Huntingtin protein is poly-neddylated at different lysine residues and can lead to autophagy.

Many CDFD faculty and scholars have been recipients of prestigious national and international awards and honours. During this period, the Manipal and Hyderabad Central Universities conferred fifteen of our research scholars with PhD degrees. Many postdoctoral fellows, project associates and summer trainees worked at CDFD and play significant roles in the Centre's development.

The Centre's permanent campus construction activities are almost completed and we will soon be shifting to our new campus.

I take this opportunity to acknowledge all the co-operation extended by the Governing Council, Research Area Panels-Scientific Advisory Committee, Academic / Finance / Building Committees and, of course, the Department of Biotechnology for the activities of CDFD. I wish to thank all the members and officials of the CDFD family for their time and effort in supporting our activities and achievements.

Ranjan Sen

March 31, 2017

सेवाएँ
Services

LABORATORY OF DNA FINGERPRINTING SERVICES

In-charge	Madhusudan Reddy Nandineni	Staff Scientist
Other members	SPR Prasad	Senior Technical Officer
	Devinder Singh Negi	Technical Officer
	Sanjukta Mukerjee	Technical Officer
	Pooja Tripathi	Technical Officer
	Kiranmai Joshi	Technical Officer
	Vijay Amrutarao Girnar	Technical Assistant
	Shruti Dasgupta	Technical Assistant
	Neelima Thota	Technical Officer (till Aug. 2016)
	Chandra Shekhar Singh	Technical Assistant (till Aug. 2016)
	Devinder Kumar	Technical Officer (till Nov. 2016)
	Ch V Goud	Technical Officer
Co-ordinator	D P Kasbekar	Haldane Chair

Objectives:

1) To provide DNA fingerprinting services in cases forwarded by law-enforcing agencies / judiciary of State and Federal Governments, relating to murder, sexual assault, paternity, maternity, child swapping, deceased identification, organ transplantation, etc.,
2) To develop human resources skilled in DNA fingerprinting, to cater to the needs of State and Federal Government agencies;
3) To impart periodical training to manpower involved in DNA fingerprinting sponsored by State and Federal Government agencies;
4) To provide advisory services to State and Federal Government agencies in establishing DNA Fingerprinting facility;
5) To create DNA marker databases of different populations of India.

Summary of services provided until the beginning of the reporting year (1st April 2015 to 31st March 2016):

A total of 397 cases were received for DNA fingerprinting examination during the reporting period 2015 – 2016. Of these, 162 cases were related to identification of deceased, 99 cases were pertaining to sexual assault (rape), 98 cases were related to paternity / maternity, 19 cases were related to murder and 19 cases were pertaining to biological relationship (organ transplantation). Twenty States and Union Territories of India and one foreign country (East Timor) have availed the DNA fingerprinting services of CDFD during this period. Madhya Pradesh forwarded the highest number of cases (176) followed by Telangana (55), Chhattisgarh (49), Andhra Pradesh (27), Punjab (21), Goa (19), Tamil Nadu (16), Puducherry (5), Karnataka (5), Kerala (3), Maharashtra (3), East Timor (3), Uttar Pradesh (3), Andaman & Nicobar (2), Bihar (2), Haryana (2), West Bengal (2), Delhi (1), Himachal Pradesh (1), Odisha (1) and Rajasthan (1)

Details of services provided in the current reporting year, (1st April 2016 to 31st March 2017):

Breakup of the cases during this reporting period is given below under following heads:

Biological relationship	21
Identity of deceased	38
Murder	02
Paternity/Maternity	70
Sexual assault (Rape)	12
Total number of cases	**143**

Prominent cases during April 1, 2016 to March 31, 2017

Cases from National Investigation Agency (NIA) involving national security and public safety, e.g.: cases of terror attacks on Pathankot Indian Air Force base, Punjab State, Uri , J & K State, etc.

Deposition of Evidence in Courts of Law

During this reporting year, the DNA experts defended their reports in 21 cases in various Honorable Courts throughout the country.

Training / Lectures / Workshops by LDFS personnel: 2016 - 2017

1. Lecture was delivered for the benefit of the Police and Judicial Officers at the SVP National Police Academy, Hyderabad on 18.08.2016
2. Lecture was delivered for the benefit of Police Officers at Telangana State Police Academy on 03.11.2016
3. Training was provided to the scientific personnel working in DNA Centre at Forensic Science Laboratory, Bengaluru, Karnataka State during 22.11.2016 to 28.11.2016
4. Lecture was delivered on "Use of SNPs and Next Generation Sequencing technology for Forensic Human Identification" at the All India Police Science Congress (AIPSC) during 8-9th December, 2016
5. Poster presentation at India International Science Festival, IISF – 2016, in DBT pavilion at CSIR – National Physical Laboratory, Delhi during 7th to 11th December, 2016 and awarded "Best Poster Award"
5. Lecture was delivered at CDFD for the benefit of the students and faculty members of Department of Biotechnology, Yashvantrao Chavan Institute of Services, Satara on 21.12.2016
6. Lecture was delivered at CDFD for the benefit of the students and faculty members from Dept. of Genetics, Aurora's Degree & Post Graduate College, Chikkadpally, Hyderabad on 05.01.2017

Summary of the State-wise breakup of DNA fingerprinting cases

Name of the State	Biological relationship	Identity of deceased	Maternity / Paternity	Murder	Sexual assault (Rape)	Total No. of Cases
Andaman & Nicobar			1			1
Andhra Pradesh			8			8
Bihar			3			3
Chhattisgarh		11	29		1	41
Delhi		5				5
Goa		8	3	2	1	14
Jammu & Kashmir			2			2
Karnataka	1		9			10
Madhya Pradesh			1			1
Maharashtra			3			3
Puducherry		2	3			5
Punjab		8	4		10	22
Tamil Nadu	11	1				12
Telangana	9		3			12
Tripura		1				1
Uttar Pradesh		1	1			2
West Bengal		1				1
Total No. of Cases.	**21**	**38**	**70**	**2**	**12**	**143**

7. Lecture was delivered at CDFD for the benefit of the students and faculty members of Sacred Heart College, Department of Zoology, Kerala University on 24.01.2017
8. Lecture was delivered at CDFD for the benefit of the Air Force Officers from Air Force Intelligence School, Lohegaon, Pune on 06.02.2017
9. Lecture was delivered at CDFD for the benefit of the students and faculty members from School of Social Work Roshni Nilaya, Mangaluru, Karnataka State on 16.02.2017
10. Lecture was delivered at CDFD for the benefit of the students from Savitribai Phule Pune University, Pune on 07.03.2017

A total of 143 cases were received for DNA fingerprinting examination during the current reporting period (2016 – 2017). Of these cases 70 cases were related to maternity/paternity, 38 cases were related to identity of deceased, 21 cases were related to biological relationship, 12 cases were related to sexual assault (rape) and 2 cases were related to murder. 15 States and two Union Territories of India have availed the DNA fingerprinting services of CDFD during this period. Chhattisgarh forwarded the highest number of cases (41) followed by Punjab (22), Goa (14), Tamil Nadu (12), Telangana (12), Karnataka (10), Andhra Pradesh (8), Delhi (5), Puducherry (5), Bihar (3), Maharashtra (3), Jammu & Kashmir (2), Uttar Pradesh (2), Andaman & Nicobar (1), Madhya Pradesh (1), Tripura (1) and West Bengal (1). (Figure 1)

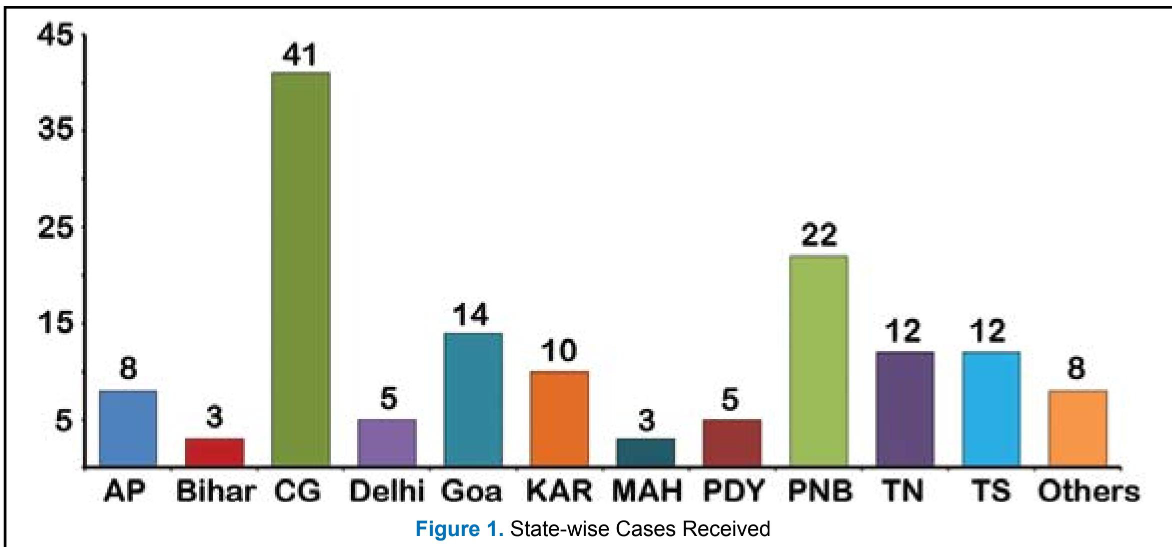

Figure 1. State-wise Cases Received

The cases involving maternity/paternity (49%), deceased identity (27%), biological relationship (15%) and sexual assault (8%) constituted the bulk of the cases received (Figure 2).

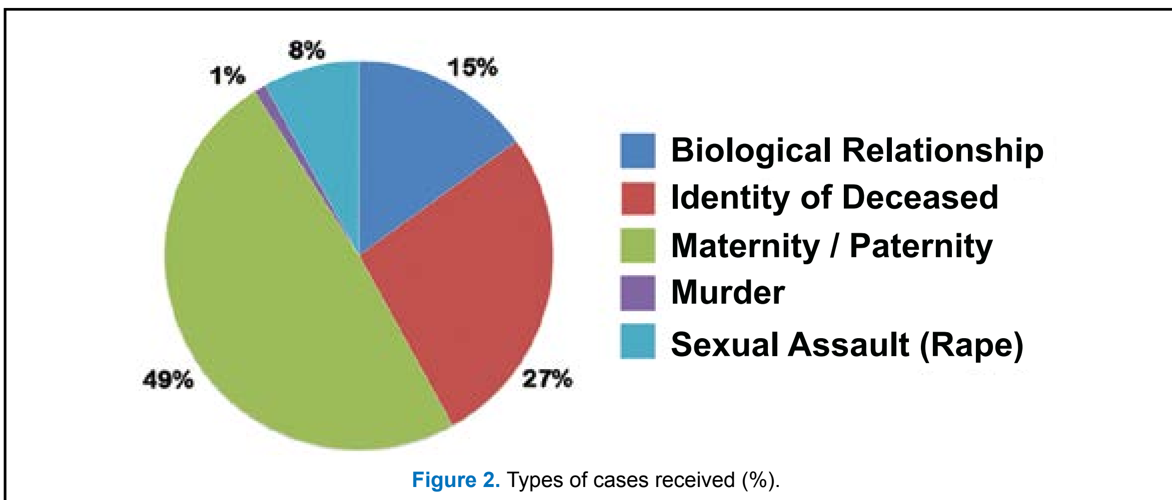

Figure 2. Types of cases received (%).

Revenues generated:

During this reporting period, an amount of ₹.34,94,503/- (Rupees thirty four lakhs ninety four thousand five hundred and three only) has been received towards DNA fingerprinting analysis charges, which is inclusive of service charge (15%) as levied by Govt. of India.

DIAGNOSTICS DIVISION

Faculty	Ashwin Dalal	Staff Scientist
PhD Students	Anusha Uttarilli	Senior Research Fellow (till April 2016)
	Anjana Kar	Senior Research Fellow
	Dipti Deshpande	Senior Research Fellow
	Sandeep	Junior Research Fellow (since Feb. 2017)
Other Members	Aneek Das Bhowmik	Research Associate
	Maria Celestina Vanaja	Research Associate
	Vineeth VS	Research Associate
	Amrita Bhattacherjee	Research Associate (since Feb. 2017)
	Avinash Pagdhune	SIAMG Fellow (till Feb. 2017)
	Krishna Reddy Ch	SIAMG Fellow (till Feb. 2017)
	Ramya	SIAMG Fellow (since Sept. 2016)
	Padmaja T	SIAMG Fellow (since Sept. 2016)
	P Divya	Project Assistant
	M Chitra	Project Assistant (since Jan. 2017)
	Sravani	Project Assistant (since March 2017)
	P. Rajitha	Technical Officer
	Angalena R	Senior Technical Officer
	Usha Rani Dutta	Technical Officer
	M Muthulakshmi	Technical Officer
	A Sobhan Babu	Technical Officer
	Jamal Md Nurul Jain	Technical Officer
	Vasantha Rani	Technical Officer
	C. Krishna Prasad	Technician
	R. Sudheer Kumar	Technician
	Prajnya Ranganath	Associate Professor, NIMS (Adjunct Faculty of CDFD)
	Shagun Aggarwal	Associate Professor, NIMS (Adjunct Faculty of CDFD)
	Dhanya Lakshmi N	Assistant Professor, NIMS (Adjunct Faculty of CDFD) (since Dec. 2016)

Objectives

1. To conduct genetic evaluation for patients/ families with genetic disorders
2. To develop new methods and assays for genetic analysis and engage in research on chromosomal and single gene disorders
3. To act as national referral center for analysis and quality control of genetic tests for few genetic diseases
4. To impart training in genetic evaluation of patients with genetic disorders

I. Services provided and Training programs during the year 2016-2017

Clinical Genetics

A total of 5469 patient samples were analyzed for genetic testing, during the year 2016-17. These consisted of patients with chromosomal disorders, monogenic disorders, mental retardation, congenital malformations, inborn errors of metabolism, and other familial disorders. The SIAMG fellowship program for training in Clinical Cytogenetics and Clinical Molecular Genetics has been initiated in collaboration with Society

for Indian Academy of Medical Genetics. One student each joined for the fellowship program and two students completed the fellowship in Clinical Cytogenetics and Clinical Molecular Genetics during 2016-17.

The Department of Medical Genetics established at Nizam's Institute of Medical Sciences, Hyderabad is functioning successfully. A total of 3707 patients were examined and counseled in the unit during 2016-17. In addition antenatal ultrasonograms were done in 425 cases, antenatal invasive procedures (chorionic villus sampling and amniocentesis) in 182 cases and foetal autopsies were conducted in 107 foetuses.

A 3 year training program for Diplomate of National Board (DNB) in Medical Genetics initiated with affiliation to National Board of Examinations, New Delhi is running successfully.

Genetic investigations done during 2016-17

Investigation	Total cases	Positives
Cytogenetics	1911	156 (8.2%)
Proband	1611	148 (9.2%)
Prenatal	310	8 (2.6%)
Molecular Genetics	2696	1094(40.5 %)
Proband	2514	1060(42.1%)
Prenatal	182	34(18.7%)
Biochemical Genetics	862	236 (27.3%)
Proband	835	220 (26.3%)
Prenatal	27	16 (59.25%)

Cytogenetics

Disease	Abnormality	No of cases
Down Syndrome	47,XY,+21 47,XX,+21	24 28
	46,XX,rob(21;21) +21	2
	mos47,XY+21/46,XY	1
	47,XY,+21,inv(9)	1
Patau Syndrome	47,SC,+13	1
Turner syndrome	Monosomy X (45,X)	5
	mos 45,X/ 46,XY	1
	mos 45,X/46,X,i(X)	1
	mos 46,XY/45,X	2
	46,X,i(X)(q10)	1
	mos 45X/46,XX	3
Kleinefelter Syndrome	47,XXY	5
	mos47,XXY/46,XY	1
Sex reversal	46,XX 46,XY	2 1

Structural chromosomal abnormalities

Inversions	
46,XX,inv(5)(p13q13)	1
46,X,inv(Y)	4
46,XX,inv(4)(p13q13)	1
46,XY,inv(9)	5
Deletions	
46,XX,del(5)	1
46,XX,del(18)q	1
46,XX,del(11)q	1
Duplications	
46,XX,10q+	1
46,XY,21q+	1
Translocations	
46,XX,t(2;3)(p21;p21.3)	1
46,XY,t(11;17)	1
47,XY,der(9)t(9;14)pat	1
46,XY,t(9;14)	1
46,XX,der(20)t(9;20)	1
46,XX,t(9;20)(p13;p13)	1
46,XY,t(1;9)(p36.1;p23)	1
46,XY,der(12),t(11;12)(q23;p13)mat	1
46,XX,t(11;12)(q23;p13)	1
46,XX,t(8;10)(q13;q22.1)	1
46,XY,t(2;5)(p23;q13)	1
45,SC,t(13;14)(q11.1;q11.1)pat	1
46,SC,t(13;15)((q14.1;q26.1)mat	1
Polymorphic variants	32

Quantitative Fluorescent PCR (QF-PCR

MLPA	Cases	Positives
Prenatal (Aneuploidy)	95	5
Postnatal (Microdeletion syndromes)	135	12

Fluorescence *in situ* Hybridization (FISH)

Disease / translocation	Probe	No of tests
Prader-Willi Syndrome	SNRPN(15q11)/PML(15q24)	6
Di-George Syndrome	TUPLE(22q11.2)/ARSA(22q13)	10
Marker chromosome	WCP-11, WCP-13, 9, 18 SE(X)/(Y), Acro-p-arm	15
Spectral karyotyping		12

Biochemical Genetics

Disease/Test	Positives
Urine & Blood Metabolic Screening tests (N=260)	**61**
Amino acid disorders (N=172)	**54**
Non Ketotic Hyperglycinemia	9
Hyperornithinemia	2
Hypermethioninemia	1
Phenylketonuria	3
MSUD	3
Increased plasma Glutamic acid	11
Other amino acid disorders	16
Hyperhomocysteinemia	9
Disease/Test	**Positive**
Lysosomal storage disorders (N=403)	**105**
Hurler syndrome(20)	9
Hunter syndrome(8)	11
Sanfilippo B (8)	4
Morquio A disease (17)	22
Arylsulphatase B (9)	6
Sly disease (13)	1
GM1-Gangliosidosis (86)	7
Gaucher disease (27)	8
Krabbe disease (20)	2
Pompe disease (4)	3
Niemann Pick disease (17)	9
Mucolipidosis	5
Metachromatic Leukodystrophy (31)	9
Fabry's disease(10)	2
Hexosaminidase A/B (27)	
Tay Sachs disease	4
Sandhoff disease	1
Multiple sulfatase	2
Alpha mannosidase (1)	0

Prenatal diagnosis (27)	**16**
Pompe's disease (2)	1
Krabbe's disease (1)	1
Metachromatic Leukodystrophy (4)	1
Gaucher's disease (1)	4
Hurler syndrome	1
Sly disease	2
Morquio A disease	1
GM1- Gangliosidosis	3
Niemann Pick disease (2)	2
Hexosamindase A/B (1)	0
Other amino acid disorders	16
Hyperhomocysteinemia	9

Molecular Genetics

Name of Disorders	No of cases	Positive	Negative		
DMD/BMD	319	231	88		
DMD Carrier Analysis	63	19	44		
Spinal Muscular Atrophy	152	62	90		
SMA Carrier Analysis	70	40	30		
Hemophilia	38	10	28		
		Normal	**Homozygous**	**Heterozygous**	**Compound Heterozygous**
Beta thalassemia and Sickle cell anemia	444	33	225	96	90
Factor V Leiden	304	289	0	15	NA
Factor II mutation	182	182	0	0	NA
Cystic Fibrosis	132	124	1	7	NA
Pancreatitis/SPINK	54	34	5	15	NA

Connexin 26	17	6	4	7	NA
Achondroplasia	24	12	0	12	NA
Alpha thalassemia	29	23	2 triplication	4	NA
Gilbert Syndrome	54	5	35	14	NA
LHON disease	5	5	0	0	NA
Leigh disease	5	4	1	0	NA
MTHFR(A222V)	11	8	0	3	NA
MTHFR (E429A)	11	2	1	8	NA
Triplet Repeat Disorder		**Positive**	**Negative**		
Friedrichs Ataxia	54	23	31		
Myotonic Dystrophy	61	39	22		
Huntington Disease	66	47	19		
SCA Panel (1,2,3,6 &7)	104	20	84		
SCA 36	03	01	02		
DRPLA	15	0	15		
Spinobulbar Muscular Atrophy (SBMA)	2	1	1		
Fragile X Syndrome	295	22	273		

Cpd Heterozygous= Compound Heterozygous, NA- Not applicable

MOLECULAR GENETICS—PRENATAL DIAGNOSIS

	No Of Cases	Positive	Negative		
DMD	18	5	13		
Spinal Muscular atrophy	24	5	19		
Cystic Fibrosis	16	1	15		
Myotonic dystrophy	03	01	02		
SCA7	01	0	01		
Fragile X Syndrome	2	1	1		
Hemophilia	4	0	4		
Achondroplasia	1	1	0		
		Normal	**Homozygous**	**Heterozygous**	**Compound Heterozygous**
β thalassemia	112	92	11	00	09
Connexin	1	0	1		

II. Diagnostics Research

Project 1: Human exome sequencing for identification of novel genes in rare mendelian disorders

Summary of work done until the beginning of this reporting year (April 1, 2015 – March 31, 2016)

Single gene disorders are rare health conditions that affect a small number of people as compared to other diseases in population. But collectively they account for important cause of morbidity and mortality. To date ~ 7000 distinct rare diseases have been documented and new rare diseases are being reported regularly. The classical methods of gene identification include chromosomal mapping, linkage analysis and Homozygosity mapping. Although these methods are persuasive, there are certain limitations, which have been overcome by new sequencing technology: Massively parallel sequencing or Next generation sequencing. Next generation sequencing has made it possible to identify candidate gene using just a few affected individuals or parent child trio.

The identification of candidate gene for single gene disorders has importance, not only in prenatal diagnosis and genetic counseling of affected families, but also in basic research towards understanding of gene function and pathophysiology of disease. Over the past years of providing services in clinical genetics, we have identified several interesting novel disorders and syndromes with a single gene pattern of Mendelian inheritance. We have employed exome sequencing to identify novel genes in such families.

Details of work done in the current reporting year (April 1, 2016 – March 31, 2017)

We studied a family wherein three siblings were affected with mental retardation, ptosis and polydactyly phenotype and born out of consanguineous marriage. A combination of homozygosity mapping followed by exome sequencing of all the three affected individuals was used. Exome sequence analysis revealed a novel synonymous splice site variant c.879G>A in *ARMC9* as a candidate gene. *ARMC9* (armadillo repeat containing protein family member 9) is a conserved protein with N-terminal Lissencephaly homology domain (LiSH) and C-terminal Armadillo repeat motif (ARM) domain. The tandem ARM repeats in ARM domains of *ARMC9* folds together as a series of helices forming a super helix that creates a surface or groove for protein interactions similar to Beta catenin and predicted to be involved in microtubule dynamics. Yeast two hybrid assay has shown that *ARMC9* interacts with Siah E3 ubiquitin protein ligase 1, which indicates that *ARMC9* may be involved in ubiquitination pathway like ARMC8. Sanger sequencing and validation of variant has been done in all affected individuals, parents and unaffected sibling, which shows autosomal recessive segregation patten. Functional analysis of c.879G>A in *ARMC9* for splicing defect using pCAS2 minigene system indicates loss of exon 9 of *ARMC9* gene due to alteration in donor site. Skipping of exon 9 in *ARMC9* gene is likely to lead to in-frame deletion of 33 amino acids from ARM domain (deletion of 261-293 aa) which is likely to influence protein binding capabilities of *ARMC9*. *In-silico*predictions also indicate that deletion of 33 aa as a result of splicing defect caused by c.879G>A will lead to disruption of its structure and hence may abolish native function of *ARMC9* (Fig 1). *ARMC9* joins an important group of highly conserved ARM repeat containing protein associated with intellectual disability, which includes Beta catenin (*CTNNB1)* and *APC2*.

Project II: Development and application of a next generation sequencing approach for molecular genetic analysis of lysosomal storage disorders. (This is a new activity)

Sanger sequencing is very useful for sequence analysis of small genes. However, when applied for large genomic regions it becomes time consuming and laborious, requiring multiple PCR reactions for generating amplicons for sequencing. The development of high throughput massively parallel sequencing strategies in recent years has revolutionized the concept of sequence analysis and has made sequencing of large genomic segments far more feasible and much less time-consuming. In the present project we amplified about 5 kb fragments of genomic DNA from specific lysosomal storage disease gene and then pool the samples for next generation sequencing based analysis. Pooling of samples from different individuals with different affected genes will help to decrease the cost of sequencing significantly.

Summary of work done until the beginning of this reporting year (April 1, 2016 – March 31, 2017)

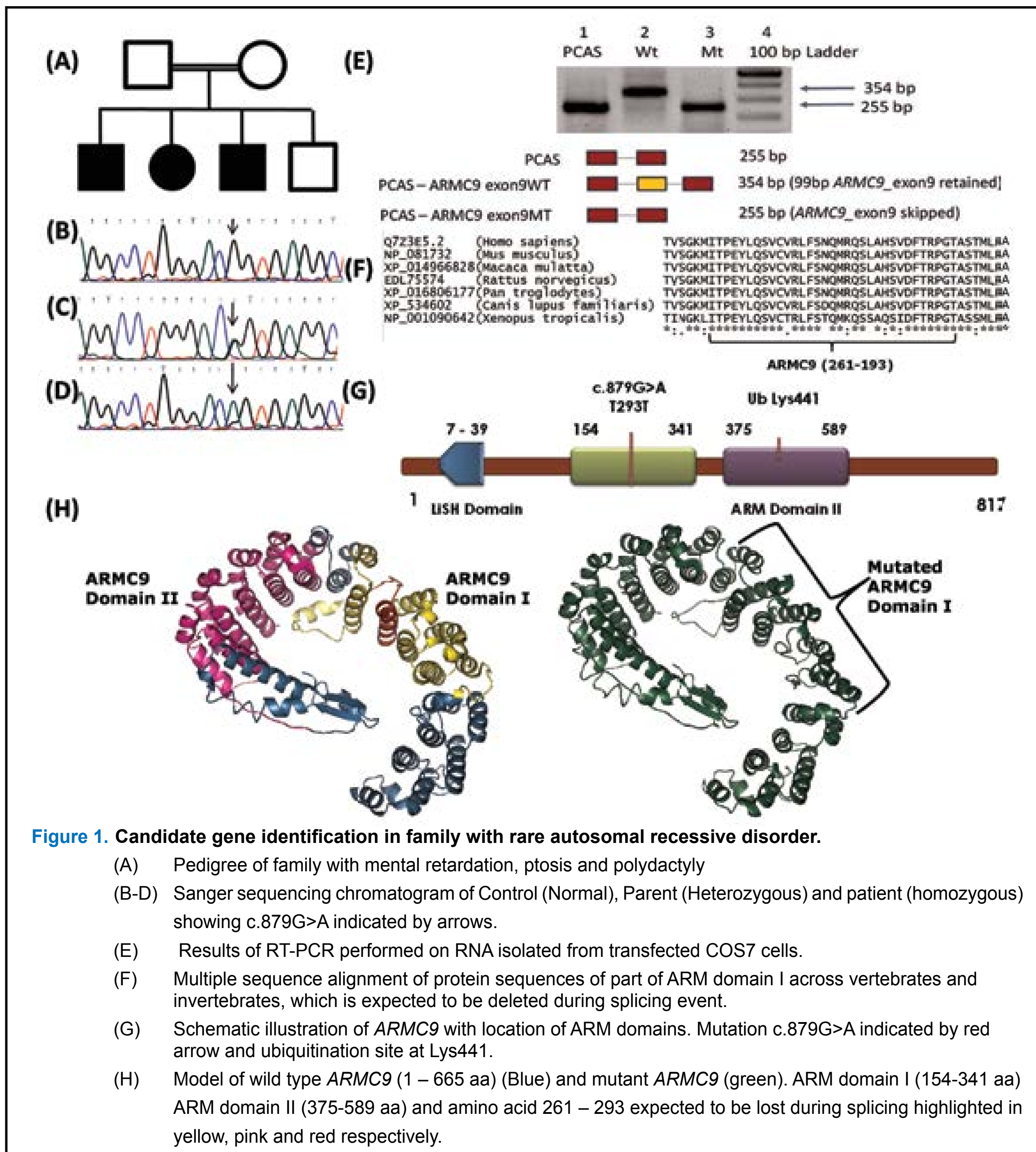

Figure 1. Candidate gene identification in family with rare autosomal recessive disorder.

(A) Pedigree of family with mental retardation, ptosis and polydactyly

(B-D) Sanger sequencing chromatogram of Control (Normal), Parent (Heterozygous) and patient (homozygous) showing c.879G>A indicated by arrows.

(E) Results of RT-PCR performed on RNA isolated from transfected COS7 cells.

(F) Multiple sequence alignment of protein sequences of part of ARM domain I across vertebrates and invertebrates, which is expected to be deleted during splicing event.

(G) Schematic illustration of *ARMC9* with location of ARM domains. Mutation c.879G>A indicated by red arrow and ubiquitination site at Lys441.

(H) Model of wild type *ARMC9* (1 – 665 aa) (Blue) and mutant *ARMC9* (green). ARM domain I (154-341 aa) ARM domain II (375-589 aa) and amino acid 261 – 293 expected to be lost during splicing highlighted in yellow, pink and red respectively.

Five different long PCR based libraries were designed which included the genes-NEU1 (Sialidosis), SMPD1 (Niemann-Pick Disease-Type B and Niemann-Pick Disease-Type A), IDUA (Mucopolysaccharidosis type I), ARSA (Metachromatic leukodystrophy), NPC1 (Niemann-Pick disease, type C1), NPC2 (Niemann-Pick disease, type C2),GBA (Gaucher disease), GAA (Pompe disease), GLB1 (GM1 gangliosidosis, GNPTAB (I-cell disease), GALNS (Morquio syndrome). Long range PCR primers were used along with TAKARA GXL DNA Polymerase for each gene for amplification of 5-10kb fragments containing the exons and intronic regions. The amplified products were diluted to 10ng/µl based on the dsDNA quantification. Each library was constructed by mixing the amplified products to make a total volume of 100µl with a final concentration of 1000ng (100µl / 1000ng). One patient for each gene was included in one library. The constructed libraries were sequenced on an Illumina MiSeq NGS platform. Quality control of the FASTQ file generated was done by FASTQC, followed by data alignment by BWA, Variant calling by GATK pipeline and Variant Annotation

by Annovar. Variants identified in each gene were then Sanger validated (Fig 2). We found 100% concordance with Sanger sequencing of suspected disease causing variants in all patients studied. We plan to conduct more such runs and hope to develop a Long-range PCR combined with next generation sequencing strategy as a cost effective, reliable and accurate tool in the molecular diagnosis of LSDs as well as other genetic diseases.

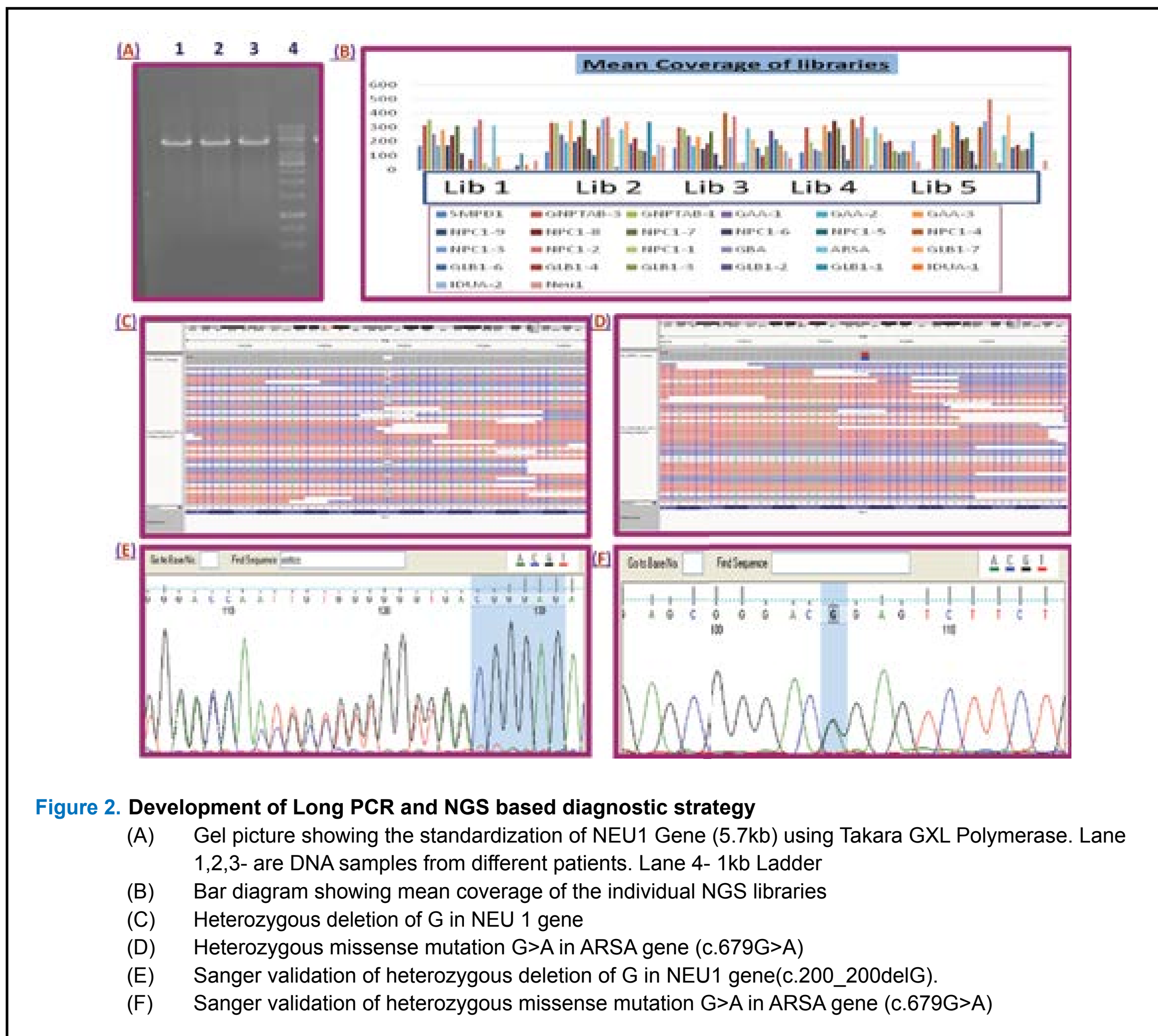

Figure 2. Development of Long PCR and NGS based diagnostic strategy

(A) Gel picture showing the standardization of NEU1 Gene (5.7kb) using Takara GXL Polymerase. Lane 1,2,3- are DNA samples from different patients. Lane 4- 1kb Ladder

(B) Bar diagram showing mean coverage of the individual NGS libraries

(C) Heterozygous deletion of G in NEU 1 gene

(D) Heterozygous missense mutation G>A in ARSA gene (c.679G>A)

(E) Sanger validation of heterozygous deletion of G in NEU1 gene(c.200_200delG).

(F) Sanger validation of heterozygous missense mutation G>A in ARSA gene (c.679G>A)

Project III: Clinical, biochemical and molecular analysis of lysosomal storage disorders

Summary of work done until the beginning of this reporting year (April 1, 2015 – March 31, 2016)

Lysosomal storage disorders are a heterogeneous group of disorders associated with specific lysosomal enzyme deficiency. The diagnosis in most of these disorders is based on enzyme assay. There is a large amount of overlap in enzyme levels among carriers and normal people; hence it is very difficult to detect carriers by enzyme assay. Mutation detection is helpful for carrier detection and accurate prenatal diagnosis. Our study aims to characterize the clinical features, biochemical parameters and molecular defects in various lysosomal storage disorders.

Details of work done in the current reporting year (April 1, 2016– March 31, 2017)

Over last seven years we have been able to identify mutations in more than 350 patients with different lysosomal storage diseases (LSDs) (Table 1). This was done as part of a National Task Force on Lysosomal Storage Diseases funded by Indian Council of Medical Research and Department of Health Research. This study has revealed the mutation spectrum in patients with LSDs in the Indian population.

Lysosomal Storage Disorder	Gene	Number of cases	Total mutations	Novel mutations
Niemann-Pick disease types A & B	SMPD1	127	81	38
Niemann- Pick disease type C	NPC1	5	3	3
Niemann- Pick disease type C	NPC2	1	1	1
Metachromatic leukodystrophy	ARSA	79	56	23
Mucopolysaccharidosis I	IDUA	31	22	15
Mucopolysaccharidosis II	IDS	33	20	7
Mucopolysaccharidosis VI	ARSB	38	24	18
Sialidosis	NEU1	5	3	3
Mucolipidosis II	GNPTAB	50	32	24
Total		**369**	**242**	**132**

Table 1. Data sheet showing mutation analysis for LSDs over last seven years

Publications

1. Ranganath P, Matta D, Bhavani GS, Wangnekar S, Jain JM, Verma IC, Kabra M, Puri RD, Danda S, Gupta N, Girisha KM, Sankar VH, Patil SJ, Ramadevi AR, Bhat M, Gowrishankar K, Mandal K, Aggarwal S, Tamhankar PM, Tilak P, Phadke SR, and Dalal A. (2016) Spectrum of SMPD1 mutations in Asian-Indian patients with acid sphingomyelinase (ASM)-deficient Niemann-Pick disease.***American Journal of Medical Genetics Part A***170(10):2719-2730.

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Members	Dr. K. Anupama	Staff Scientist (since Sept. 2016)
	Dr. V.V. Satyavathi	Technical Officer (till March 2017)
	Neelima Thota	Technical Officer (since Sept. 2016)
	Lakshmi Vaishna	Technical Assistant (since March 2017)
	G. Shivaram	Office supporting staff (since Sept. 2016)
Other Members	Binod Bihari Pradhan	Technical officer
	Krishnamurty	Tradesman

Objectives

1. Testing the purity of Basmati samples received from Export Inspection Council (EIC), Ministry of Commerce, Government of India, Basmati rice exporters from India, and other countries; and
2. To assess the genetic purity of rice hybrids and parental lines used in rice hybrid seed production.

Summary of the work done until the beginning of this reporting year (upto March 31, 2016)

A total of 209 Basmati samples were analyzed out of which 63% of samples were pure samples, 26% of the samples were adulterated with non-basmati rice below 15% and only 1% of the samples were adulterated above 15%.

Details of progress made in the current reporting year (April 1, 2016 - March 31, 2017)

Objective 1: Testing the purity of Basmati samples received from Export Inspection Council (EIC), Ministry of Commerce, Govt. of India, Basmati rice exporters from India and other countries.

During the current reporting year, a total of 153 samples were analyzed and the number of samples indicating the percentage of adulteration with non-basmati rice is shown in Figure 1.

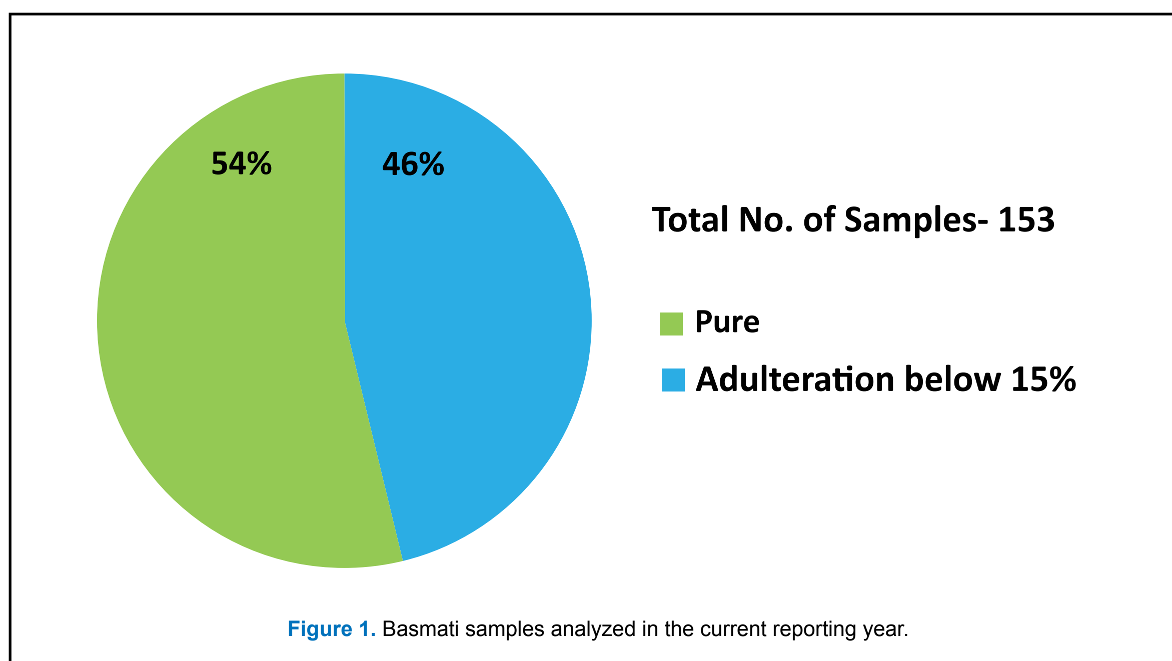

Figure 1. Basmati samples analyzed in the current reporting year.

The protocol for DNA testing of Basmati rice was initially developed for detection of adulteration using eight Simple Sequence Repeats (SSRs) marker assay with eleven notified Basmati varieties. In view of the complexities and challenges arising in adulteration testing, efforts are being made to further expand as well as fine tune the present protocol as mentioned below:

i) Updating the database of Basmati varieties.

At present twenty six varieties of Basmati rice have been notified by Department of Agriculture and Cooperation (DAC) under Seeds Act, 1966. We have extended our method of multiplexed eight markers panel analysis for identification of twenty two notified varieties to generate a comprehensive database. The profiles of the remaining varieties will be generated at the earliest.

ii) Single grain analysis for varietal identification.

On the unknown rice samples, where the sample was predominantly one variety, the identification using our standardized method is in good agreement. However, for identification of rice varieties in samples of complex mixtures, single grain analysis is now being used.

iii) Increase the number of SSRs in the panel for better resolution of complex mixtures and varietal identification

With the constant release of new Basmati rice varieties, it becomes imperative to incorporate more number of SSR markers in the present assay. SSR markers having high polymorphic information content (PIC) are selected and are presently being tested to identify markers that help in clear identification of Basmati varieties.

Objective 2: To assess the genetic purity of rice hybrids and parental lines used in rice hybrid seed production.

Three-line system is widely used in India for hybrid seed production. The three lines used are (a) Cytoplasmic Male Sterile (CMS/A) line (b) Maintainer (B) line and (c) Restorer (R) line. According to Indian Seed Act, purity of hybrid rice should be of 98% and that of the cytoplasmic male sterile line should be of 98%. It is estimated that 1% impurity in hybrid seed reduces the yield by 100Kg/hectare. CMS and maintainer lines are iso-nuclear lines but differ in the sequence of the gene present in the mitochondrial genome that governs male sterility. Several molecular markers (both co-dominant and dominant) that can differentiate these lines are available.

In the current reporting year, we have developed three co-dominant markers that differentiate CMS and maintainer lines. We have labeled the 5'-end of the forward primers of the above mentioned markers along with some other reported markers with fluorescent fluorophores and are currently involved in developing an assay to test genetic purity of bulked seed samples (mixed in different ratios of CMS and maintainer lines) using capillary electrophoresis.

शोध
Research

LABORATORY OF BACTERIAL GENETICS

Studies on gene regulation, transcription termination, and amino acid and ion-transport in *Escherichia coli*

Faculty	Abhijit A Sardesai	Staff Scientist
	R Harinarayanan	Staff Scientist
	J Gowrishankar	J C Bose National Fellow
Ph D Students	Amit Pathania	Senior Research Fellow (till June 2016)
	Aanisa Nazir	Senior Research Fellow (till September 2016)
	Rajvardhan M Kapshikar	Senior Research Fellow
	Suchitra Upreti	Senior Research Fellow
	Nalini Raghunathan	Senior Research Fellow
	Rajeshree Sanyal	Senior Research Fellow
	Nida Ali	Senior Research Fellow
	Ravish Sharma	Senior Research Fellow
	J Mallikarjun	Junior Research Fellow
	Sayantan Goswami	Junior Research Fellow
	Swati Dubey	Junior Research Fellow
	Vani Singh	Junior Research Fellow
	Neeraj Kumar	Junior Research Fellow (since July 2016)
	Yogesh Patidar	Junior Research Fellow (since Feb. 2017)
Other Members	V K Mishra	Staff Scientist (till June 2016)
	K Anupama	Staff Scientist (till Aug. 2016)
	J Krishna Leela	Technical Officer
	T S Shaffiqu	Technical Officer
	Vimala Allada	Research Associate
	P Hima Bindu	Research Associate
	Saswat Mohapatra	Research Associate (till Aug.2016)
	Soni Priya Valeru	Research Associate (since Aug. 2016)

The Laboratory of Bacterial Genetics comprises three research groups engaged in investigations on several aspects of the physiology and genetics of *Escherichia coli,* and is majorly supported by the Department of Biotechnology as a Centre of Excellence for Microbial Biology. The work undertaken in this reporting year is described below under the following objectives:

Objectives

1. Occurrence of pathological R-loops and their consequences;
2. Essentiality and oligomerization features of RNase E;
3. The PtsP-PtsO-PtsN phosphorelay and potassium (K+) metabolism;
4. Studies on basic amino acid export*;*
5. To understand the role of basal (p)ppGpp in the growth rate dependent modulation of cell division;
6. Studies on the consequences of SpoT depletion;
7. Genetic and molecular characterization of the glycerol induced growth stasis in the *glpD* mutant;

Summary of work done until the beginning of this reporting year (up to March 31, 2016)

The work undertaken in earlier years on each of the objectives has been summarized in the first parts of the corresponding description below.

Details of progress made in the current reporting year (April 1, 2016 – March 31, 2017)

Several years ago, this laboratory was the first to propose that nascent transcripts in all living systems are prone to annealing with the upstream template DNA strand to generate toxic RNA-DNA hybrids or R-loops, and that mechanisms of co-transcriptional engagement of the mRNA by various proteins have

accordingly been selected in evolution to prevent R-loop formation. According to this model, in bacteria such as *Escherichia coli*, it is the binding of ribosomes to the nascent transcripts (i.e., transcription-translation coupling, which is a defining feature of the prokaryotic lifestyle) that protects them from annealing with the DNA. We had further proposed that the process of Rho-dependent transcription termination (RDTT) in *E. coli* (which is mediated by the proteins Rho and NusG) also serves to reduce R-loop occurrence, since RDTT acts to terminate the synthesis of transcripts which are not being simultaneously translated.

In support of this model, subsequent work from our group has shown that the lethality of knock-out mutations in *rho* or *nusG* in *E. coli* can be rescued by the ectopic expression of UvsW, an R-loop helicase of T4 phage, implying that these mutants are inviable solely because of excessive R-loops in them. Furthermore, we had determined the distribution of R-loops by a next-generation-sequencing (NGS) approach and identified more than 75 hotspots of their occurrence across the genome; many of these hotspots were several kb long and included regions of both sense and antisense transcription. Finally, we have also been interested in studying one of the pathological consequences of R-loop formation, namely the aberrant initiation of chromosomal DNA replication from the R-loop sites, which is referred to as constitutive stable DNA replication (cSDR).

In the current year, we have undertaken investigations with respect to two aspects of this project: (i) the inter-relationships between antisense transcription, RDTT, and R-loop formation; and (ii) the features and mechanisms of cSDR. Each of them is briefly described below.

It had earlier shown by another group that a major target of RDTT in *E. coli* is antisense transcription (which, by definition, is not translated), and they had also identified the large number of antisense RNAs that are synthesized upon Rho inhibition. As mentioned in last year's Report, we had compared their findings with our genome-wide R-loop mapping results to discover an unexpected inverse correlation between the two data sets, that is, the regions with substantial antisense transcription exhibited less R-loops, and vice versa. This finding was counterintuitive, since in our model R-loops are expected to be generated from untranslated nascent mRNAs such as antisense transcripts. To explain these observations, we had then postulated that an antisense transcript at a very highly R-loop prone locus would immediately form an R-loop and inhibit further transcription at the locus, so that the abundance of detected antisense transcripts at this locus would be minimal.

In the current year, we have tested one major prediction of our model, namely that the 'hidden' R-looped antisense loci will be revealed by the combination of RDTT inhibition and R-loop helicase expression. Accordingly, we have performed NGS RNA-Seq experiments (in collaboration with Prof. Philippe Bouloc) in delta-*rho* and delta-*nusG* mutants expressing the UvsW helicase. The results of these experiments are completely consistent with the proposed hypothesis, and we have identified more than 200 new antisense loci across the *E. coli* genome that are expressed only in the situation where both RDTT is absent and an R-loop helicase is expressed; these loci are also the ones that were identified as R-looped in our earlier studies.

Thus, our present work allows us to conclude that in E.*coli* strains in which both constraints are relaxed (RDTT, and R-loop formation), antisense transcription occurs from > 50% of genes, and that they account for around 22% of all non-rRNA transcript abundance in the cells. Accordingly, we refer to this phenomenon as the "dark matter of bacterial antisense transcription". The corollary also is that lethality is associated not with excessive antisense transcription per se, but with the R-loops that are being formed from such transcripts.

With reference to cSDR (that is, aberrant chromosomal replication initiation), one experimental hallmark of the phenomenon is its ability to confer viability to mutants that are defective for DnaA-mediated replication initiation at *oriC* (e.g., to *dnaA* mutants). By this criterion, other investigators have demonstrated cSDR in *E. coli* strains deficient for RNase H1 or RecG (which remove R-loops by hydrolysis or unwinding, respectively), and the presence of some additional mutations (*tus* and *rpoB*35*), which are expected to resolve the impediments associated with replication proceeding in the "wrong" direction around the circular chromosome, have been shown to improve the efficacy of cSDR.

Another distinctive feature of cSDR in the RNase H1- or RecG-deficient mutants is that, when they are DnaA-proficient, they exhibit a characteristic "mid-terminus peak" in marker frequency analysis experiments, which we have attributed to the low-frequency, stochastic, genome-wide distribution of aberrant replication initiation sites in the population. It may also be noted that some other groups have offered explanations alternative to R-loop formation for the occurrence of cSDR in *recG* mutants, including replication initiated either from replication fork collisions, or in the retrograde direction from double strand break repair events.

In the current year, we have identified two additional and novel instances of cSDR. The first is with different combinations of mutations in the following DNA exonucleases: exonucleases I, V, and VII, SbcCD and RecJ. The second is in the absence of Dam methylase, which is involved in methyl-directed mismatch repair. In both cases, we have observed the signature "mid-terminus peak" in NGS experiments of marker frequency analysis. The combined results from other experiments with these strains appear to suggest that the former cSDR may be mediated by R-loops and the latter by double strand break repair. We have also shown that mutation in *rho* can contribute to cSDR in the former instance.

Essentiality and oligomerization features of RNase E

The enzyme RNase E is essential for *E. coli* viability, and exists as a dimer of homodimers of a polypeptide whose length is a little over 1000 amino acid residues. Its N-terminal half (NTH) possesses (i) the catalytic site for endoribonucleolytic activity, as well as (ii) a "5'-sensor" pocket that renders the enzyme most active on RNA substrates bearing a 5'-terminal monophosphate. The non-catalytic C-terminal half (CTH) of RNase E, which is dispensable for viability, is intrinsically unstructured and serves as the scaffold for assembly of a multi-protein complex called the degradosome. The reason for RNase E's essentiality is unclear, and it has been variously suggested that it stems from the need of its activity for mRNA degradation, for tRNA maturation, for rRNA processing, and so on.

In work reported last year, we had shown that inviability associated with reduced RNase E activity can be rescued by reduction in stable RNA levels in the cell, which could be achieved by perturbations such as increase in basal ppGpp levels, overexpression of protein DksA, introduction of "stringent" RNA polymerase mutations, or reduction in genomic ribosomal RNA operon copy number from seven to two. Accordingly, we had advanced the suggestion that the reason for RNase E's essentiality is indeed joint and several, such that if in cells with limiting enzyme activity the need for stable RNA processing is reduced then sufficient activity would still be available for mRNA degradation and hence for viability.

In the present year, additional experiments were undertaken to confirm this model and to exclude alternative explanations. Thus, we showed the stringent RNA polymerase mutations which rescued viability of strains with limiting RNase E were neither associated with increased RNase E polypeptide levels (as determined by Western blotting) nor with alteration of *rne-lac* expression. The growth rescue of strains with limiting RNase E occurred only with perturbations that lead to reduced stable RNA, but not with other pertubations that non-specifically reduced the growth rate, such as mutations in *crp* or *hfq*, or with sub lethal concentrations of rifampicin. Finally, we have also shown deletions of the CTH of RNase E beyond residue 494 or beyond residue 530 (the latter corresponds to the polypeptide for which the X-ray crystal structure has been determined) behave identically with respect to the various phenotypes described above.

We had also reported last year an example of apparent inter-subunit complementation in RNase E. In this case, two variant RNase E polypeptides – one with an R169Q mutation that abolishes 5'-end sensing, and the other with a D346A catalytic active site substitution – which are individually lethal were able to nevertheless confer viability when co-expressed. We had suggested that these results provide confirmation for the model derived from the enzyme's crystal structure that RNA 5'-end recognition and cleavage are distinct properties which are spatially separated in different subunits of the oligomer.

In the current year, we have shown that such inter-subunit complementation confers viability even under extremely stringent conditions such as very low basal ppGpp levels and loss of the paralogous enzyme RNase G. Furthermore, viability is retained even when the polypeptides

bearing the individual 5'-sensor and active site mutations are only 395 amino acids long, that is, without the small-domain interactions or the "zinc-link" that contribute to oligomer assembly. Our results therefore indicate that non-covalent interface interactions between a pair of large domains are sufficient for productive oligomer assembly of RNase E. In control experiments, we have also shown that if both substitutions R169Q and D346A are borne on a single polypeptide, cells expressing such an RNase E variant are inviable.

Finally, we have also obtained evidence that RNase E overexpression (even of a variant bearing the active site mutation D346A) is lethal, and that this lethality is associated primarily with the intrinsically unstructured CTH region of the polypeptide. We speculate that the CTH region undergoes toxic aggregation in the bacterial cytoplasm, akin perhaps to that described for amyloidogenic or prionogenic proteins in eukaryotic cells.

The PtsP-PtsO-PtsN phosphorelay and potassium (K^+) metabolism

Earlier studies in this project have examined a physiological link between the phosphoenol pyruvate dependent phosphotransferase system comprising PtsP-PtsO-PtsN and K^+ ion metabolism in *E. coli*. These studies have delineated the basis behind a potassium sensitive growth phenotype (K^S) displayed by a deficiency of PtsN, the terminal phospho-acceptor of the PtsP-O-N phosphorelay, as the external K^+ concentration ($[K^+]_e$) is increased above 1 mM. Genetic and physiological studies on the K^S have shown that the K^S is associated with cellular K^+ limitation that is mediated by YcgO, a predicted inner membrane protein belonging to the CPA1 family of proteins mediating monovalent cation/ proton antiport. Additional studies implicate the involvement of dephospho-PtsN as a negative regulator of YcgO.

Overall our studies are consistent with a model which postulates that K^S in the Δ*ptsN* mutant occurs due to K^+ limitation resulting from unfettered K^+ efflux mediated by YcgO, owing to the absence of dephospho-PtsN with K^+ efflux being additionally stimulated by $[K^+]_e$. Repression of the high affinity Kdp K^+ uptake system by $[K^+]_e$ is thought to contribute to the maintenance of K^+ limitation in the Δ*ptsN* mutant. It is speculated that YcgO mediated K^+ limitation may be an output of a response to certain stress(es) which by modulating the phospho-transfer capacity of the PtsP-O-N phosphorelay, leads to growth cessation and stress tolerance.

Earlier, we had also described the characterization of a chromosomal suppressor mutation of the K^S of the Δ*ptsN* mutant obtained after transposon mutagenesis and reported that the absence of a small integral membrane protein YajC alleviated the K^S. Additional studies have indicated that the Δ*yajC* mutation exerts its suppressive effect only in the absence of PtsN and does not ordinarily perturb cellular K^+ content.

Our identification of involvement of YajC in mediating the K^S of the Δ*ptsN* mutant was based upon the isolation of the *yajC** allele that suppressed the K^S. *yajC** represents a transposon insertion in *yajC* that led to a complex phenotype, namely that *yajC** in combination with the Δ*ptsN* mutation (i) displayed a requirement for K^+ in media containing low K^+, and (ii) alleviated the K^S. Dissection of this dual phenotype has revealed that the former is related to impaired expression of the *secD*/*secF* genes located in the same operon as and downstream of *yajC,* whereas the latter occurs purely due to the absence of YajC.

Additional studies have indicated that damped down SecD/SecF activity alone also mediates suppression of the K^S. As described earlier, a non-polar knock out of *yajC* (Δ*yajC*), unlike the *yajC** allele, only caused suppression of the K^S and substantially suppressed the K^S of YcgO overproduction that is known to correlate with K^+ limitation. Furthermore YcgO levels remained unaltered in the Δ*yajC* background. *trans*-dominant mutations have been isolated in *yajC* whose conditional expression suppressed the K^S, and an additional category of K^S suppressing *trans*-dominant *yajC* alleles were also recovered whose phenotypes are equivalent to those resulting from damped down SecD/SecF activity.

These observations are best explained under a scenario which postulates that YajC may function as a positive regulator of YcgO and the SecD/SecF proteins modulate the K^S in a YajC independent fashion. The isolation of *trans*-dominant *yajC* alleles that mediate damping of SecD/SecF activity adds credence to the notion that YajC additionally may participate in protein secretion, perhaps in a redundant manner with SecD and SecF, a notion that has hitherto remained genetically unsubstantiated.

Current studies are directed towards testing the notion that YajC may interact with YcgO, and this is being tested by two-hybrid analyses and co-purification studies. For the latter, a functional epitope tagged version of YajC has been constructed. In addition, cysteine substituted versions of YajC have been constructed which will aid in determination of YajC topology as also to obtain topological correlates of amino acid substitutions in YajC yielding a *trans*-dominant phenotype.

Studies on basic amino acid export

Towards studies on regulation of basic amino acid export in *E. coli,* we have previously reported genetic and physiological studies on the ORFs *yggA* and *ybjE* encoding, respectively, the L-arginine (Arg) and L-lysine (Lys) exporters ArgO and LysO. In addition, the delineation of the topology of ArgO in the cytoplasm, residues important for ArgO function and the inference that the functional state of ArgO in vivo is a monomer, arrived at from intragenic suppressor studies, has also been reported.

Prior studies from another laboratory had shown that *Corynebacterium glutamicum* lacking LysE, the ortholog of *E. coli* ArgO is rendered sensitive to L-arginylalanine (Arg-Ala) and L-lysylalanine (Lys-Ala) dipeptides with the sensitivities correlating with increased intracellular levels, respectively, of Arg and Lys that are thought to be growth inhibitory. This phenotype of the *C. glutamicum* Δ*lysE* mutant is thus compatible with its role as an Arg/Lys exporter. On the other hand, we had previously observed that in *E. coli,* absence of ArgO did not lead to sensitivity to Arg-Ala whereas absence of LysO caused sensitivity to Lys-Ala. This observation suggested that *E. coli* may possess additional mechanism(s) to mitigate the potential toxicity arising due to elevated intracellular level of Arg following the catabolism of Arg-Ala into its constituent amino acids after its uptake into the cytoplasm.

Towards uncovering the genetic basis of resistance to Arg-Ala displayed by an *argO* mutant, we had earlier reported the isolation of transposon insertions in *ydhE* encoding an inner membrane protein belonging to the multidrug and toxin extrusion (MATE) family, which rendered an *argO* mutant extremely sensitive to Arg-Ala. Further studies have shown that the Arg-Ala sensitive (RA^S) phenotype correlated with the absence of YdhE.

In addition, we noted that expression of an ortholog of YdhE, NorM from *Vibrio cholerae,* complemented the RA^S phenotype, indicating that the capacity to mediate resistance to Arg-Ala may be common to orthologs of YdhE. Closer examination revealed that to a large extent the RA^S phenotype resulted from absence of YdhE that was accentuated by the *argO* mutation. Furthermore, absence of YdhE did not lead to a discernible sensitivity to canavanine, an Arg antimetabolite, implying that unlike ArgO, YdhE may not play any role in mediating Arg export. Circumstantial evidence indicated that the RA^S phenotype of the *argO ydhE* double mutant did not occur due to elevated intracellular levels of Arg but was specific to Arg-Ala, as the *argO ydhE* double mutant was not inhibited by the presence of the L-alanylarginine (Ala-Arg) dipeptide in the medium. In addition, it was found that the RA^S phenotype could be partially alleviated by the presence of 20 amino acids in the medium.

In order to delineate the physiological defect in the *argO ydhE* double mutant causal to its RA^S phenotype, suppressor studies were performed which showed that a variety of recessive genetic lesions in *tppB,* encoding the di-tripeptide permease, suppressed the RA^S phenotype. The property of TppB to mediate preferential uptake of dipeptides bearing a positively charged amino acyl R group at the N-terminus, provides a rationale to account for the suppression of the RA^S phenotype by mutations in *tppB*. Based on these studies it is suggested that YdhE may mediate export of Arg-Ala and that ArgO may also contribute to the export. Furthermore, it is speculated that Arg-Ala may serve as a proxy for an as yet unknown, naturally occurring substrate for YdhE (and ArgO), probably an antimicrobial compound. The physiological defect causal to the RA^S phenotype of the *argO ydhE* double mutant is under further investigation.

To understand the role of basal (p)ppGpp in the growth rate dependent modulation of cell division

Previous work from this laboratory has shown that basal (p)ppGpp contributes to the regulation of cell division by positively regulating the level of FtsZ, the structural protein involved in septum formation. This regulation, which is not essential for the maintenance of cell division under normal growth conditions, is essential for septum formation in absence of the Lon protease. The latter synthetic phenotype arises consequent

to increased activity of the SulA protein which is an inhibitor of FtsZ function and is normally degraded by the Lon protease. In a related study, it was observed that null mutation in the (p)ppGpp synthase gene *relA* confers synthetic growth defect in the presence of the hypomorphic *ftsZ84* allele. Based on these phenotypes, a genetic study was initiated to decipher the role of (p)ppGpp in the modulation of cell division.

Since FtsZ protein levels were reduced in the strain lacking (p)ppGpp, in order to study *ftsZ* expression *ftsZ-lacZ* reporter fusions (operon and gene fusion) were made on the genome. FtsZ being an essential gene, these fusions were made in the presence of the plasmid encoded *ftsZ*. B-galactosidase assays done in the wild type and ppGpp^0 strain show a 30% reduction in activity. Further work is in progress to use the fusions to study the reported positive regulation by (p)ppGpp and the role of other factors, if any, that contribute to the regulation. A collection of genetic suppressors of the *relAftsZ84* growth defect were identified by transposon mutagenesis or by using a plasmid over-expression library. Our studies show that both the *relA ftsZ84* and the *relA lon* synthetic growth defects are restricted to fast growth conditions which suggests that there could a common mechanistic basis for the two defects. Studies are in progress to make use of the genetic suppressors identified to address this question.

Studies on the consequences of SpoT depletion

By cloning the *spoT* gene under an inducible promoter in a plasmid and modulating its expression in the Δ*spoT* strain it was confirmed that depletion of SpoT was associated with growth inhibition. SpoT protein is capable of (p)ppGpp synthesis and hydrolysis and the latter activity is essential for growth. Experiments were done to monitor the accumulation of (p)ppGpp in the Δ*spoT* /pRCspoT strain during the course of SpoT depletion. It was observed that, during growth in rich medium, associated with SpoT depletion, there was a concomitant increase in ppGpp, but no pppGpp was detectable; an increase in the doubling time corresponding with an increase in the cellular ppGpp levels was also observed. The absence of pppGpp accumulation suggested that the GppA (guanosine penta phosphate hydrolase) activity that converts pppGpp to ppGpp may be stimulated during SpoT depletion. We therefore asked if the adaptations associated with changes in the SpoT activity were perturbed in the *gppA* mutant background. It was reported that the increase in basal (p)ppGpp level during down-shift and carbon starvation are mediated by changes in SpoT activity. However, we did not observe any significant difference between the wild type and the *gppA* mutant in terms of their growth response to these changes. These results suggest that the absence of pppGpp accumulation during SpoT depletion may not arise from GppA activation.

We had previously observed that the GppA activity is required to alleviate the growth inhibition arising from the loss of SpoT activity, and that this was the case even in the presence of the hypomorphic *relA* alleles that were isolated as suppressors of Δ*spoT* lethality. Further, our studies show that a reduction in SpoT hydrolase activity made GppA function indispensable for growth. These results indicated that it was essential to keep the level of pppGpp (but not ppGpp) low in the cell in order to sustain growth and that this was accomplished through the combined hydrolase activities of SpoT and GppA. Following the provocation of stringent response using valine, the accumulation of ppGpp (without pppGpp) and growth arrest was seen in the *rlmD*::FRT and the *rlmD*::FRT Δ*spoT* strains, and interestingly, growth resumed following the addition of isoleucine in the latter strain after a lag despite the continued presence of ppGpp indicating that the latter molecule cannot solely produce growth arrest (Figure 1). Preliminary results suggest the reduction in pppGpp level (relative to ppGpp) could be due to the reduced RelA– dependent synthesis of the molecule. Prior studies had implicated ppGpp (as compared to pppGpp) as the more potent inhibitor of functions associated with the stringent response that leads to growth arrest. Our results are consistent with the idea that, during growth in rich medium, there is a constant turnover of (p)ppGpp through the RelA-dependent synthesis and the SpoT mediated degradation. The reason for what seems like a futile cycle is unclear and is being investigated.

Genetic and molecular characterization of the glycerol induced growth stasis in the *glpD* mutant.

It has been reported that the addition of glycerol or glycerol-3-P induced growth arrest in the *glpD* mutant of *E. coli* with a concomitant decrease in the levels of nucleotides; the molecular basis of this effect remains unclear. We had found that the

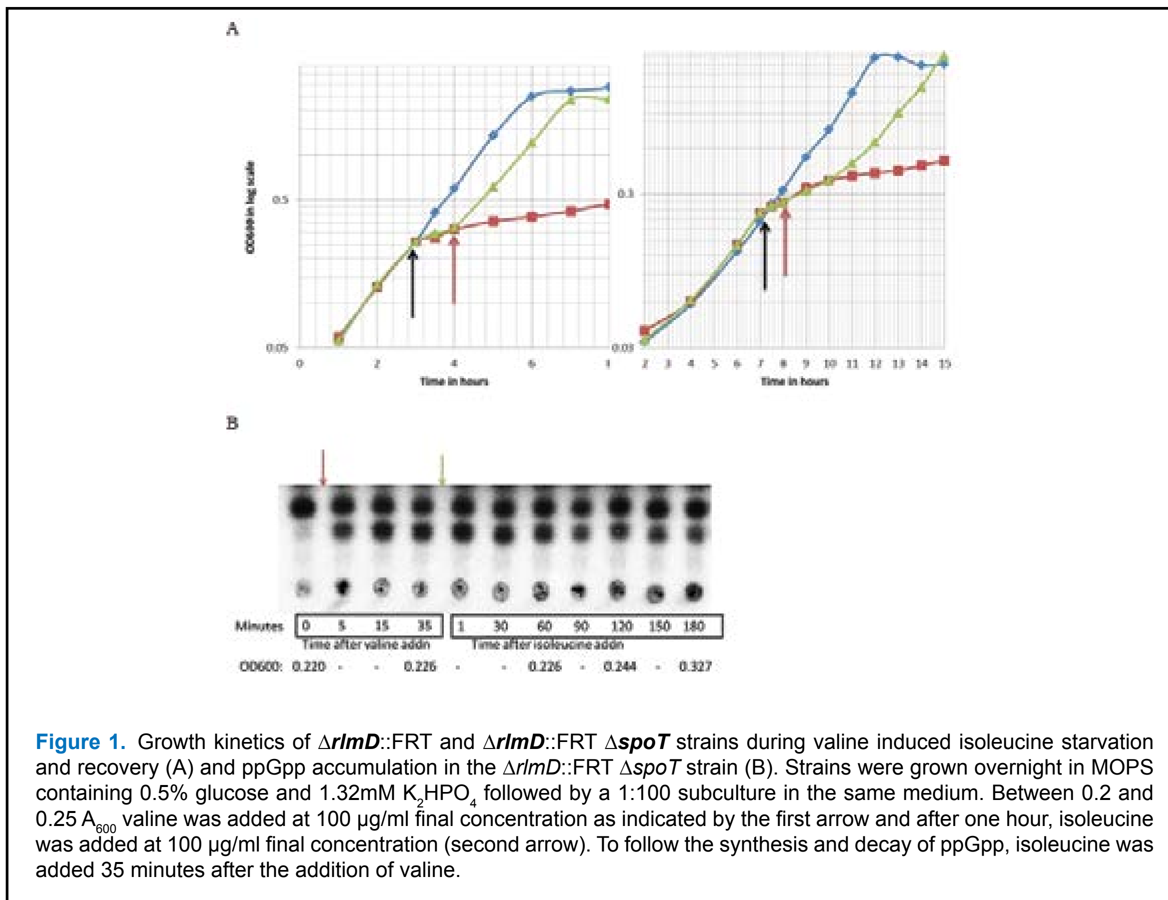

Figure 1. Growth kinetics of Δ***rlmD***::FRT and Δ***rlmD***::FRT Δ***spoT*** strains during valine induced isoleucine starvation and recovery (A) and ppGpp accumulation in the Δ*rlmD*::FRT Δ*spoT* strain (B). Strains were grown overnight in MOPS containing 0.5% glucose and 1.32mM K_2HPO_4 followed by a 1:100 subculture in the same medium. Between 0.2 and 0.25 A_{600} valine was added at 100 µg/ml final concentration as indicated by the first arrow and after one hour, isoleucine was added at 100 µg/ml final concentration (second arrow). To follow the synthesis and decay of ppGpp, isoleucine was added 35 minutes after the addition of valine.

growth arrest induced can be rescued by ribose or pyrimidine nucleosides through the synthesis of ribose-5-P and phosphoribosylpyrophosphate (PRPP). In this reporting period we have studied the kinetics of the nucleotide and PRPP perturbation during glycerol or glycerol-3-P induced stasis in the *glpD* mutant and in the *glpD* mutant with constitutive *glpK* expression (*glpK*^{C}) or GlpK activity that is insensitive to feed-back inhibition. The findings can be summarized as follows.

(i) A decrease in the level of the purine nucleotides and PRPP was evident, however, there was no perceptible drop in the level of the pyrimidine nucleotides.

(ii) Following the addition of glycerol the drop in PRPP level was almost instantaneous while the decrease in the level of the purine nucleotides was evident after a lag of about 30 minutes. The drop in PRPP level was not instantaneous during glycrol-3-P induced stasis.

(iii) In the *glpDglpK*^{C} mutant where the glycerol induced stasis is accentuated over and above that seen in the *glpD* mutant the restoration of the PRPP pool by glucose was also delayed as compared to that seen in the *glpD* mutant.

Based on these results we propose that the growth stasis induced by glycerol is caused by the inhibition of PRPP synthesis, which leads to a decrease in the purine nucleotide pool. This could be due to the inhibition of PRS (PRPP synthase) activity following the depletion of ATP and the accumulation of ADP from the unfettered GlpK activity. The same cannot be said for the glycerol-3-P induced stasis as the decrease in PRPP pool is concomitant with that of the nucleotides.

Since glycerol induced stasis was proportionate to the GlpK activity, genetic studies were carried out to find out the factors that modulate this activity. The following could be summarized from these studies, (i) GlpF (glycerol facilitator) activity was required for the glycerol induced stasis, indicating that the GlpK activity could be positively regulated by GlpF; (ii) the regulation of the GlpK activity by GlpF was not seen when *glpK* was expressed from a non-native promoter; (iii) the positive regulation of GlpK function by GlpF

required the co-transcription of the two genes; (iv) when *glpK* expression was independent of catabolite repression and the GlpK activity independent of the fructose 1,6 bisphosphate mediated feed-back inhibition, glucose continued to rescue the glycerol induced stasis, suggesting that glucose rescue operates independent of the above two regulations.

Publications

1. Pathania A, Gupta A, Dubey S, Gopal B, and Sardesai AA. (2016). The topology of the L-arginine exporter ArgO conforms to an N_{in}-C_{out} configuration in *Escherichia coli*: Requirement for the cytoplasmic N-terminal domain, functional helical interactions and an aspartate pair for ArgO function. ***Journal of Bacteriology*** 198: 3186-3199.

2. Sharma R, Shimada T, Mishra VK, Upreti S, and Sardesai AA. (2016). Growth inhibition by external potassium of *Escherichia coli* lacking PtsN ($EIIA^{Ntr}$) is caused by potassium limitation mediated by YcgO. ***Journal of Bacteriology.*** 198: 1868-1882.

3. Vimala A and Harinarayanan R (2016). Transketolase activity modulates glycerol-3-phosphate levels in *Escherichia coli*. ***Molecular Microbiology*** 100: 263-277.

4. Nazir A and Harinarayanan R (2016). (p) ppGpp and the bacterial cell cycle. ***Journal of Bioscience*** 41: 277-282.

Laboratory of Cell Cycle Regulation

Elucidating the role of effector proteins in G1 to S phase progression

Faculty	Shweta Tyagi	Staff Scientist
Ph D Students	Aamir Ali	Senior Research Fellow
	Zaffer Ullah Zargar	Senior Research Fellow
	Swathi Chodisetty	Senior Research Fellow
	Amit Mahendra Karole	Senior Research Fellow
	Kausika Kumar Malik	Senior Research Fellow
	Akash Nitin Chinchole	Junior Research Fellow
	Kaiser Ahmed Lone	Junior Research Fellow
Other Members	V N Sailaja	Technical Officer
	Mallikharjuna Rao	Project JRF (till May 2016)

Objectives

1. Identification of new effector proteins involved in regulation of E2F-responsive promoters.
2. Study of chromatin modifying proteins in cell cycle regulation.

Project 1: Identification of new effector proteins involved in regulation of E2F-responsive promoters.

One of the major roles of E2F proteins is to regulate the transition from G1 to S phase. However, how E2Fs affect passage into S phase is still poorly understood. In this project we aim to identify new effector proteins involved in regulation of E2F-responsive promoters and better understand how these effectors influence transcriptional regulation during G1 to S phase progression.

Summary of work done until the beginning of this reporting year (up to March 31, 2016)

We showed that RBP2 interacts with pocket protein p130 and this interaction is dependent on the LxCxE motif in RBP2.

Details of the progress made in the current reporting year (April 1, 2016 –March 31, 2017)

RBP2 has been shown to bind to E2F-responsive promoters during differentiation (Beshiri et al., Proc. Natl. Acad. Sci. 2012; van Oevelen et al., Mol. Cell. 2013). To extend this observation to dividing cells, we asked whether RBP2 associated with E2F-responsive promoters during the cell cycle. As the association of RBP2 with p130 and E2F4 is primarily seen in early G1(data not shown), we used cells from two cell-cycle stages for performing our chromatin immunoprecipitation (ChIP) experiments—early G1, where these promoters are inactive due to repressive E2Fs binding, and G1/S phase, where these promoters are active and repressive E2Fs are displaced by activating E2Fs (Takahashi et al., Genes Dev. 2000). We selected 6 E2F-regulated promoters that have been studied before by others. For negative control, we used U2 snRNA gene ($U2_C$). We also analyzed two mitochondrial promoters to which RBP2 binds, but these promoters are not known to be E2F-responsive or cell cycle regulated—ATP50 and MTRF1. Consistent with previous reports, we observed that association of E2F4 and p130 proteins on these E2F-responsive promoters was prominent in early G1 while E2F1 protein showed binding predominantly in G1/S fraction (Figure 1A). Consistent with our hypothesis, RBP2 bound these promoters primarily in early G1.

Previously we have shown that H3K4me3 was deposited on E2F-responsive promoters in G1/S and S phase, by recruitment of H3K4 HMTs in these cell-cycle phases, to activate transcription (Tyagi et al., Mol. Cell. 2007). In accordance with previous results, we observed high fold enrichment of H3K4me3 mark on E2F-responsive promoters in G1/S over early G1 samples (Figure 1B).

Our previous results suggest that RBP2 may be recruited to E2F-responsive promoters by p130 to erase the H3K4me3 mark and prepare the promoters for next cycle of activation. If this

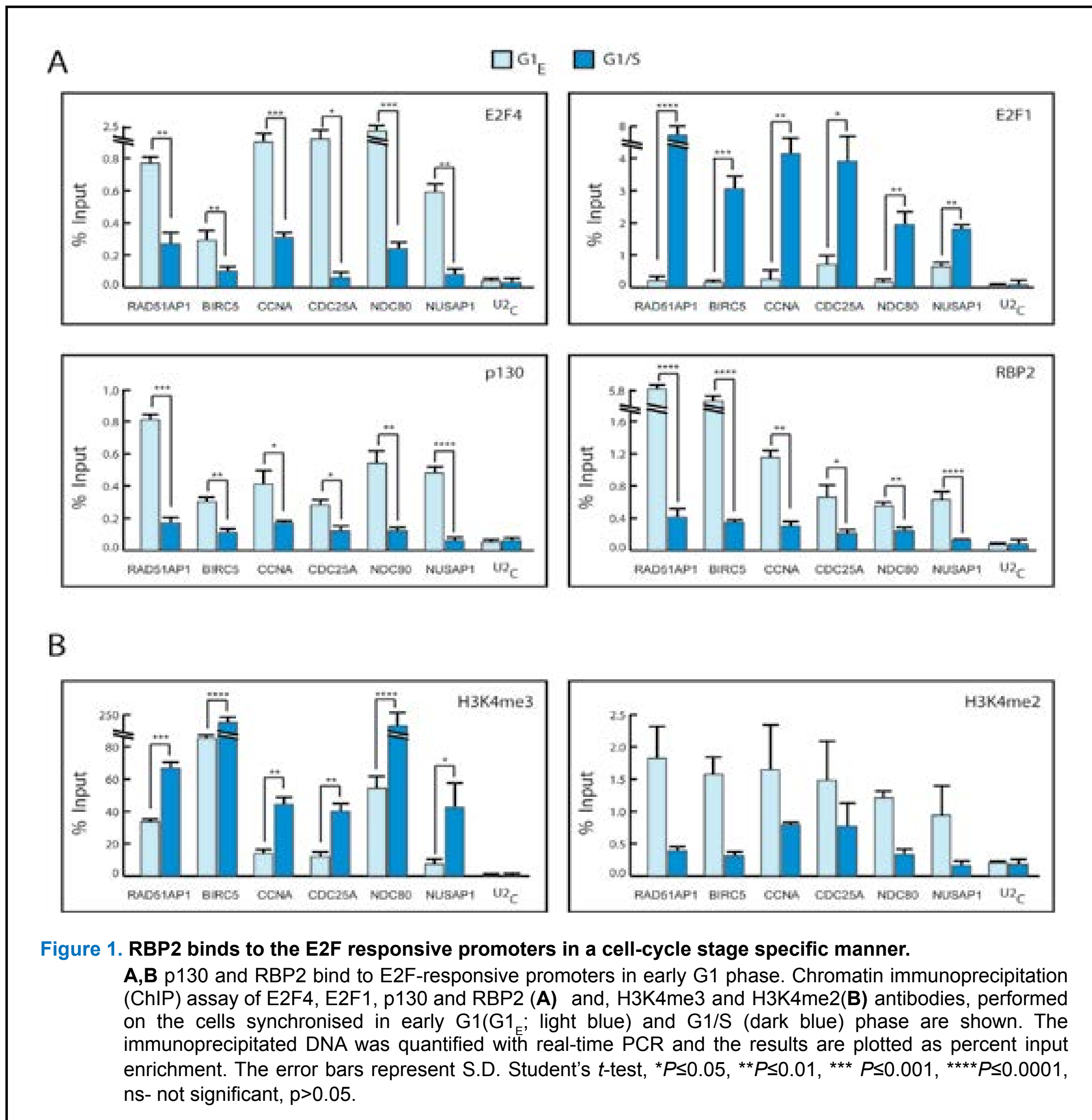

Figure 1. **RBP2 binds to the E2F responsive promoters in a cell-cycle stage specific manner.**
A,B p130 and RBP2 bind to E2F-responsive promoters in early G1 phase. Chromatin immunoprecipitation (ChIP) assay of E2F4, E2F1, p130 and RBP2 (**A**) and, H3K4me3 and H3K4me2(**B**) antibodies, performed on the cells synchronised in early G1($G1_E$; light blue) and G1/S (dark blue) phase are shown. The immunoprecipitated DNA was quantified with real-time PCR and the results are plotted as percent input enrichment. The error bars represent S.D. Student's *t*-test, **P*≤0.05, ***P*≤0.01, *** *P*≤0.001, *****P*≤0.0001, ns- not significant, p>0.05.

hypothesis is correct then loss of p130 by RNAi should lead to loss of RBP2 recruitment to E2F-responsive promoters during the early G1 phase. We put our hypothesis to test by depleting p130 in HeLa cells using shRNA, synchronizing them in early G1 and performing ChIP with these cells. As shown in Figure 2A, p130 shRNA transfection depleted majority of p130 protein. As a consequence, the p130 binding on E2F-responsive promoters was also reduced (Figure 2B).

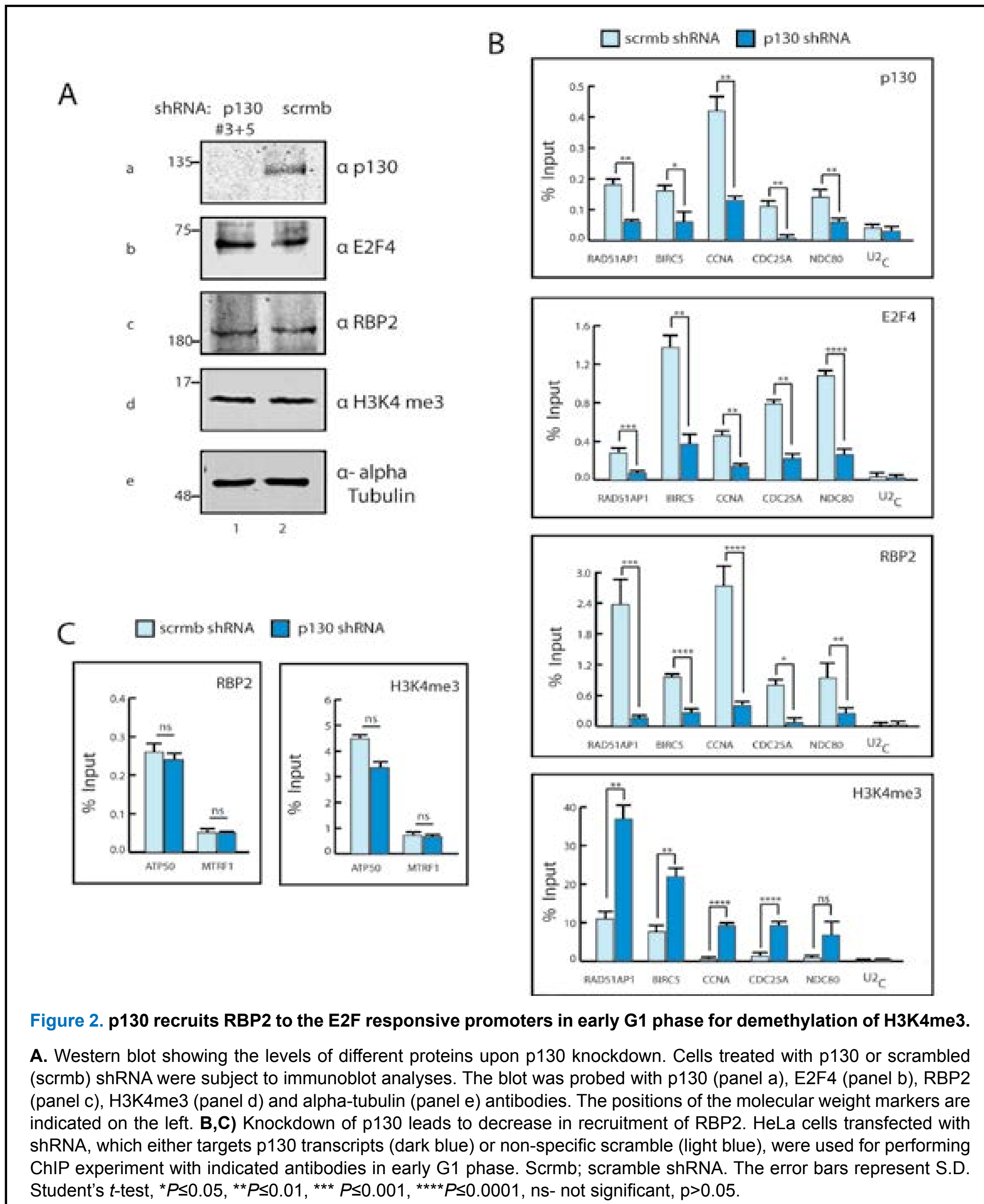

Figure 2. **p130 recruits RBP2 to the E2F responsive promoters in early G1 phase for demethylation of H3K4me3.**

A. Western blot showing the levels of different proteins upon p130 knockdown. Cells treated with p130 or scrambled (scrmb) shRNA were subject to immunoblot analyses. The blot was probed with p130 (panel a), E2F4 (panel b), RBP2 (panel c), H3K4me3 (panel d) and alpha-tubulin (panel e) antibodies. The positions of the molecular weight markers are indicated on the left. **B,C)** Knockdown of p130 leads to decrease in recruitment of RBP2. HeLa cells transfected with shRNA, which either targets p130 transcripts (dark blue) or non-specific scramble (light blue), were used for performing ChIP experiment with indicated antibodies in early G1 phase. Scrmb; scramble shRNA. The error bars represent S.D. Student's *t*-test, **P*≤0.05, ***P*≤0.01, *** *P*≤0.001, *****P*≤0.0001, ns- not significant, p>0.05.

Consistent with our hypothesis, there was analogous decrease in the RBP2 binding to these promoters. However we also observed a decreased binding of E2F4 on these promoters. It has been shown that the nuclear localization of E2F4 is impaired in absence of p130 (Lindeman et al., Proc. Natl. Acad. Sci.1997) and this can be a reason for low E2F4 binding. In any case, this experiment proves our hypothesis where E2F4 and p130 recruit RBP2 to E2F-responsive promoters, and RBP2 removes the H3K4me3 mark to reset the E2F-responsive promoters and repress transcription. Consistent with the latter, and decreased RBP2 binding, H3K4me3 mark was significantly increased on E2F-responsive promoters, but not globally (Figure

2B and 2A panel d). Our results indicate that just like acetylation marks, H3K4me3 also needs to be actively removed during the cell cycle progression.

We also analyzed the non-E2F-responsive promoters ATP50 and MTRF1. ATP50 and MTRF1 did not show any significant variation in RBP2 binding in control vs. knockdown samples (Figure 2C). Similarly, the H3K4me3 levels were largely unaffected on these promoters upon p130 knockdown (Figure 2C). These results indicate that p130 is engaged in recruitment of RBP2 to E2F-responsive promoters specifically and recruitment of RBP2 to non-E2F-responsive promoters may be carried out in different manner.

Project 2: Study of chromatin modifying proteins in cell cycle regulation.

Histone 3 lysine 4 trimethylation is linked to active gene expression, but its precise role in cell cycle regulation is now being uncovered. In this project, we are looking at other roles of this chromatin-modifying complex that may influence cell cycle regulation.

Summary of work done until the beginning of this reporting year (up to March 31, 2016)

We showed that both subunits of MLL—MLL_N and MLL_C as well as core components of MLL complex like WDR5 and RbBP5 localize to spindle apparatus throughout mitosis.

Details of the progress made in the current reporting year (April 1, 2016 –March 31, 2017)

We previously observed prolonged pro-metaphase in MLL- and WDR5-depleted cells which indicates a defect in chromosome congression and/ or in attachment of chromosomes to mitotic-spindle microtubules (MTs). These defects may also culminate in the formation of micronuclei, as reported previously (Ali et al., Nucleic Acids Res. 2014). We assessed the distribution of chromosomes in mitotic cells by IFS in control, MLL, or WDR5 siRNA-treated cells. Among MLL and WDR5 siRNA-treated cells, we observed an increase in number of cells with late pro-metaphase like chromosome arrangement in which a partial metaphase plate had formed, but many chromosomes were dispersed away from the metaphase plate (Figure 3A, B compare panels a, d with b, e). Given the general role of MLL in transcription, including regulation of genes involved in DNA synthesis and replication, the observations made here raise the possibility of cells undergoing mitosis with under- or un-replicated genome. However, upon CENPA staining we observed intact chromosomes with attached sister chromatids lying away from metaphase plate thus ruling out the above mentioned scenario (Figure 3C). When quantified, approximately 80% of MLL or WDR5 depleted cells had difficulty in aligning chromosomes in a tight metaphase plate (Figure 3D graph a).

We also observed defects in the mitotic spindle in MLL and WDR5 siRNA-treated cells (Figure 3A, B panels b, c, e, f see α-tubulin staining). Instead of the continuous fusiform shape seen in control cells, the spindle apparatus was either i) very long with dense MTs (Figure 3A, B panel b, e, Figure 3D), or ii) exhibited spindles with MTs of poor intensity (Figure 3A, B panel c, f). When the inter-polar distance was measured for all cells, MLL and WDR5 siRNA treated cells displayed longer spindles when compared to control siRNA treated cells (Figure 3F). Over all, we could determine that about 82% of MLL and 65% of WDR5 siRNA-treated cells had problems in the mitotic spindle (low MTs or MT-rich long spindle; Figure 3D graph b). About 10% of these cells also showed multipolar spindles (Figure 3B panel f, 3D graph c).

As both abnormal spindle conditions (poor spindle or MT-rich long spindle) displayed misaligned chromosomes in MLL or WDR5 depleted cells, we decided to study this phenotype further. In order to discern the region of MLL required for the regulation of chromosome congression, we depleted the endogenous MLL protein using siRNA directed against 3'untraslated region of MLL transcript, in stable cell lines expressing the recombinant MLL wild type or mutant proteins as described (Ali et al., Nucleic Acids Res. 2014). We quantified chromosome alignment by calculating the DNA spread parallel to the spindle poles in cells treated with control and MLL siRNA (Figure 4A, arrows indicate extent of DNA spread). Control siRNA-treated cells displayed a tight chromosome congression of 5-7 µm, while MLL depleted cells displayed 9-12 µm (Figure 4B, compare sample 1 and 2). Whereas reconstitution of full-length MLL (MLL_{FL}) and MLL_C subunit was able to largely rescue this phenotype, expression of MLL_N could not (Figure 4B) indicating that MLL_C subunit had a more direct role in chromosome congression than MLL_N subunit.

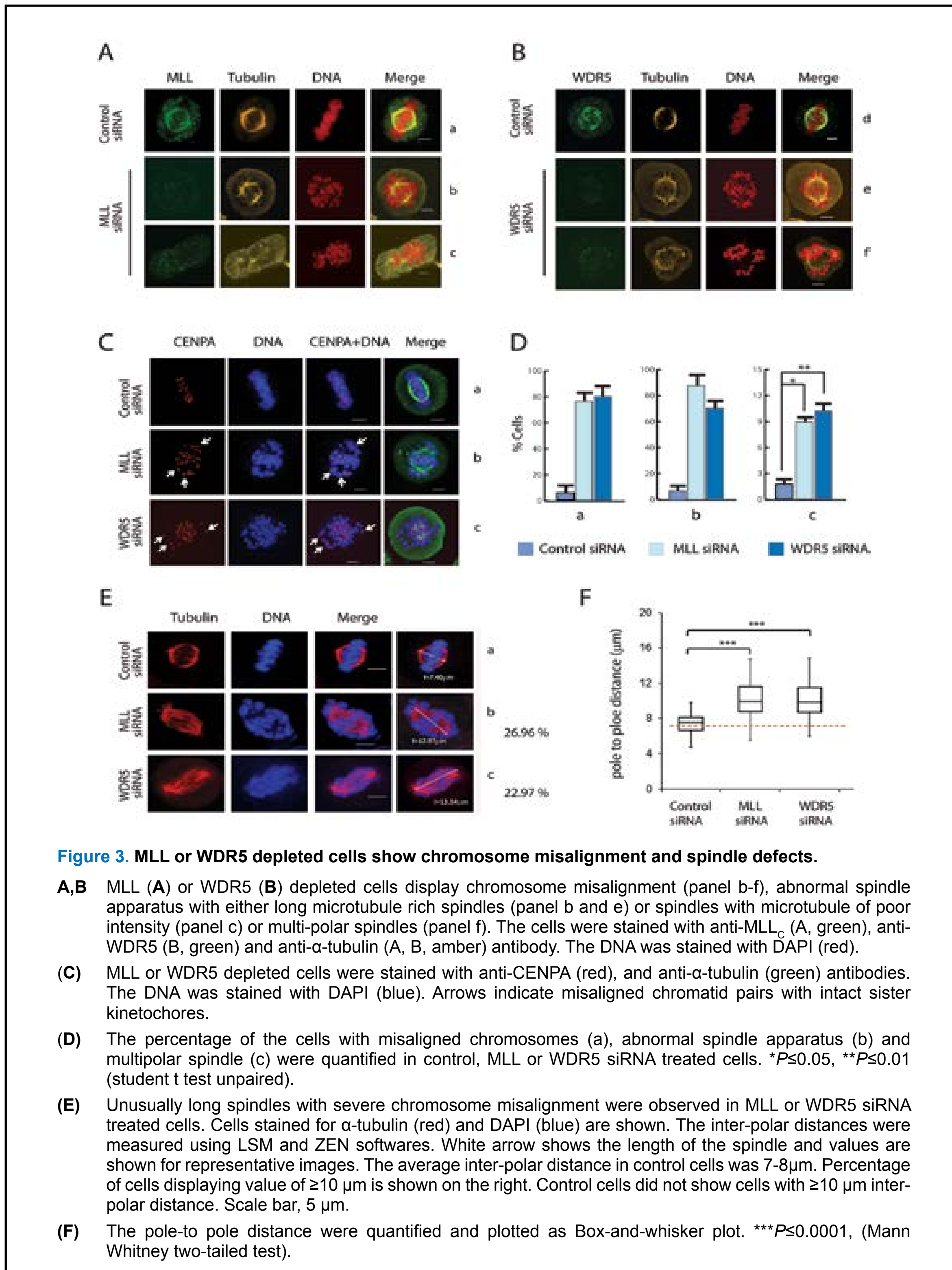

Figure 3. MLL or WDR5 depleted cells show chromosome misalignment and spindle defects.

A,B MLL (**A**) or WDR5 (**B**) depleted cells display chromosome misalignment (panel b-f), abnormal spindle apparatus with either long microtubule rich spindles (panel b and e) or spindles with microtubule of poor intensity (panel c) or multi-polar spindles (panel f). The cells were stained with anti-MLL_C (A, green), anti-WDR5 (B, green) and anti-α-tubulin (A, B, amber) antibody. The DNA was stained with DAPI (red).

(**C**) MLL or WDR5 depleted cells were stained with anti-CENPA (red), and anti-α-tubulin (green) antibodies. The DNA was stained with DAPI (blue). Arrows indicate misaligned chromatid pairs with intact sister kinetochores.

(**D**) The percentage of the cells with misaligned chromosomes (a), abnormal spindle apparatus (b) and multipolar spindle (c) were quantified in control, MLL or WDR5 siRNA treated cells. *$P≤0.05$, **$P≤0.01$ (student t test unpaired).

(E) Unusually long spindles with severe chromosome misalignment were observed in MLL or WDR5 siRNA treated cells. Cells stained for α-tubulin (red) and DAPI (blue) are shown. The inter-polar distances were measured using LSM and ZEN softwares. White arrow shows the length of the spindle and values are shown for representative images. The average inter-polar distance in control cells was 7-8μm. Percentage of cells displaying value of ≥10 μm is shown on the right. Control cells did not show cells with ≥10 μm inter-polar distance. Scale bar, 5 μm.

(F) The pole-to pole distance were quantified and plotted as Box-and-whisker plot. ***$P≤0.0001$, (Mann Whitney two-tailed test).

Similar to previous observations (Ali et al., Nucleic Acids Res. 2014), here also deletion of SET or TAD domains has no greater effect than that observed for MLL_C expression (compare Figure 4C sample 2 and 4 with Figure 4B sample 8). However, mutation in Win motif of MLL could not restore proper chromosome alignment indicating that Win motif of MLL, and therefore, MLL's interaction with WDR5, is crucial for chromosome congression (Figure 4C sample 6).

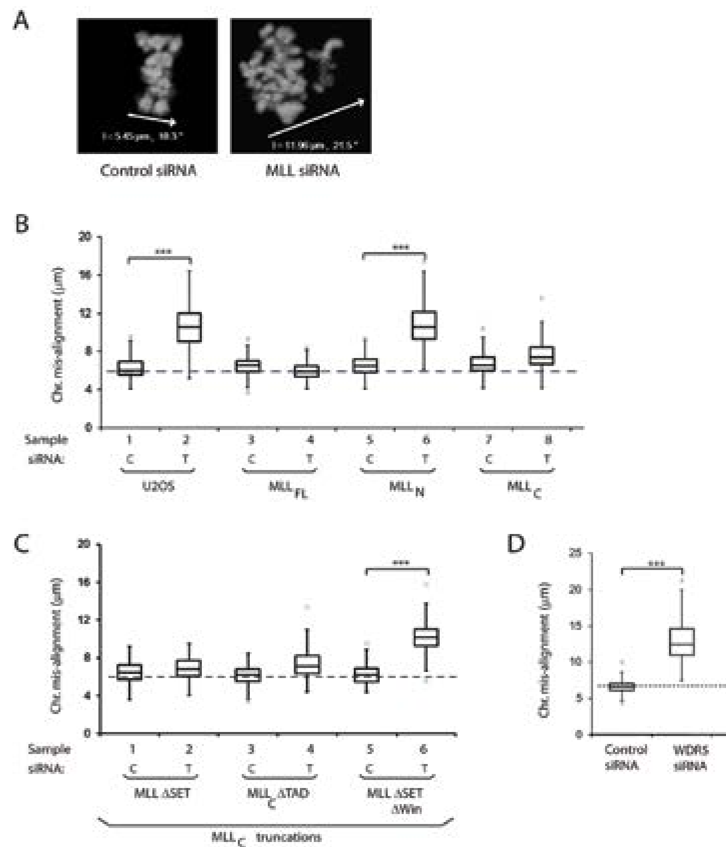

Figure 4. Mapping domain of MLL for chromosome misalignment defect.

(**A**) To calculate the extent of chromosome misalignment, the DNA spread parallel to spindle pole axis was measured using LSM and ZEN softwares as shown. White arrow shows the extent of spread and values are shown in µm inset.

(**B**) Extent of chromosome (Chr.) misalignment was quantified and plotted as Box-and-whisker plot in wild type U2OS cells, U2OS cells stably expressing MLL full length (MLL_{FL}); MLL_N representing the N subunit; MLL_C representing the C subunit, upon control (C) or MLL (T) siRNA treatment. *** $P≤0.0001$, (Mann Whitney two-tailed test).

(**C**) Extent of chromosome (Chr.) misalignment was quantified and plotted as Box-and-whisker plot in U2OS cells stably expressing MLL ΔSET lacking the SET domain; MLL_CΔTAD lacking the transcriptional activation domain (TAD), MLL ΔSETΔWin lacking the SET domain and point mutation in Win motif (R3765A) upon control (C) or MLL (T) siRNA treatment. *** $P≤0.0001$, (Mann Whitney two-tailed test).

(**D**) Extent of chromosome (Chr.) misalignment was quantified upon WDR5 or control siRNA treatment in U2OS cells and plotted as Box-and-whisker plot. ***$P≤0.0001$ (Mann Whitney two-tailed test).

Further, we found that WDR5 knockdown recapitulated the chromosome misalignment phenotype observed with the knockdown of MLL protein (Figure 4D). To conclude, our results indicate that MLL_C subunit and its association with WDR5 is essential for the proper alignment of chromosomes during mitosis.

Now, we are in the process of understanding, the exact role of MLL/WDR5 complex in spindle organization.

Others Publications:

Ali A and Tyagi S (2017) Diverse roles of WDR5-RbBP5-ASH2L-DPY30 (WRAD) complex in the functions of the SET1 histone methyltransferase family. **Journal of Bioscience** 42(1):155-159. Review.

LABORATORY OF CELL DEATH & CELL SURVIVAL

Functional protein networks controlling cellular pathways

Faculty	Maddika Subba Reddy	Staff Scientist & Wellcome Trust-DBT India Alliance Senior Fellow
PhD Students	P. V. Vivek Reddy	Senior Research Fellow (till March 2017)
	G. Narmadha Reddy	Senior Research Fellow
	Swapnil R. Shinde	Senior Research Fellow
	Parveen Kumar	Senior Research Fellow
	Varun J. Shah	Senior Research Fellow
	Prajakta Tathe	Junior Research Fellow (Since May 2016)
	Vaishna V	Junior Research Fellow (Since July 2016)
	Hilal A Reshi	Junior Research Fellow (Since Feb. 2017)
Other Members	Naga Lakshmi K	Research Associate (till Oct.2016)
	Debjani Bhattacharya	Project-JRF
	M. Prathyusha	Project-SRF (till June 2016)
	KVS Rammohan Chowdary	Project-SRF (till June 2016)
	Nanci Rani	Technical Assistant

Objectives

1. To dissect the functional network of phosphatases regulating cellular pathways.
2. To understand the cellular functions of canonical and non-canonical ubiquitination.

Summary of work done until the beginning of this reporting year (upto March 31, 2016)

Phosphatases play a critical role in nearly every cellular process, including metabolism, gene transcription, translation, cell-cycle progression, protein stability, signal transduction, and apoptosis. We initiated our studies on functional phosphatase network with the identification of interacting partners of every phosphatase in human cell. We cloned 143 human protein phosphatases in a gateway compatible triple tagged (SBP-Flag-S protein) vector and each of them was individually expressed in HEK293T cells. Protein complexes were isolated by tandem affinity purification and interacting proteins were identified by using LC-MS/MS analysis. A total of 76773 interactions were obtained from 143 phosphatase purifications. During the course of this work, we have already identified and characterized WWP2, PNUTS and TOPK as novel functional partners of PTEN. Recently, we identified a new cellular function for PTEN where we have shown that PTEN via interacting with Rab7 functions in endosome maturation. In addition to PTEN interactome, we also started developing networks of other cellular phosphatases. We identified PPM1G as a molecular switch that controls differential cellular role of E3 ligase WWP2, by controlling the transition between monomeric WWP2 and WWP2/WWP1 heterodimer. In another example, we demonstrated an important role of non-receptor tyrosine phosphatase PTPN5 in cytokinetic absicision.

Details of progress in the current reporting year (April 1, 2016 - March 31, 2017)

Theme 1. Functional studies on phosphatase networks

Currently, we are focused on actively expanding the functional network of different families of phosphatases in the cell. Depending on the substrate residue they act on, protein phosphatases are broadly classified into two classes such as (A) Tyrosine phosphatases and (B) Serine/Threonine phosphatases. Firstly, we started to analyse the interactome

of human tyrosine phosphatases. By using biochemical purification and mass spectrometric identification, we found a total of 42262 interactions from 82 tyrosine phosphatase purifications. By using a SAINT score cut off of 0.8, FCA> 3, FCB >2.5, IS >1 and WD score >1, we identified 2172 high confident interactions (HCIs) mediated by 1021 proteins (HCIPs) for these tyrosine phosphatases. A comparison of our data with known interactions revealed 294 (~ 14%) known interactions and 1878 (~86%) novel interactions in the list.

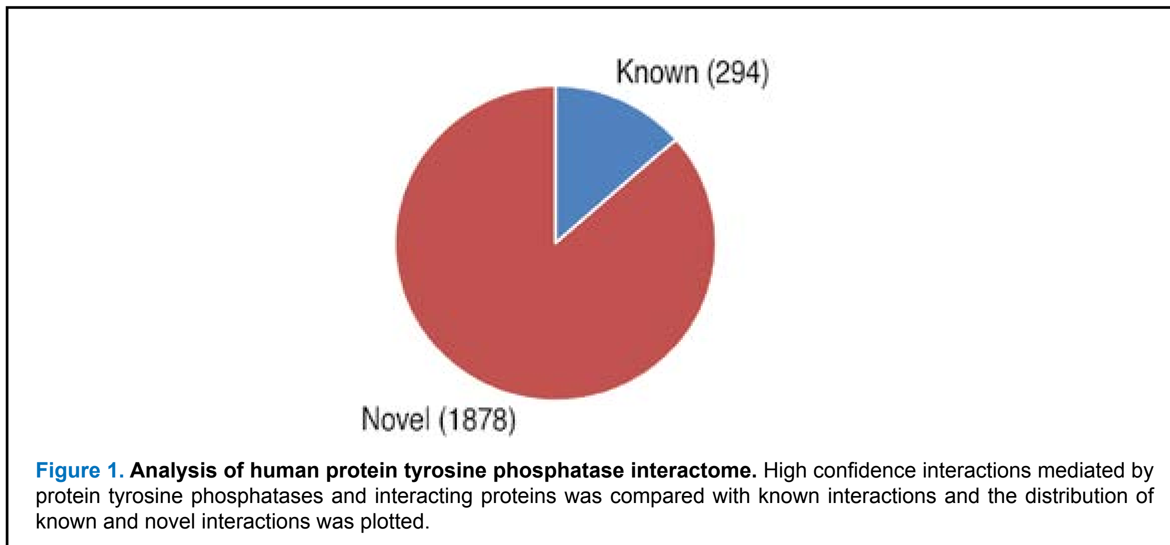

Figure 1. Analysis of human protein tyrosine phosphatase interactome. High confidence interactions mediated by protein tyrosine phosphatases and interacting proteins was compared with known interactions and the distribution of known and novel interactions was plotted.

In order to further understand the functional role of these interactions, we annotated them to KEGG pathways. Importantly, several key cellular signaling pathways such as PI3-K/Foxo, Hippo-YAP, Wnt, Hedgehog, HIF-1, mTOR, Ras-MAPK, AMPK, RAP1 and VEGF were highly enriched for HCIPs of different phosphatases. Further, we used OMIM annotated disease linked genes and analysed for interaction of phosphatases with these diseases linked genes. We identified 270 disease-linked proteins that interact with 79 phosphatases. We found several diseases such as 3M syndrome, Charcot-Marie-Tooth disease, Parkinson disease, cardimyopathies, Cowden syndrome, Fanconi anemia, and X-linked mental retardation linked to phosphatases. Further, we also matched phosphatase interactome to COSMIC (cancer gene census) dataset that contain genes mutated in human cancers. Nearly 70% of phosphatases are associated with cancer-linked proteins.

In addition to mapping the phosphatase network, we simultaneously started to characterize several of putative functional interactions of these purified phosphatases. To this end, we made significant progress in understanding multiple novel phosphatase interactions in the lab. The data generated from some of the exciting interactions has been presented below.

1.1. PHLPP facilitates kinetochore assembly by regulating SGT1

PHLPP is a tumor suppressor phosphatase that plays critical roles in cell survival. In this study, we identified PHLPP1 as an essential protein required for proper assembly of kinetochores in cells. We found SGT1 as one of the potential interacting partners of PHLPP1. Since SGT1 is critical for proper kinetochore assembly during mitotic cycle, we tested if loss of PHLPP1 phenocopies SGT1 loss from cells. Time-lapse imaging revealed that silencing of PHLPP1 in HeLa cells lead to delayed progression of cells in mitosis. Delayed progression of cells in mitosis upon PHLPP1 depletion is accompanied with multiple severe mitotic defects such as misaligned chromosomes, multipolar spindles and abnormal centrosomes. We found that outer kinetochore proteins such as HEC1 and CENP-E failed to localize to kinetochores in PHLPP1 depleted cells. In contrary, localization of core inner kinetochore protein CENP-A is unaffected by PHLPP1 loss. As, depletion of PHLPP1 caused severe reduction in recruitment of outer kinetochore proteins, we next tested if kinetochore-microtubule attachment is affected in these cells. In deed, co-staining of kinetochores and microtubules with CENP-A and α-tubulin respectively revealed that PHLPP1 depletion

lead to defective attachment of microtubules with kinetochores. Mechanistically, we found that loss of PHLPP1 from cells lead to SGT1 degradation and thus causes defective assembly of kinetochores. We found RNF41 as a novel E3 ligase that ubiquitinate and degrade SGT1 in a phosphorylation dependent manner.

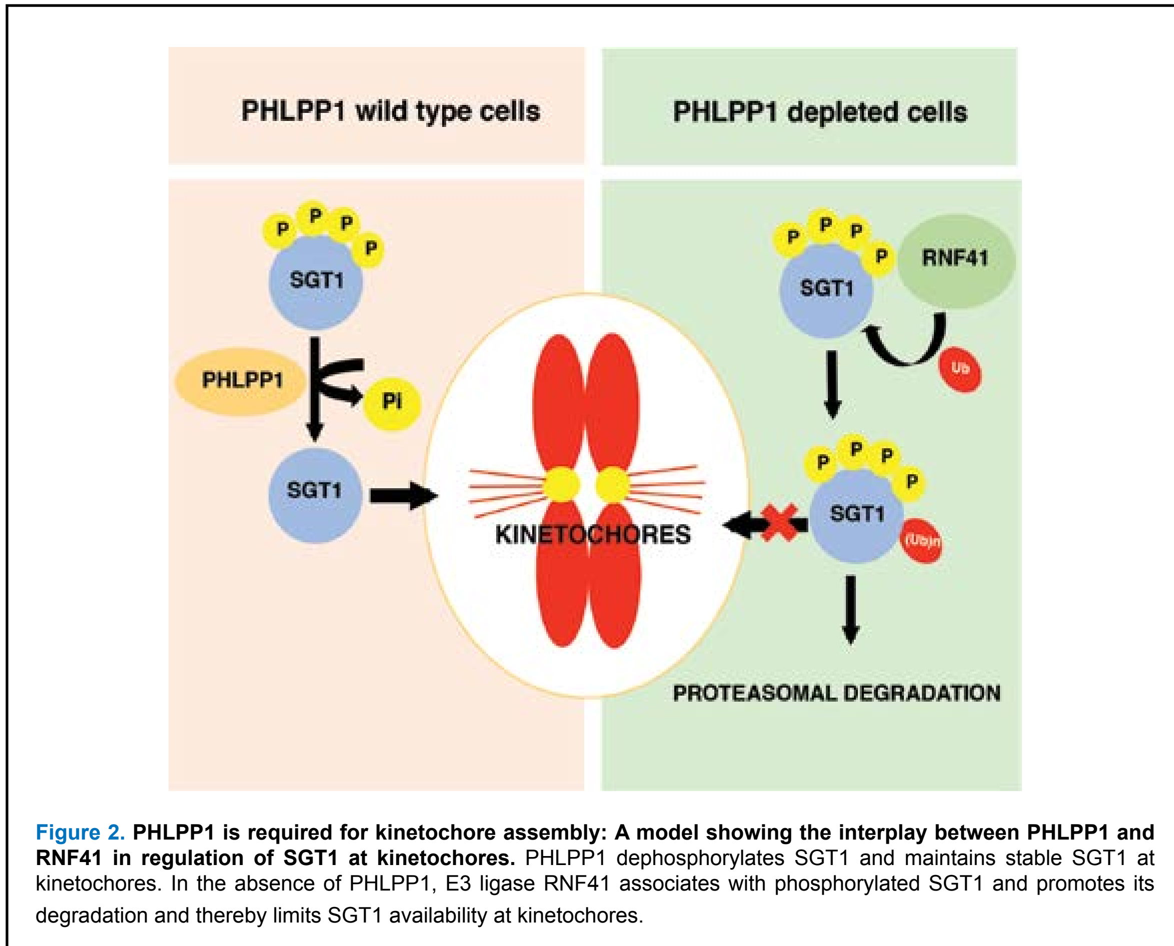

Figure 2. PHLPP1 is required for kinetochore assembly: A model showing the interplay between PHLPP1 and RNF41 in regulation of SGT1 at kinetochores. PHLPP1 dephosphorylates SGT1 and maintains stable SGT1 at kinetochores. In the absence of PHLPP1, E3 ligase RNF41 associates with phosphorylated SGT1 and promotes its degradation and thereby limits SGT1 availability at kinetochores.

Interaction of SGT1 with RNF41 is dramatically enhanced in the absence of PHLPP1 and conversely exogenous expression of PHLPP1 lead to loss of SGT1 interaction with its E3 ligase. Thus, PHLPP1 protects SGT1 from polyubiquitination and degradation by interfering with SGT1 interaction with its E3 ligase RNF41. PHLPP1 dephosphorylates SGT1 at four conserved residues and thereby prevents SGT1 association with RNF41 and thus counters its degradation. Importantly, either depletion of RNF41 or expression of non-phosphorylatable SGT1 mutant rescued the kinetochore defects caused due to PHLPP1 loss. Taken together, our results suggest that PHLPP1 play an important and dynamic role in the assembly of kinetochores by counteracting RNF41 mediated SGT1 degradation.

1.2. PTEN controls glucose transport by impairing GLUT1 recycling

PTEN is a well-known tumor suppressor that acts to down-regulate cell proliferation, survival and metabolic signaling pathways, majorly through its lipid phosphatase activity. Recently, we have demonstrated that PTEN regulates EGFR signaling by promoting late endosome maturation by virtue of its protein phosphatase activity. PTEN promotes endosome maturation by dephosphorylating Rab7 on two conserved residues. In addition to its role in endosome maturation, now we identified a critical regulatory role of PTEN in endosomal recycling of GLUT1 and glucose transport in a phosphatase independent manner. Depletion of PTEN in cells resulted in significant increase in GLUT1 levels at the plasma membrane. On the other hand,

overexpression of full length PTEN reduced GLUT1 levels at plasma membrane. Intriguingly, PTEN ∆PDZ binding motif mutant, although had intact phosphatase activity, failed to suppress GLUT1 membrane levels possibly indicating a phosphatase independent function of PTEN in regulation of GLUT1. Expression of full length PTEN led to significant reduction in co-localization of GLUT1 with sorting endosomes. Defective sorting of GLUT1 to recycling endosomes due to PTEN expression resulted in rerouting of GLUT1 to lysosomes. GLUT1 is widely expressed in almost all types of cells and tissues and is required for the basal glucose uptake. As we observed that PTEN regulates membrane GLUT1 levels, we next tested the importance of PTEN in glucose transport. Depletion of PTEN significantly enhanced cellular uptake of glucose. We found different components of recycling endosomes in our PTEN proteomic data. Currently, we are trying to understand the mechanistic link between PTEN and recycling endosomes.

Theme 2: Roles of canonical and non-canonical ubiquitination in cells

Ubiquitination is an ATP-dependent, highly ordered multistep enzymatic process, which results in covalent attachment of ubiquitin to the substrate. Ubiquitin linked to the substrates serves as a molecular tag that marks proteins for either degradation by proteasome dependent pathway or to function in wide variety of processes in a proteasome independent manner. In this project we are interested in studying both the canonical and non-canonical functions of ubiquitination in cells. During previous years, we have reported that an oncogenic E3 ligase WWP2 ubiquitinates PTEN and p73 in a canonical K48 linkage that leads to their degradation through proteasome. On the other hand, we also found that WWP2 mediates a non-canonical linkage on DVL2, a critical component of Wnt signaling pathway.

2.1. Ubiquitination of Dvl2 is required for signalosme formation in Wnt pathway

We found that WWP2 ubiquitinates DVL2 but interestingly does not lead to its degradation. In our functional experiments we found that WWP2 is required for activation of Wnt signaling pathway. Our mapping experiments revealed that WWP2 ubiquitinates DVL2 on sites located in its PDZ domain. Several lysines were found in PDZ domain of DVL2. Mutation of Lysine 343 to Arginine hampered DVL2 ability to form signalosomes upon Wnt activation. This probably suggests that WWP2 might ubiquitinate DVL2 at K343 residue and thereby promotes its association with Wnt signalosomes. Interestingly, we found several ubiquitin-binding domain containing proteins in the interacting list of DVL2 upon Wnt stimulation. It is possible that non-canonically ubiquitinated DVL2 might specifically interact with UBA containing proteins, which is critical for its tranlocation to the sites of Wnt induced signalosomes. We are currently probing the interactions of various UBA domain proteins with ubiquitinated DVL2, which will help us to mechanistically understand the basis of Wnt induced signalosome formation.

2.2. Non-canonical functions of HECT type E3 ligases

Earlier, while studying the role of ubiquitination in extracellular protein secretion, we used YB-1 as a model protein and identified the indispensable role of ubiquitination in this process. Importantly, we discovered HECT type E3 ligase, HACE1 as YB-1 interacting E3 ligase that has the ability to generate functional K27 linked non-canonical ubiquitin linkages on its substrate. K27 ubiquitin linkages on YB-1 are necessary for its interaction with Tumor Susceptibility Gene 101 (TSG101), a component of the Multi Vesicular Body (MVB) pathway, which facilitates its secretion. In summary, we identified a novel functional role for non-canonical ubiquitin linkages in mediating protein secretion.

In addition, we found an alternate mechanism for HACE1, where it mediates protein degradation in an ubiquitination independent manner but proteasome dependent manner. We identified that E3 ligase binds to proteasome directly and delivers the substrates to 20S proteasome independent of its catalytic activity. We are currently trying to understand the functional relevance of non-canonical degradation of these substrates by HECT-type E3 ligases.

2.3. Identification of new functional E3 ligase complexes and their substrates

E3 ligases are critical proteins in the final step of the ubiquitination process where they recruit ubiquitin charged E2 enzymes along with specific substrates. In this work, we aim to identify new complexes for E3 ligases by using proteomics approach and further characterize their substrates by using human protoarrays. In one example we identified that SMU1, a LisH domain

containing protein, orchestrates the assembly of a functional E3 ligase complex. We identified that SMU1 assembles CRL type of E3 ligase that contains DDB1, CUL7 and a RING type E3 ligase as core components. SMU1 acts as a substrate recognition component in the E3 ligase complex. siRNA mediated depletion of SMU1 lead to loss of substrate interaction with E3 ligase and there by resulted in diminished substrate ubiquitination. We found that appropriate ubiquitination of substrates by SMU1-E3 ligase complex is necessary for maintaining the genomic stability.

Publications

1. Shinde SR, and Maddika S (2016). PTEN modulates EGFR late endocytic trafficking and degradation by dephosphorylating Rab7. ***Nature Communications.*** 7: 10689.
2. Raychaudhuri K, Chaudhary N, Gurjar N, D'Souza R, Limzerwala J, Maddika S, and Dalal SN (2016). 14-3-3σ gene loss leads to activation of the epithelial to mesenchymal transition due to the stabilization of c-Jun protein. ***Journal of Biological Chemistry.*** 291(31): 16068-81.
3. Joshi K, Shah VJ, and Maddika S (2016). GINS complex protein Sld5 recruits SIK1 to activate MCM helicase during DNA replication. ***Cell Signaling*** 28(12): 1852-62.
4. Shinde SR, and Maddika S (2016). A modification switch on a molecular switch: Phosphoregulation of Rab7 during endosome maturation. ***Small GTPases***. 7(3): 164-7.
5. Shinde SR, and Maddika S (2017). Post-translational modifications of Rab GTPases. ***Small GTPases.*** 1-8.

Other Publications

Kumar P, and Maddika S (2017). Cellular dynamics controlled by phosphatases. Journal of Indian Institute of Science. 97 (1): 129-145.

LABORATORY OF CELL SIGNALLING

Investigating the role of inositol pyrophosphates in eukaryotic cell physiology

Faculty	Rashna Bhandari	Staff Scientist
PhD Students	Rathan Singh Jadav	Senior Research Fellow (till Oct. 2016)
	Manasa VL Chanduri	Senior Research Fellow
	Aushaq Bashir Malla	Senior Research Fellow
	Sitalakshmi Thampatty R	Senior Research Fellow
	Shubhra Ganguli	Senior Research Fellow
	Akruti Shah	Junior Research Fellow
	Jayraj Sen	Junior Research Fellow (since July 2016)
Other Members	Ruth Manorama Ravoori	Technical Assistant
	Debaditya De	Project SRF (since Sept. 2016)
	Prathyusha Munnangi	Project JRF (since Aug. 2016)
	Vineesha Oddi	Project Technical Assistant (since Dec. 2016)
	K. Viswakalyan	Project Technical Assistant (since Dec. 2016)
	Ravichand Palakurti	Research Associate (since Jan. 2017)
Collaborators	Nagaraj Balasubramanian	IISER, Pune
	Roop Mallik	TIFR, Mumbai
	Kana M. Sureshan	IISER, Thiruvananthapuram

Objectives

Inositol pyrophosphates are derivatives of inositol that contain pyrophosphate or diphosphate moieties in addition to monophosphates. They include diphosphoinositol pentakisphosphate (PP-IP_5, or IP_7) and bis-diphosphoinositol tetrakisphosphate ($[PP]_2$-IP_4 or IP_8), which participate in diverse biological functions, including DNA recombination, vesicular trafficking, rRNA transcription and osmotic regulation. The beta phosphate group of inositol pyrophosphates can be transferred to pre-phosphorylated serine residues on proteins to form pyrophosphoserine. Pyrophosphorylation occurs on several proteins within the cell, including proteins involved in ribosome biogenesis, vesicular trafficking and glycolysis. 5PP-IP_5 (5-IP_7) is synthesised from inositol hexakisphosphate (IP_6) and ATP by IP_6 kinases. Mammals have three isoforms of IP_6 kinase, IP6K1, IP6K2 and IP6K3, whereas *Saccharomyces cerevisiae* have a single IP_6 kinase, Kcs1.

Our aim is to understand the molecular mechanisms by which various cellular phenomena are regulated by inositol pyrophosphates. We utilise *S. cerevisiae,* mammalian cell lines, and knockout mouse strains as model systems to investigate the signalling and metabolic pathways that are altered when inositol pyrophosphate levels are perturbed. In particular, we focus on the following objectives:

1. Investigate the cellular functions of mammalian inositol hexakisphosphate kinase 1 (IP6K1).
2. Understand the molecular details of protein pyrophosphorylation by inositol pyrophosphates.
3. Study the role of inositol pyrophosphates and IP_6 kinases in whole animal physiology.

Summary of work done until the beginning of this reporting year (upto March 31, 2016)

A gene expression microarray analysis comparing *Ip6k1* knockout (*Ip6k1*$^{-/-}$) mouse embryonic fibroblasts (MEFs) with wild type (*Ip6k1*$^{+/+}$) MEFs, revealed that regulation of the actin cytoskeleton is altered in the absence of IP6K1. We observed that *Ip6k1*$^{-/-}$ MEFs spread more slowly on fibronectin coated surfaces compared with their *Ip6k1*$^{+/+}$ counterparts. Stable expression of shRNA directed against *Ip6k1* in the human colon cancer cell line HCT116 resulted in 60% knockdown of IP6K1 levels and a significant reduction in intracellular IP_7. These cells showed a decrease in chemotactic migration towards

serum-rich medium, and reduced collective cell migration in a wound healing assay.

In an earlier publication (Jadav *et al*., J. Biol. Chem. 2013), we described a role for inositol pyrophosphates synthesised by IP6K1 in homologous recombination (HR) mediated repair of DNA double strand breaks in mammalian cells. *Ip6k1*$^{-/-}$ MEFs show decreased viability and reduced recovery after induction of DNA damage by the replication stress inducer, hydroxyurea (HU). Markers for HR repair, including γH2AX, Rad51 and BLM, are recruited to DNA damage sites but persist up to 6-10 h after HU removal in knockout, but not in wild type MEFs, indicating that HR-mediated DNA repair is initiated but incomplete in cells lacking IP6K1. Expression of catalytically active but not inactive IP6K1 can restore the repair process in knockout MEFs, implying that inositol pyrophosphates are required for HR-mediated repair. MUS81, a nuclease involved in resolution of Holliday junctions towards the end of the HR repair pathway, is recruited to DNA damage foci during recovery from HU treatment in wild type, but not in *Ip6k1*$^{-/-}$ MEFs, suggesting that HR repair is stalled in knockout MEFs prior to the formation of Holliday junctions.

We have earlier reported that *Ip6k1*$^{-/-}$ male mice are sterile due to azoospermia, the absence of mature spermatozoa in the epididymides. We observed that IP6K1 is expressed at high levels in late pachytene and diplotene spermatocytes and in round spermatids. While following the first wave of spermatogenesis, we noted that *Ip6k1*$^{-/-}$ testes display a delay in the completion of meiosis and a major defect in spermiogenesis, the differentiation of round to elongated spermatids. We observed that elongating spermatids in *Ip6k1*$^{-/-}$ tubules stain positive in a TUNEL assay and also contain cleaved caspase 3, indicating that these spermatids undergo apoptosis and are eventually lost.

Details of progress made in the current reporting year (April 1, 2016 - March 31, 2017)

Project 1: Cellular functions of mammalian inositol hexakisphosphate kinase 1 (IP6K1)

We explored the effect of IP6K1 depletion on the invasive property of cancer cells by a matrigel invasion assay, which mimics early steps in tumor metastasis. IP6K1 depleted HCT116 cells showed significantly reduced invasion compared with NT control cells (Figure 1A, B). To determine if the reduced invasion potential of IP6K1 depleted cancer cells extends to *Ip6k1* knockout cells *in vivo* we utilized the 4 nitroquinoline 1-oxide (4NQO) oral squamous cell carcinoma model. 4NQO is a water-soluble quinoline derivative, which when administered to mice in drinking water induces a temporal progression of the different phases of carcinogenesis from hyperplasia to dysplasia to invasive carcinoma. After 24 weeks of continuous exposure to 4NQO, we observed 100% survival in both *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ mice. Histopathological examination of tissues from the upper aerodigestive tract revealed hyperplasia and dysplasia in tongue and esophagus of both *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ mice (Figure 1 C, D). However, invasive carcinoma, defined by the migration of dysplastic epithelial cells into sub-epithelial tissues, was less in case of *Ip6k1*$^{-/-}$ mice, suggesting that these mice are protected against 4NQO induced carcinogenesis. The invasive potential of epithelial cells has been shown to inversely correlate with the expression of the epithelial biomarker E-cadherin which promotes cell-cell adhesion. We observed higher levels of E-cadherin in sh*IP6K1* cells compared with NT cells (Figure 1E, F), correlating with their reduced invasion potential. Taken together, our studies in cells and mice lacking IP6K1 identify a role for this protein in coordinating multiple cellular events to regulate cell migration, invasion and carcinogenesis.

Project 2: Role of inositol pyrophosphates in maintaining genome stability

To examine the function of IP6K1 in DNA repair independent of any alteration in p53-dependent signalling pathways, we conducted shRNA-mediated knock down of IP6K1 expression in U-2 OS human osteosarcoma cells which carry wild type p53. U-2 OS cells stably expressing shRNA directed against IP6K1 showed an approximately 70% decrease in IP6K1 levels compared with non-targeted cells (Figure 2A). Our previous studies had shown than MEFs lacking IP6K1 display persistent DNA damage upon long-term treatment with the ribonucleotide reductase inhibitor HU, which leads to replication fork collapse. To determine whether the role of IP6K1 in DNA repair is also evident when DNA damage occurs via other pathways, we treated U-2 OS cells with the interstrand crosslinker mitomycin C, the radiomimetic neocarzinostatin, and the DNA intercalator phleomycin. We monitored the extent of DNA damage by counting the number

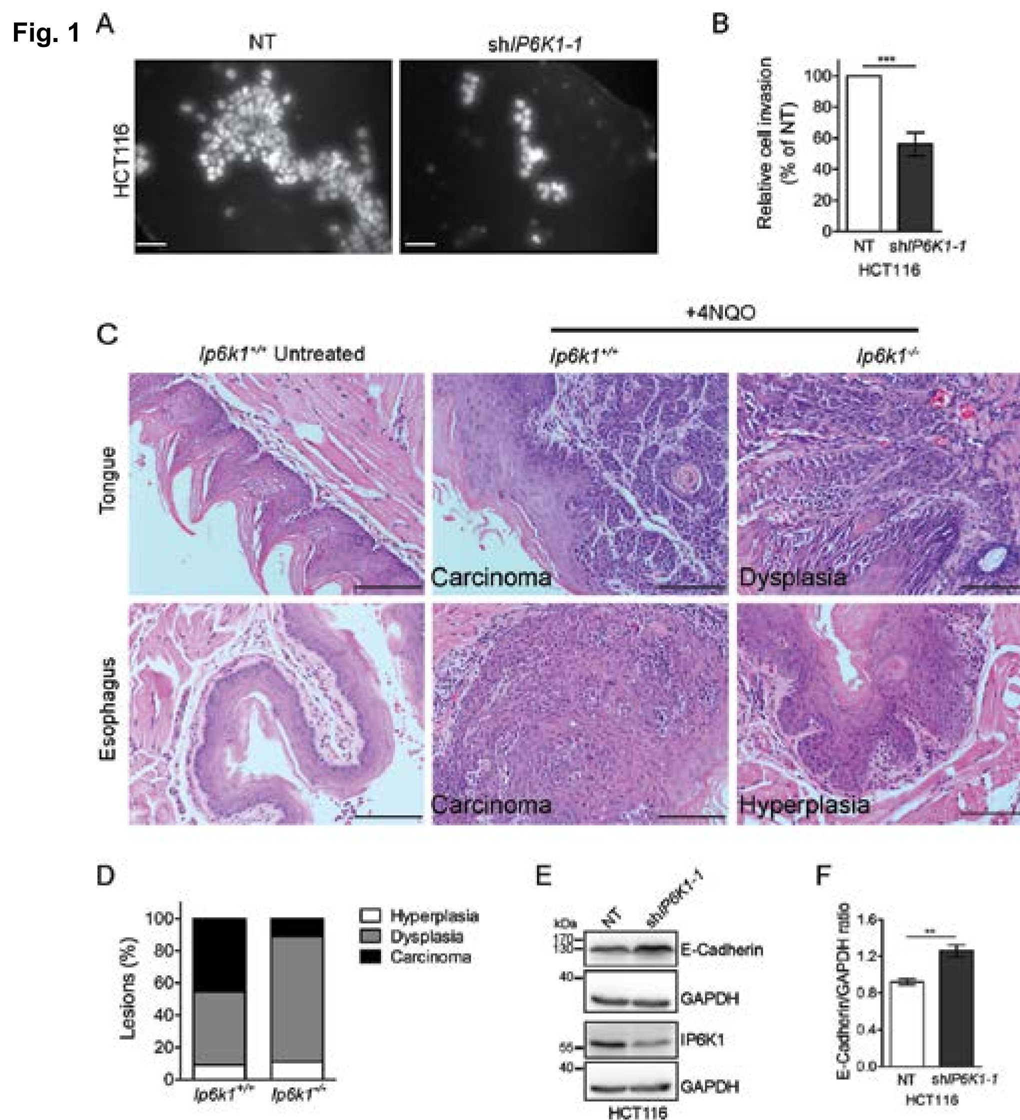

Figure 1. IP6K1 modulates the invasive potential of cancer cells. (A) HCT116 cells stably expressing NT or sh*IP6K1* were allowed to invade a matrigel matrix to move towards a high-serum gradient for 24 h. Representative images show cells that migrated through the gel to the other side of the membrane, visualized by staining with DAPI. Scale bars represent 50 µm. (B) Quantification of (A); bar graphs show the number of invaded cells normalized to the NT control. Data are mean ± SEM from five independent experiments, and were analyzed by a one sample *t*-test. (C) *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ mice were administered the oral carcinogen 4NQO in drinking water continuously for 24 weeks. Representative images of haematoxylin and eosin stained tissues show the normal epithelium of the tongue and esophagus of untreated *Ip6k1*$^{+/+}$ mice (left panel), induction of invasive carcinoma in the tongue and esophagus of *Ip6k1*$^{+/+}$ mice (middle panel), and the same tissues in *Ip6k1*$^{-/-}$ revealing dysplasia and hyperplasia (right panel). Scale bars represent 100 µm. (D) Stacked bars represent the percentage of different types of lesions observed in mice of the indicated genotypes. n = 11 and 9 for *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ mice respectively. (E) Immunoblot analysis of the epithelial marker E-cadherin in HCT116 cells expressing NT or sh*IP6K1*. (F) Quantification of (E); levels of the epithelial marker E-cadherin are indicated as a ratio with respect to the levels of GAPDH which was the loading control. Data represents mean ± SEM from three independent experiments and was analyzed using a two tailed unpaired Student's *t*-test. ** $P \le 0.01$, *** $P \le 0.001$.

of γH2AX foci per nucleus and noted that all three drugs induce the same extent of damage in non-targeted and IP6K1 knockdown U-2 OS cells (Figure 2B-G). However, when cells were allowed to recover after treatment, we observed fewer γH2AX foci, indicative of greater recovery from DNA damage, in non-targeted cells compared to cells with reduced IP6K1. These

observations suggest that the role of IP6K1 in recovery from DNA damage is independent of the mode of damage and support our hypothesis that IP_7 is essential for the HR-mediated DNA repair pathway downstream of Rad51 recruitment, but upstream of Holliday junction formation. We are currently attempting to identify the molecular targets of IP_7 in DNA repair pathways activated upon treatment of U-2 OS cells with mitomycin C.

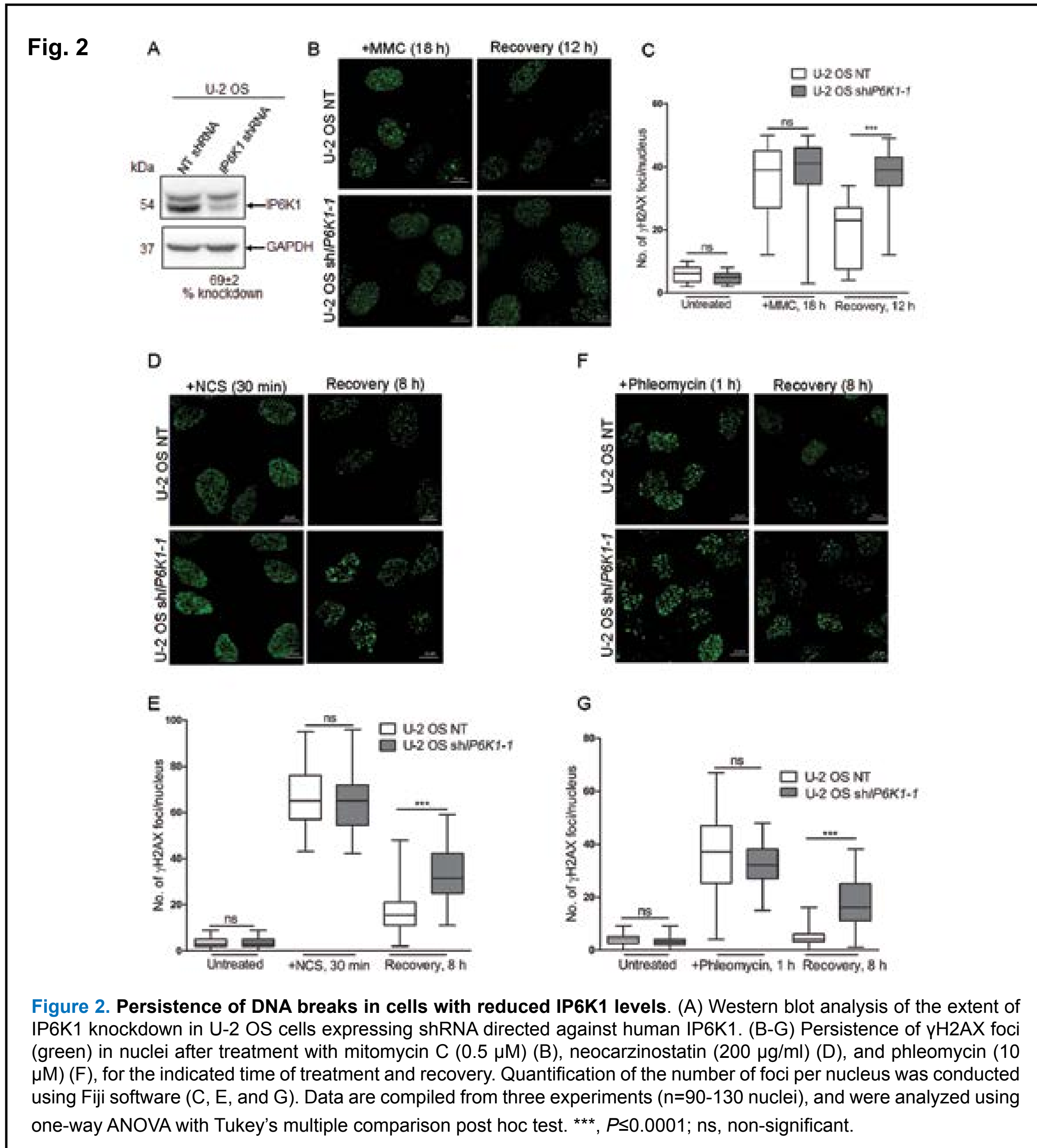

Figure 2. Persistence of DNA breaks in cells with reduced IP6K1 levels. (A) Western blot analysis of the extent of IP6K1 knockdown in U-2 OS cells expressing shRNA directed against human IP6K1. (B-G) Persistence of γH2AX foci (green) in nuclei after treatment with mitomycin C (0.5 μM) (B), neocarzinostatin (200 μg/ml) (D), and phleomycin (10 μM) (F), for the indicated time of treatment and recovery. Quantification of the number of foci per nucleus was conducted using Fiji software (C, E, and G). Data are compiled from three experiments (n=90-130 nuclei), and were analyzed using one-way ANOVA with Tukey's multiple comparison post hoc test. ***, $P \leq 0.0001$; ns, non-significant.

Project 3. Role of IP6K1 in mouse spermatogenesis

To closely examine the development of *Ip6k1*$^{-/-}$ spermatids, we identified the 16 developmental steps of spermiogenesis based on the shape of the nucleus and acrosome by co-staining testes sections with DAPI and peanut agglutinin (PNA), a lectin that binds to glycoconjugates on the outer acrosomal membrane (Figure 3A, B). Analysis of adult stage XI seminiferous tubules revealed that in *Ip6k1*$^{-/-}$ mice the round spermatids advance to step 10-11 elongating spermatids, but their

nuclear morphology is abnormal compared to *Ip6k1*$^{+/+}$ mice (Figure 3A). By stage VIII fully condensed ready-to-release spermatids were seen in *Ip6k1*$^{+/+}$ tubules, but were entirely absent in *Ip6k1*$^{-/-}$ mice (Figure 3B). Stage VIII tubules also show step 8 round spermatids with a fully developed acrosome, which appear identical in *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ mice. We isolated elongated spermatids corresponding to steps 13 to 16 of spermatid differentiation by transillumination-assisted microdissection of seminiferous tubules, and stained them with DAPI to detect their nuclei. *Ip6k1*$^{-/-}$ spermatids displayed irregularly shaped heads and a bent or blunt apex, lacking the typical hook-shaped appearance of *Ip6k1*$^{+/+}$ spermatids (Figure 3C). Consistent with this,

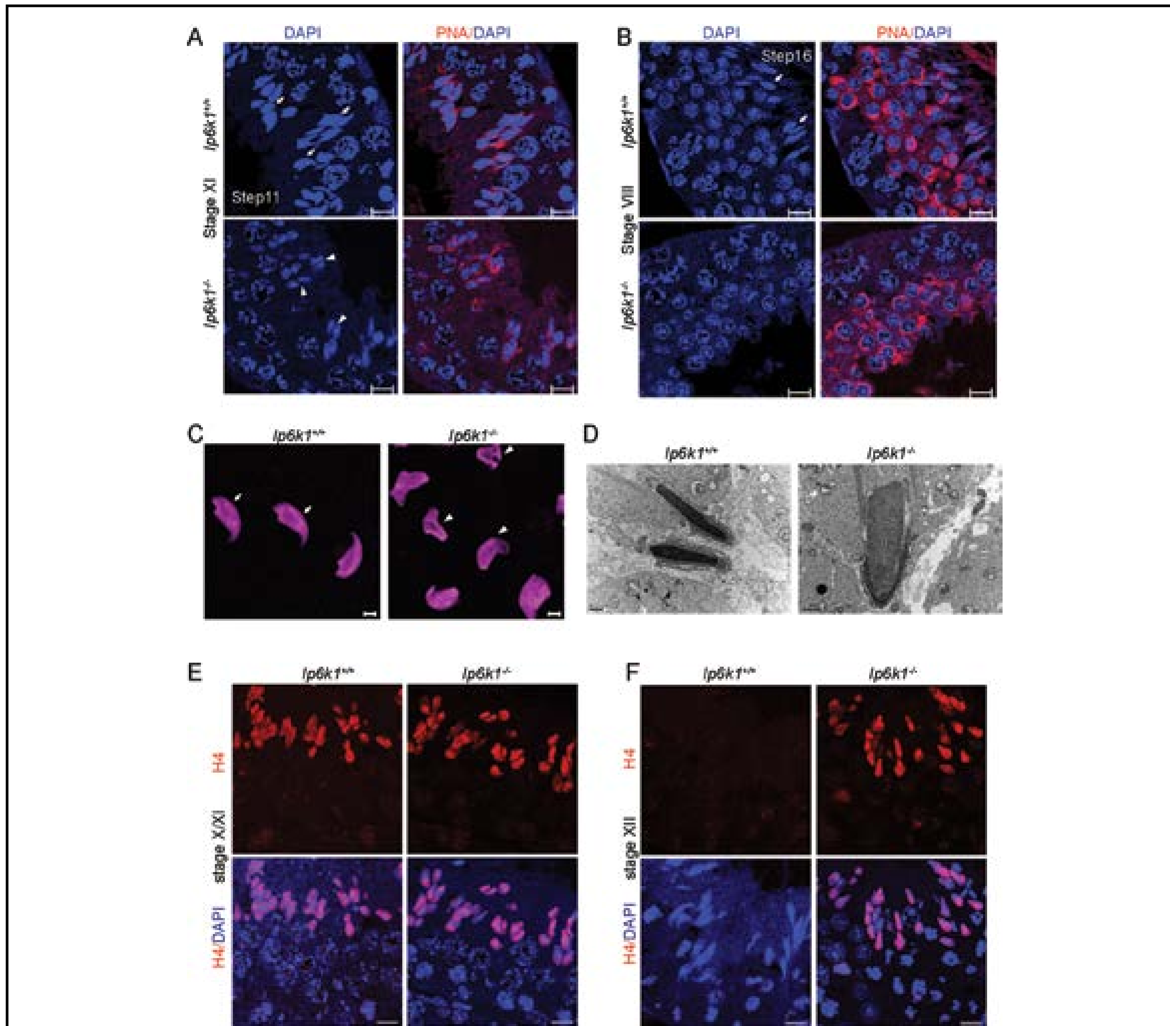

Figure 3. Loss of IP6K1 leads to abnormal elongation and DNA condensation in mouse spermatids. (A, B) *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ testes cross sections were stained with peanut agglutinin (PNA, red) to detect the outer acrosomal membrane and DAPI to mark nuclei. The stage of the seminiferous epithelium was determined for each tubule cross section by examining the presence, morphology and position of different cell types. Scale bar is 10 µm. In stage XI tubules, arrows indicate step 11 condensing spermatids in *Ip6k1*$^{+/+}$ testes and arrowheads point to abnormally condensing spermatids in *Ip6k1*$^{-/-}$ testes (A). In stage VIII tubules, fully condensed step 16 elongated spermatids (arrows) are present in *Ip6k1*$^{+/+}$ testes but are absent in *Ip6k1*$^{-/-}$ testes, which only contain round spermatids (B). (C) Elongated spermatids stained with DAPI (pseudo-coloured pink) indicating spermatid head morphology. Arrows point to the typical hook-shape of elongated spermatids in *Ip6k1*$^{+/+}$ testes, and arrowheads indicate abnormally condensed elongated spermatids in *Ip6k1*$^{-/-}$ testes. Scale bar is 2 µm. (D) Transmission electron microscopy (TEM) images of an abnormally condensed and loosely packed *Ip6k1*$^{-/-}$ elongated spermatid, and fully condensed and tightly packed *Ip6k1*$^{+/+}$ elongated spermatids. Scale bar is 0.5 µm. (E, F) Immunostaining of *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ testes sections shows abnormal retention of histone H4 (red) in *Ip6k1*$^{-/-}$ elongating spermatids. Spermatid nuclei were counterstained with DAPI (blue). Histone H4 is detected in step 10-11 (stage X/XI) *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ elongating spermatids (E). H4 is completely evicted in step 12 (stage XII) *Ip6k1*$^{+/+}$ elongating spermatids, but is retained in *Ip6k1*$^{-/-}$ spermatids in stage XII (F). Scale bar is 10 µm.

transmission electron microscopy of elongating/elongated spermatids revealed less condensed and loosely packed deformed nuclei with uneven density in *Ip6k1*$^{-/-}$ spermatids, in comparison to tightly packed and homogeneously condensed *Ip6k1*$^{+/+}$ spermatids (Figure 3D).

To follow the process of DNA condensation during spermiogenesis, we tracked the presence of histone H4 in *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ elongating spermatids. As expected, histone H4 was visible in both *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ step 10-11 (stage X/XI) early elongating spermatids (Figure 3E). As *Ip6k1*$^{+/+}$ spermatids advanced to step 12 (stage XII), histone H4 was no longer visible, but *Ip6k1*$^{-/-}$ spermatids in stage XII tubules contained histone H4, suggesting that these spermatids do not progress beyond step 11 (Figure 3F). These data suggest that improper nuclear condensation of *Ip6k1*$^{-/-}$ elongating spermatids may arise due to deficiencies in sperm DNA condensation. We are currently investigating the molecular functions of IP6K1 during spermiogenesis.

Publications

(i) Research papers published in the calendar year 2016 (in print with final page numbers)

1. Jadav RS, Kumar D, Buwa N, Ganguli S, Thampatty SR, Balasubramanian N and Bhandari R (2016). Deletion of inositol hexakisphosphate kinase 1 (IP6K1) reduces cell migration and invasion, conferring protection from aerodigestive tract carcinoma in mice. ***Cellular Signalling*** 28: 1124-1136.
2. Chanduri M, Rai A, Malla AB, Wu M, Fiedler D, Mallik R and Bhandari R (2016). Inositol hexakisphosphate kinase 1 (IP6K1) activity is required for cytoplasmic dynein-driven transport. ***Biochemical Journal*** 473: 3031-3047.

(iv) Other publications:

1. Chanduri M and Bhandari R (2016). Protein pyrophosphorylation by inositol pyrophosphates. ***Cell Biology Newsletter, published by Indian Society of Cell Biology*** 35: 30-35.
2. Shah A, Ganguli S, Sen J and Bhandari R (2017). Inositol pyrophosphates: energetic, omnipresent and versatile signalling molecules. ***Journal of the Indian Institute of Science*** 97: 23-40.

Laboratory of Chromatin Biology and Epigenetics

Understanding functions and regulation of Sirtuin family protein deacetylases

Faculty	Devyani Haldar	Staff Scientist
PhD Students	Lahari Konada	Senior Research Fellow
	Raghavendra Vadla	Senior Research Fellow
	Amrita Sengupta	Senior Research Fellow
	Shalini Aricthota	Senior Research Fellow
Other Members	Nirupama Chatterjee	Technical Officer
Collaborators	Manojit Pal	DRILS, Hyderabad, India
	Gopalakrishnan Bulusu	TCS Innovation Labs (Life Science Division), TCS Limited, Hyderabad, India.

Objectives

Reversible acetylation/deacetylation of proteins regulates numerous important cellular processes. The Sirtuin family NAD+ dependent protein/histone deacetylases (HDAC) are conserved from yeast to mammals. They carry out a broad range of crucial cellular functions ranging from transcriptional silencing to DNA damage response, cell cycle regulation, metabolism and aging etc. Their molecular functions in DNA metabolic processes such as DNA replication and repair has not been studied extensively. During some of these processes, the expression level of specific sirtuins are known to alter, indicating conditional regulation of these proteins. However, the molecular functions and mechanism of regulation of sirtuin sunder many of these conditions remain elusive.

Our aim is to understand the molecular functions and mechanism of regulation of sirtuins during DNA damage response and repair. We use yeast and human cell lines as model systems. Based on our findings in yeast, we would like to extend our working hypothesis to mammalian cells. There are seven sirtuins (SIRT1-7) in mammals. The mammalian sirtuins have different sub-cellular localization for e.g. SIRT1, SIRT6 and SIRT7 localizes to nucleus, SIRT2 to cytoplasm while SIRT3, SIRT4 and SIRT5 to mitochondria. Besides, a few sirtuins exhibit shuttling between different subcellular compartments and this distinct sub-cellular localization determines their function. Since fission yeast, *S. pombe* is more closely related to higher eukaryotes and sirtuins are conserved from yeast to mammals, we use fission yeast,*S. pombe* as a model systems to study sirtuin biology. Fission yeast, *S. pombe* has three sirtuins, Sir2, Hst2 and Hst4. Deletion analysis and other studies have shown that all these genes function in transcriptional silencing. However, deletion of only *hst4*+ gene, not *sir2*+ and *hst2*+ genes, show interesting phenotypes such as slow growth, elongated morphology, fragmented DNA and DNA damage sensitivity indicating it could have additional functions. These phenotypes are very useful tools to uncover novel signalling pathways where Hst4 could be functioning. It has been shown to function in maintenance of genome stability. Interestingly, the level of Hst4 decreases when cells are exposed to DNA damage.

We focused on the following objectives:

1) Understanding the molecular functions and mechanism of regulation of fission yeast sirtuin Hst4 during DNA damage response.
2) Investigation of nuclear localisation and function of human sirtuin 3 (SIRT3).

Project 1: Understanding the molecular functions and regulation of sirtuin family NAD+ dependent histone deacetylase Hst4 of fission yeast, *Schizosaccharomyces pombe*.

The expression of Hst4 decreases during the S phase of the cell cycle as well as when cells are exposed to DNA damage. The timely regulation of Hst4 is important for maintenance

of genomic integrity. However, the implication of Hst4 degradation, signaling mechanism and the molecular machinery required for its degradation on exposure to specific DNA damaging agents such as Methy methane sulphonate (MMS) are not known. This project is aimed at investigating mechanism of regulation of Hst4 during DNA damage stress and also, to gain further insights into the replication stress associated DNA damage pathway in fission yeast.

Summary of work done until the beginning of this reporting year (upto March 31, 2016)

HDACs are known to be regulated by different mechanisms. The kind of regulation depends on the specific functions. Our earlier work has shown that the levels of Hst4 decreases during S phase of the cell cycle and during DNA damage. Thus, to determine whether this decrease is due to transcriptional or translational regulation,the *hst4* transcript levels were checked by RT-PCRin untreated and MMS treated cells. We observed very little reduction in transcript level. Since the reduction was less than 2 folds, we hypothesized that the decrease in Hst4 level is mediated by post-translational regulation such as ubiquitination. In order to check the role of proteosome in the regulation of Hst4, half life of Hst4 was determined in the wild type and proteo some mutant (mts2-1) strain after cycloheximide treatment. The levels of Hst4 were stabilized in the proteosome mutant significantly as compared to wild type. Further the levels of Hst4 on DNA damage was checked in the mutant strain. There was no decrease in Hst4 level in *mts2-1* strain onMMS treatment as compared to wild-type strains. Thus, these results showed that Hst4 is regulated by ubiquitin mediated proteosomal degradation.

E3 ligases are the most important in ubiquitination as they specify the substrates targeted for ubiquitination. The SCF ubiquitin ligase is a conserved E3 ligase which regulates the expression of many cell cycle proteins which in turn regulates the G1/S switch. To check whether the SCF ubiquitin ligase is involved in the regulation of Hst4, stability of Hst4 protein was determined in SCF mutant strain. Hst4 was stabilized in SCF mutant significantly as compared to wild type. This was comparable to the stability of Hst4 observed in proteosomal mutants. Hst4 is also known to be down regulated when cells are exposed to DNA damaging agent MMS. To examine if decrease in the level of Hst4 on DNA damage is also mediated through SCF ubiquitin ligase, Hst4 levels were determined in SCF mutant by western blot in MMS treated cells. The level of Hst4 did not decrease on MMS treatment in SCF mutant. Further, the degradation of Hst4 was rescued by the plasmid complementation of SCF component back in the null background. Collectively, these results show that Hst4 is regulated by ubiquitin ligase SCF mediated proteolysis in response to DNA damage.

Details of progress made in the current reporting year (April 1, 2016 - March 31, 2017)

The above result showed the stabilization of Hst4 in the proteosome mutant, therefore, next we wanted to determine whether Hst4 is directly modified by Ubiquitination and targeted for degradation via proteosome. For this, we used a His-Ubiquitin pull down by Nickel affinity strategy. We over expressed His-tagged Ubiquitin in the proteasome mutant strain and looked for ubiquitinated Hst4 by western blot after pulling down the His-Ubiquitin using Nickel NTA beads. The experiment was performed with both untreated as well as MMS treatment cells. Untransformed strains were used as control. Figure 1A shows the higher mobility modified bands of Hst4 being visible in the proteosome mutant strain. Further, we found the bands were enhanced on MMS treatment. This result proves that Hst4 is modified by ubiquitination and thus confirming, its targeted degradation via 26S proteasome.

Covalent modification of proteins with ubiquitin plays an important role in a wide array of cellular processes. The E3 ubiquitin ligases are central to determining the timing and specificity of substrate proteolysis. There are two conserved ubiquitin ligases that regulate cell cycle progression: anaphase promoting complex/ cyclosome (APC/C) and Skp1-Cdc53/Cullin-1-F-box (SCF). APC/C helps in regulation of G2/M progression and SCF in G1/S transition. Since, Hst4 is highly abundant in G2/M phase and its levels go down S phase and on treatment with DNA damaging agents that cause replication stress, such as MMS, we hypothesized the role of SCF ubiquitin ligase complex in the regulation of Hst4. SCF ligases are multi-subunit E3 ligases and F box protein component of the complex dictates the specificity by interacting with the phosphorylated substrate. Figure. 1B and 1C show that Hst4 is stabilized on MMS treatment

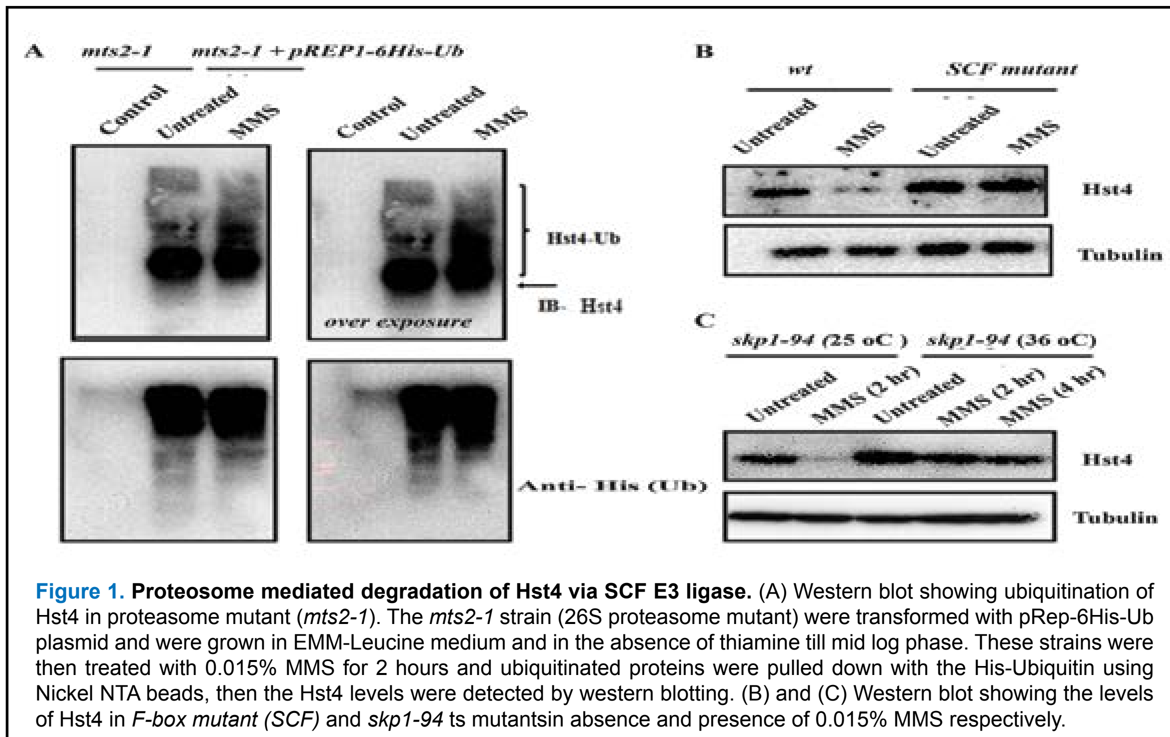

Figure 1. Proteosome mediated degradation of Hst4 via SCF E3 ligase. (A) Western blot showing ubiquitination of Hst4 in proteasome mutant (*mts2-1*). The *mts2-1* strain (26S proteasome mutant) were transformed with pRep-6His-Ub plasmid and were grown in EMM-Leucine medium and in the absence of thiamine till mid log phase. These strains were then treated with 0.015% MMS for 2 hours and ubiquitinated proteins were pulled down with the His-Ubiquitin using Nickel NTA beads, then the Hst4 levels were detected by western blotting. (B) and (C) Western blot showing the levels of Hst4 in *F-box mutant (SCF)* and *skp1-94* ts mutantsin absence and presence of 0.015% MMS respectively.

in both skp1(skp1-94) and F- box protein mutant (SCF mutant) strains where the components of SCF ligase complex were inactivated. Work is underway to determine whether degradation of Hst4 on DNA damage is phosphorylation dependent as SCF complex recognize phosphorylated substrate proteins and if the degradation of Hst4 is mediated by DNA damage checkpoint proteins.

Project 2: Investigation of nuclear localisation and function of human sirtuin 3 (SIRT3).

Mammalian sirtuins have a conserved HDAC domain and flanking N and C terminal domains. The subcellular localization is regulated by the presence of NES or NLS at either the N or C-terminal domains, for example, the import and export of SIRT1 and SIRT2 into the nucleus is dependent on nuclear localization sequence (NLS) and nuclear export sequence (NES) respectively. For instance, SIRT1 on phosphorylation by JNK-1 enters the nucleus, inside the nucleus it has important substrates, like NF-kB subunits and histone marks, H3K56ac, H3K9ac, H4K16ac etc., while in cytoplasm, it deacetylates acetyl-CoA synthase 1 and hydroxy-3-methylglutaryl CoA synthase 1.Similarly, SIRT2 which is primarily cytoplasmic, moves to the nucleus during mitosis and deacetylates H4K16ac.Human SIRT3 (hSIRT3) is a major mitochondrial deacetylase that deacetylates acetyl-CoA-synthetase (AceCS), glutamate dehydrogenase (GDH), succinate dehydrogenase and complex I functioning in mitochondria. Few reports have shown that the full-length SIRT3 (FL-SIRT3) also localizes to nucleus and functions as transcriptional regulators of nuclear genes regulating metabolic processes in mitochondria. It deacetylates Ku70 and abrogates Ku70-Bax interaction and regulate the transcription of stress related genes as well.

This is a new activity, which aims to understand nuclear functions of mammalian sirtuin, SIRT3. In an earlier study, we observed that overexpression of human SIRT2, SIRT3 and SIRT6 in HEK cells resulted in reduction of acetylation of H3K56 levels, which is a known core domain histone H3 modification. SIRT2 and SIRT6 localizes to nucleus, SIRT3, however, was reported to reside mostly in mitochondria but few studies had indicated it could have nuclear functions as well. Thus, we propose to investigate and decipher novel human SIRT3 interacting proteins in the nucleus and determine its nuclear functions.

Details of progress made in the current reporting year (April 1, 2016 - March 31, 2017)

The nucleus to mitochondrial translocation of SIRT3 is dependent on its mitochondrial translocation sequence (MTS). During an earlier study, we observed that the overexpression of SIRT3 resulted in reduction of H3K56ac levels

indicating, it could be a potential substrate of SIRT3. Thus, to confirm the nuclear localization of SIRT3, HeLa cells were treated with leptomycin B (LMB), which specifically inhibits CRM1 dependent nuclear export, and IF was performed using antibody against SIRT3 at indicated time points to observe its localization (Figure2A). The increased levels and retention of SIRT3 in the nucleus was observed in a time dependent manner, 120 mins showed the maximum retention inside the nucleus. The NES containing proteins are exported to the cytoplasm in a CRM-1 dependent manner and this export is inhibited by treatment with LMB. Since, SIRT3 retained inside the nucleus on LMB treatment, therefore, we checked the presence of NES sequence in the SIRT3 protein using NES prediction software (Net NES1.1 Server). The predicted NES with a score above 0.5 were selected and aligned with previously known similar NES containing proteins (Figure 2B). The NES was predicted to be present between the amino acids 314 to 324 of SIRT3 and contains a cluster of hydrophobic amino acids. To map the SIRT3 NES, a GFP-tagged SIRT3 deletion construct lacking the C-terminal region (amino acid 314-399) was generated (Figure 2C). The wild type SIRT3 and the deletion constructs were overexpressed in HeLa cells by transient transfection and the percentage of transfected cells with nuclear SIRT3 were counted. As shown in (Figure 2D and E), around 94% of cells overexpressing (NES Δ 314-399) showed nuclear retention. Next, to identify the hydrophobic residues crucial

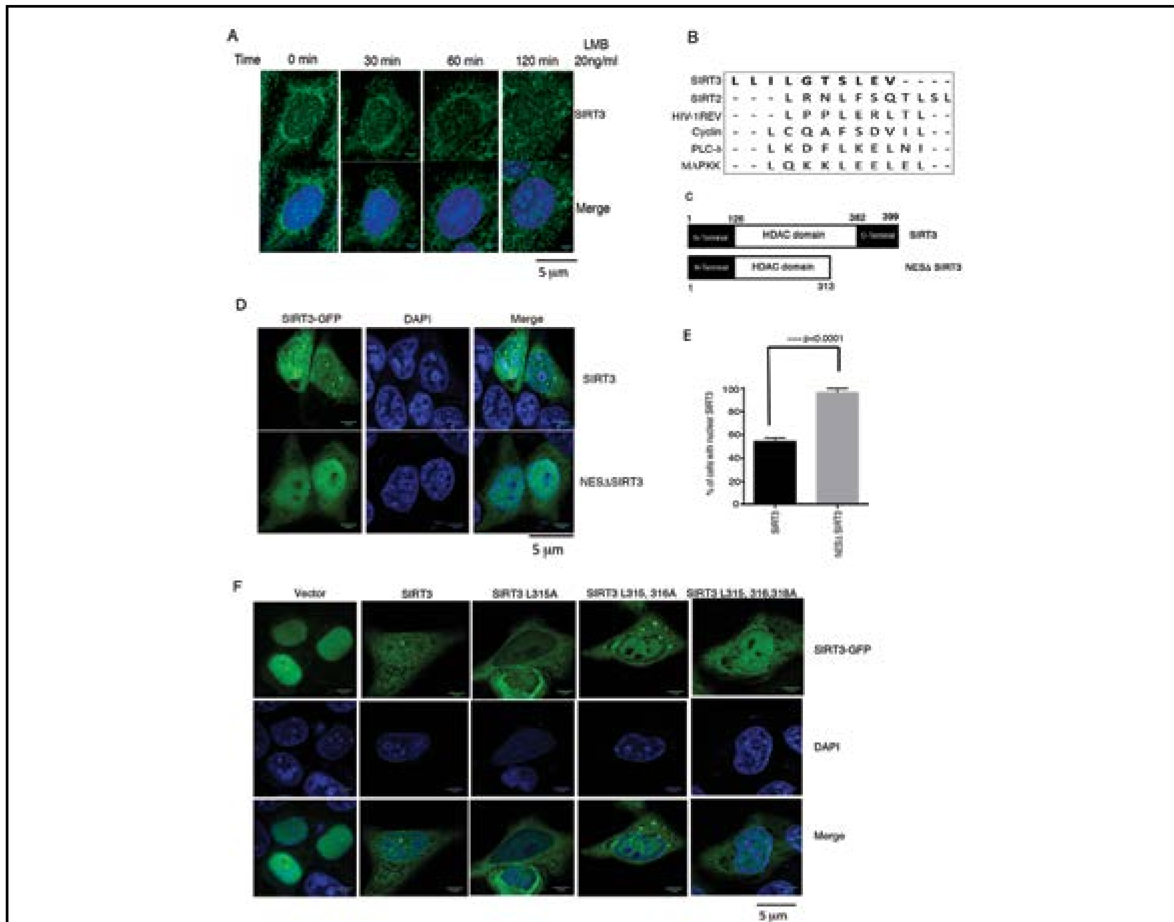

Figure 2. Nuclear export sequence shuttles SIRT3 from nucleus to cytoplasm. ***(*A*)*** Immunofluorescence (IF) of HeLa cells treated with 20ng/ml of Leptomycin B at an indicated time points. Endogenous SIRT3 staining was performed using antibody against SIRT3 and the nucleus was stained with DAPI. (**B)** Multiple sequence alignment of SIRT3 NES with known NES using CLUSTAL OMEGA tool. (**C**) Schematic diagram to compare the sizes of SIRT3 and SIRT3 deletion construct. (**D**) IF of HeLa cell lines overexpressing GFP -SIRT3 constructs as indicated. Transiently overexpressed cells were counterstained with DAPI and visualized under confocal microscope. (**E**) 200 cells for each overexpressed construct were counted and percentage of cells with nuclear localization of SIRT3 was plotted using Graph pad Prism software. (**F**) IF of HeLa cell lines overexpressing GFP tagged SIRT3 mutant constructs as indicated. The cells were stained with DAPI and visualized under confocal microscope.

for NES function, the first three leucine residues in the predicted NES were mutated to alanine [(L315A), (L315, 316A) and (L315, 316, 318A)] using site-directed mutagenesis. The GFP tagged mutation constructs were generated and expressed, GFP expression was quantified as percentage of cells expressing mutant SIRT3 in cytoplasm alone (%C), in nucleus alone (%N) and both in cytoplasm and nucleus (%C+N). The SIRT3 mutants (L315A) and (L315, 316A) exhibited similar localization with ~ 60 % of cells showing both cytoplasmic and nuclear localization. However, 94% of SIRT3 mutant (L315, 316, 318A) was detected in nucleus alone, indicating amino acids 315-324 contains the NES as shown in (Fig 2F). These results confirm presence of NES in SIRT3, disruption of which restricts it in the nucleus. Overall, these results demonstrate a novel NES dependent shuttling mechanism of SIRT3 which shuttles it from the nucleus to cytoplasm.

Publications

Research paper

Ghosh A, Sengupta A, Seerapu GPK, Ali N, Ramarao EVVS, Bung N, Bulusu G, Pal M and Haldar D (2017) A novel SIRT1 inhibitor, 4bb induces apoptosis in HCT116 human colon carcinoma cells partially by activating p53. (2017) Biochem. Biophys. Res. Commun.488 (3), 562–569.

LABORATORY OF COMPUTATIONAL BIOLOGY

Computational studies on protein structure, function and interactions

Faculty	H A Nagarajaram	Staff Scientist (on lien from Feb. 2017)
PhD Students	Suryanarayana Seera	Senior Research Fellow
	V A Ramesh	Senior Research Fellow
	Rakesh Trivedi	Senior Research Fellow
	Arijita Mitra	Junior Research Fellow
	K Guruprasad	Junior Research Fellow
Other Members	Rahul Dhakne	Project JRF (till Feb. 2017)
	Rajkishore Mohapatra	Project JRF (till Oct. 2016)
	U Raghavender	DST-SERB Young Scientist
Collaborators (The New Indigo Project):		
	Srikanth Rapole	NCCS, Pune
	Jochen Schubert	University of Rostock, Germany
	Josè Càmara	University of Madeira, Portugal

Objectives

1. Sequence and structural analyses of disease causing mutations in human proteins
2. Investigations on the evolution and the conformational heterogeneity of instrinsically disordered regions in proteins
3. Understanding the presence and role of mutations at the interfaces of protein-peptide complex structures.
4. A prototype relational database was designed and built to hold the data of volatile compounds detected in human breath, saliva and urine samples.

Summary of work done until the beginning of this reporting year (April 1, 2015 – March 31, 2016)

1. A new version of HANSA was built, and its performance was evaluated, for predicting the functional impact (as disease or benign) of missense mutations in human proteins using their network centrality values in protein-protein interaction network.
2. Studies were performed toward development of a tool for predicting the functional impact of missense mutations in the disordered regions of proteins. For this amino acid conservation index as measured by Jensen-Shannon divergence (JSD) information was tested for its utility as a discriminating feature.
3. Studies were carried out with an aim to build a novel substitution scoring matrix reflecting substitution frequencies of amino acid residues in the intrinsically disordered regions of proteins.

Details of progress made in the current reporting year (April 1, 2016 - March 31, 2017)

Project 1: Computational Studies on Intrinsically Disordered Proteins (IDPs) harboring disease causing missense mutations

1. It is known that some disease causing missense mutations are found in the intrinsically disordered regions of proteins. It is thought that these mutations affect the intrinsic conformational heterogeneity of the disordered regions and thereby affect their biological roles. In order to investigate the effect of disease causing mutations on the intrinsic conformational heterogeneity of disordered regions we carried out MD simulation studies (100ns) on the C-terminal segment of RIP domain of RPGRIP1 with the D1114G missense mutation (Fig.1) in conjunction with the wild-type.
2. Analysis of MD tranjectories revealed that the disordered region in the wildtype displays higher conformational variability than its disease mutant form. Cluster analysis of the snapshots saved during simulations indicated that the mutant form adopts very few conformational states of which one is found about 70% of the simulation time, whereas the wildtype adopts several

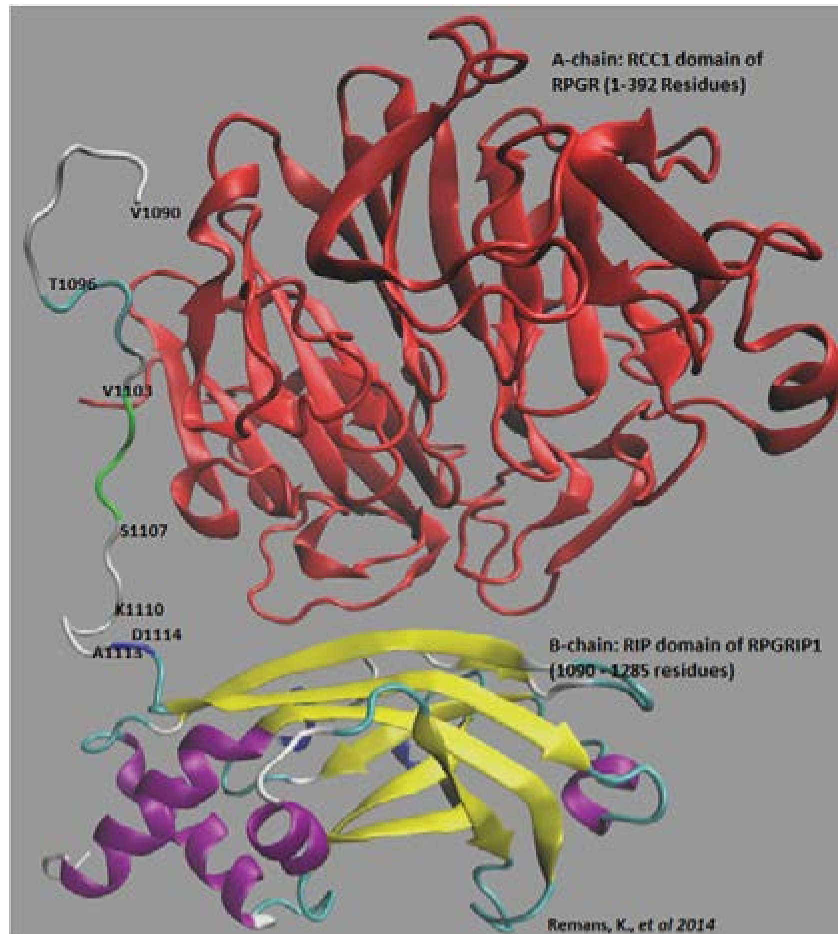

Figure 1. Complex of RCC1 domain of RPGR (A-chain) and RIP domain of RPGRIP1 (B-chain). The disordered region of our interest is V1090 to D1114 present in the B-chain. It has been proposed that D1114G mutation disrupts the interaction between the domains and leads to an abnormal condition called Laber congenital amaurosis 6, where the retina is severely damaged. The MD simulation studies were carried out on the wildtype peptide as well as the disease mutant form. GROMACS suite was used for MD simulations with OPLS force-field parameters and for various analyses of trajectories.

transient conformational states suggesting that the disease causing mutation affects the intrinsic conformational heterogeneity of the peptide. Further investigations revealed that G in the mutant undergoes a conformational transition (which otherwise not possible for the wildtype D), which further gets stabilized by intra segmental hydrogen bonds. In the wild-type this conformational transition is stereochemically precluded because of D at the position and hence the domain remains conformationally very mobile.

Project 2: Computational Studies on Intrinsically Disordered Proteins (IDPs): Construction of substitution scoring matrix specific to disordered regions

Multiple sequence alignments of only higher eukaryotic proteins harboring disordered regions, belonging to 4198 families were obtained. From the aligned blocks three different matrices viz., ordered, disordered and order-disordered (mixed regions) substitution scoring matrices were compiled using the well known Henikoff`s method (Henikoff and Henikoff, 1992). The matrices were compared with BLOSUM62 and those previously developed for disordered proteins. The relative entropy (H), expected scores (E) and Matrix average values revealed that the newly calculated matrices have better scores than the previously published matrices.

Further studies of refining the matrices and their performance evaluation are underway.

Project 3: Development of SVM-based tool for prediction of functional impact of missense mutations in disordered regions

The present version of HumVar dataset shows 1,722 disease mutations in the disordered regions of human proteins indicating that disordered regions also harbor a substantial number of disease causing missense mutations and hence calls for development of a predictive tool specific to the mutations in the disordered regions. This is because the prediction tools currently available, including HANSA developed by us, are largely based on features that characterize ordered regions. In order to train a SVM model suitable for mutations in disordered regions, as a first attempt, we considered only the position-specific residue propensity features (a total of 4 features). The SVM model so built was evaluated by performing 10-fold cross-validation studies. We also performed 10-fold cross validation of HANSA on the same dataset of mutations in the disordered regions. Comparison of HANSA and the SVM model built only for the disordered regions revealed that the latter was, surprisingly, performing poorly as compared to the former (the AUC values for HANSA and the SVM built for disordered regions are 0.88 and 0.82 respectively) indicating that the features set considered for disordered regions is not sufficient. Further studies are underway.

Future plans and directions

1. Continuation of studies on IDPs harboring disease causing mutations
2. Classification and analysis of disordered regions in proteins
3. Studies on tissue-wise PPI networks integrated with drug-protein interaction data

Publications

1. Kiran M and Nagarajaram H A (2016) Interaction and Localization Diversities of Global and Local Hubs in Human Protein-Protein Interaction Network ***Molecular Biosystems*** 12: 2875 – 2882
2. Radha Rama Devi A, Ramesh V A, Nagarajaram H A, Satish S.P.S, Jayanthi U, and Lingappa L (2016) Spectrum of Mutations in Glutaryl-CoA Dehydrogenase gene in Glutaric Aciduria Type I - Study from South India ***Brain & Development*** 38: 54-60
3. Chaudhary A K, Mohapatra R, Nagarajaram, H A, Ranganath P, Dalal A, Dutta A, Danda S, Girisha K, and Bashyam M D (2016) The novel missense EDAR p.L397H mutation causes autosomal dominant hypohidrotic ectodermal dysplasia. ***Journal of European Academy of Dermatology and Venearology*** 31:e17-e20

Other publications

1. *Advanced Computing and Communication Technologies Proceedings of the 9th ICACCT, 2015* Choudhary, R K, Mandal, J K, Auluck, N, Nagarajaram, H A (Eds.) Advances in Intelligent Systems and Computing, Springer (2016)

LABORATORY OF COMPUTATIONAL AND FUNCTIONAL GENOMICS

Computational and functional genomics of biological organisms

Faculty	Akash Ranjan	Staff Scientist
PhD Students	Mr. Rohan Misra	Senior Research Fellow
	Mr. Bhavik Sawhney	Senior Research Fellow (till Sept. 2016)
	Mr. Ajit Roy	Senior Research Fellow
	Mr. Rajendra Kumar Angara	Senior Research Fellow
	Mr. Abhishek Kumar	Senior Research Fellow
	Mr. Debasish K Ghosh	Senior Research Fellow
	Mr. Shailesh Kumar Gupta	Senior Research Fellow
	Mr. S. Akshaykumar Nanaji	Junior Research Fellow
Other Members	Dr. U S Raghavender	SERB-DST Young Scientist
	Ms Archana Tomar	Bioinformatician
	Mr. G Rajalingam	Skilled Work Assistant
	Mr. Jenige Aravindh Kumar	Lab Help
Collaborators	Anthony Addlagatta	CSIR-IICT Hyderabad, India
	Lothar H Wieler	Robert Koch Institute, Berlin, Germany.
	M. Sritharan	University of Hyderabad, Hyderabad, India.
	V. Vindal	University of Hyderabad, Hyderabad, India.

Objectives

The primary research objective of our group is to understand the cellular functions coordinated by regulatory genes encoded in various genomes. We use a combination of computational and experimental approaches to achieve our goal.

Project 1. Structure-function studies of *Escherichia coli* transcription regulator HosA and its complexes with cognate DNA & 4-hydroxy benzoic acid

Summary of work done until the beginning of this reporting year (upto March 31, 2016)

In the previous studies, the structure of an *Escherichia coli* MarR type transcription regulator, HosA was solved by us at resolution of 2.92Å. The structure showed presence of helix-wing-helix type of conformation.

Details of progress made in the current reporting year (April 1, 2016 - March 31, 2017)

This year, we had successfully crystallised co-complexes for HosA with its cognate DNA and its ligand 4-hydroxy benzoic acid (PHBA). The co-crystals were diffracted at the Synchrotron facility (INDUS-II beam line, RRCAT, Indore, India) with highest resolution of 2.42Å. The diffracted structures were solved in Coot (Figure 1). In the HosA-PHBA structure, the PHBA was found to be interacting with the HosA at the dimerization domain. Such binding of PHBA would have impact on dimerization stability of HosA and subsequent DNA binding activity since only dimer form of HosA is compatible with DNA binding. HosA-DNA complex showed how the protein exactly recognized the palindrome in the DNA.

Project 2. Functional studies on Rv2989 (an IclR-like protein) in the physiology of *M. tuberculosis*

Summary of work done until the beginning of this reporting year (upto March 31, 2016)

In our previous studies, we had characterised promoter and binding site of Rv2989 (an IclR like protein) in the intergenic region of *leuCD-Rv2989*. Using acetamide inducible expression system, we found that Rv2989 expression triggers growth arrest in *M. smegmatis*. However, the growth arrest was not because of leucine auxotrophy. The growth arrested cells were elongated, non-acid-fast and with intracellular lipid vacuoles suggesting an early dormancy like stage.

Details of progress made in the current reporting year (April 1, 2016 - March 31, 2017)

In the current study, we examined the progression of dormancy like phenotype using

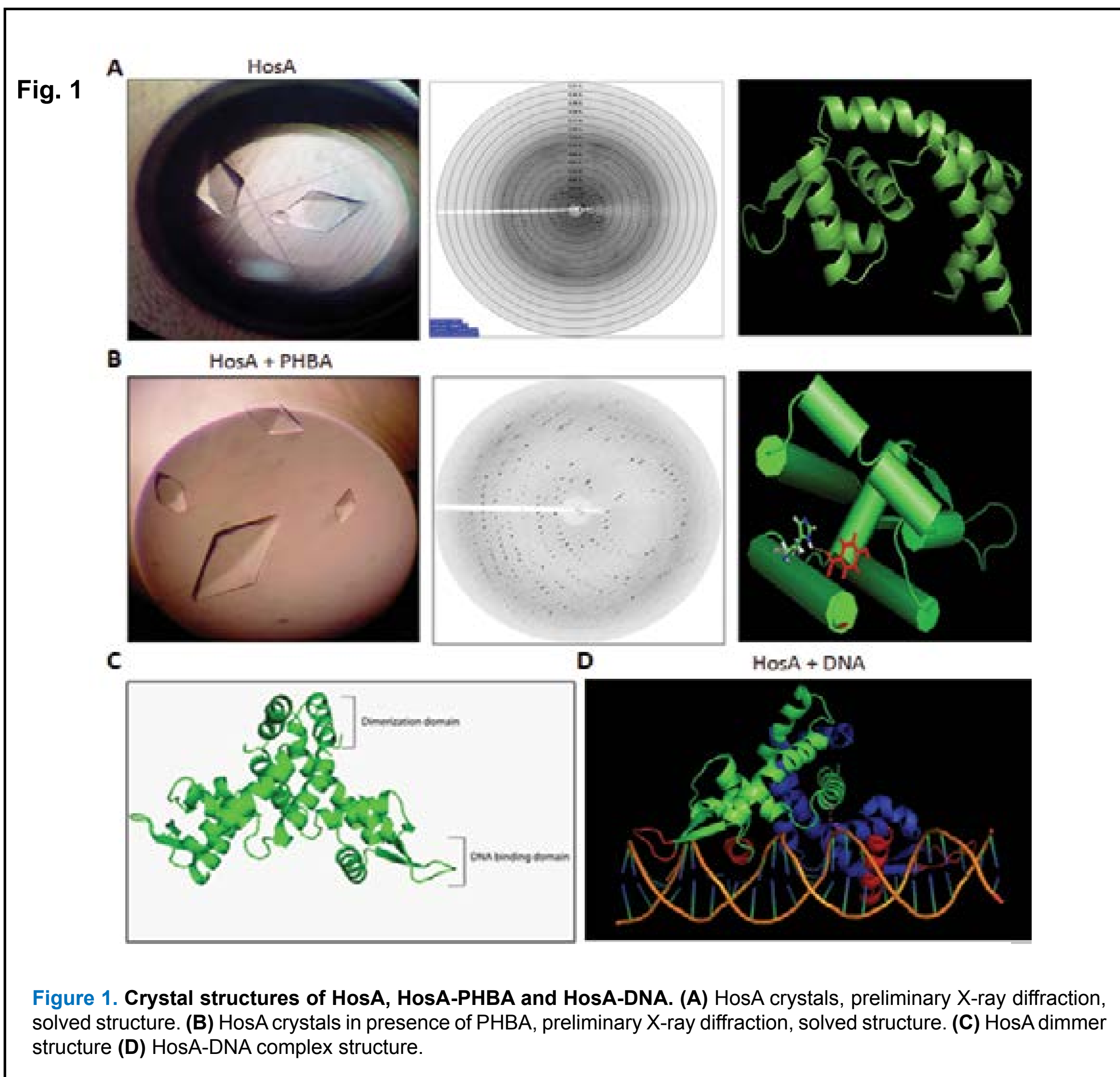

Figure 1. Crystal structures of HosA, HosA-PHBA and HosA-DNA. (A) HosA crystals, preliminary X-ray diffraction, solved structure. **(B)** HosA crystals in presence of PHBA, preliminary X-ray diffraction, solved structure. **(C)** HosA dimmer structure **(D)** HosA-DNA complex structure.

additional markers. In order to meet the energy requirements during dormancy, it is well known that mycobacteria stores triacylglycerol (TAG) in the form of lipid droplets. Nile red, a lipophilic stain was used to reveal accumulation of neutral lipid inside the cell. We applied this staining procedure to confirm the TAG accumulation in *M. smegmatis* pJV2989 growth arrested cells. When we induced Rv2989 expression using 0.2% acetamide in the media, unlike *M. smegmatis* pJV2989 uninduced cells, *M. smegmatis* pJV2989 induced cells showed positive staining for Nile red, indicating an accumulation of lipid droplets (Figure 2A). It is well known that the accumulation of intracellular lipid droplets influences the buoyant density of cells. In order to understand the progression of accumulation of lipid droplets, induced cells collected at different time points were centrifuged in a Percoll density gradient. We observed that, uninduced cells (at 0hr of induction) remain close to the bottom (region of higher density) of buoyant density gradient while the induced cells shifted towards lower buoyant density region in the upper phase of the tube (Figure 2B). The shift in the bands of induced cells increased with increase in incubation time after induction, reflecting an increase in accumulation of lipid droplets. These changes are consistent with our conclusion that induction of Rv2989 expression caused progressive changes in lipid accumulation resulting in increasing percentage of cells with dormant features.

Fig. 2

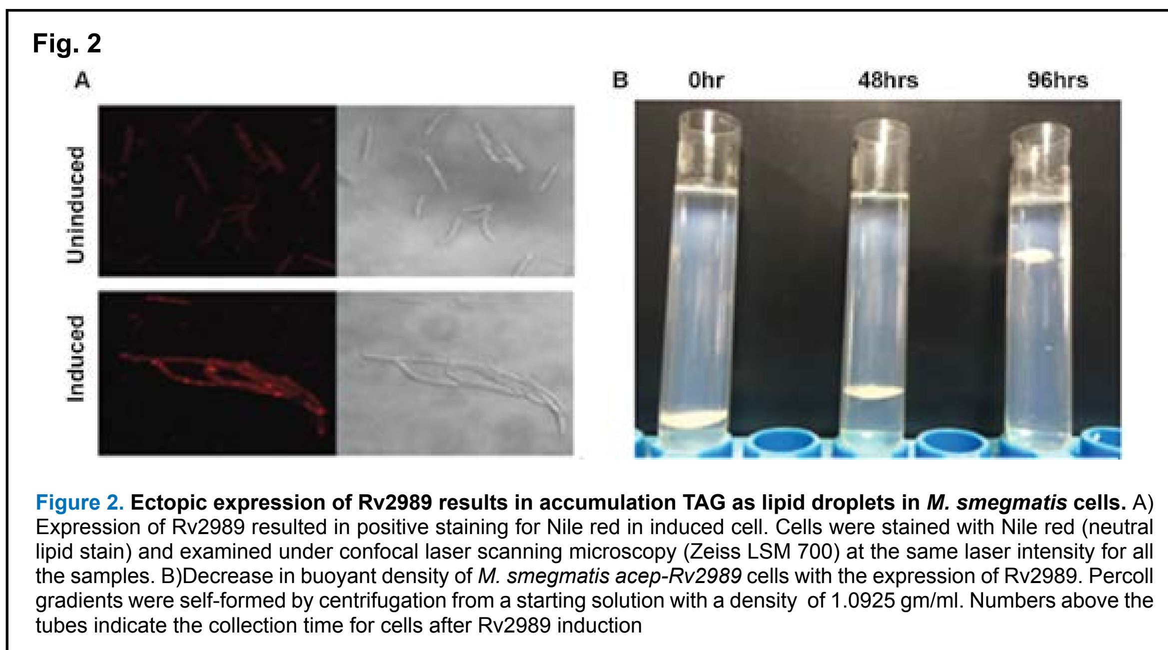

Figure 2. **Ectopic expression of Rv2989 results in accumulation TAG as lipid droplets in *M. smegmatis* cells.** A) Expression of Rv2989 resulted in positive staining for Nile red in induced cell. Cells were stained with Nile red (neutral lipid stain) and examined under confocal laser scanning microscopy (Zeiss LSM 700) at the same laser intensity for all the samples. B)Decrease in buoyant density of *M. smegmatis acep-Rv2989* cells with the expression of Rv2989. Percoll gradients were self-formed by centrifugation from a starting solution with a density of 1.0925 gm/ml. Numbers above the tubes indicate the collection time for cells after Rv2989 induction

Project 3. Characterisation and functional studies of FadR like proteins from *M. tuberculosis*

Summary of work done until the beginning of this reporting year (upto March 31, 2016)

FadR proteins have been shown to play significant roles in cellular physiology and virulence. *M. tuberculosis* genome encodes five proteins (Rv0043c, Rv0165, Rv0494, Rv0586 and Rv3060c) belonging to the FadR family. We identified binding sites of Rv0494 and Rv0586 and further characterised Rv0494 as auto-regulatory, lipid responsive and starvation inducible.

Details of progress made in the current reporting year (April 1, 2016 - March 31, 2017)

In the current study, we characterised Rv3060c which is interesting because of unusual size (54kDa) among the FadR family of proteins. We identified Rv3060c as an auto-regulator like other FadR proteins (Figure 3A). We further tested its regulatory role on adjacent genes present in the neighbourhood. To test regulatory activity, 300bp upstream of target genes were amplified and cloned in pEJ414 reporter vector. Using β–galactosidase assay, the target gene expression in presence and absence of ectopically overexpressed Rv3060c was evaluated in *M. smegmatis.* Among all neighbouring genes, *ligB* and *fadE22* were negatively regulated (approximately two fold) by Rv3060c (Figure 3B and 3C). The gene *ligB* encodes a probable ATP-dependent DNA ligase and FadE22 is a probable acyl-CoA dehydrogenase. Other proteins of FadR family (Rv0165, Rv0494 and Rv0586) were taken as negative control and they didn't show any effect on *ligB* and *fadE22* expression.

Project 4. Functional studies on Huntingtin Interacting Protein K (HYPK)

Summary of work done until the beginning of this reporting year (upto March 31, 2016)

Earlier, we had characterised Huntingtin Interacting Protein K (HYPK) as a sensor and global regulator of toxic aggregating proteins like Huntingtin, α-Synuclein A53T and SOD1-G93A. We had identified a unique macro-molecular complex of HYPK named 'Annulosome' that sequesters other different toxic aggregates. The Prion-like properties of HYPK mediate the sequestration process. The molten globule state of HYPK results in high oligomerization that changes the nature of aggregation from annular to amorphous. While the UBA domain associated hydrophobic regions in HYPK cause annular

Fig. 3

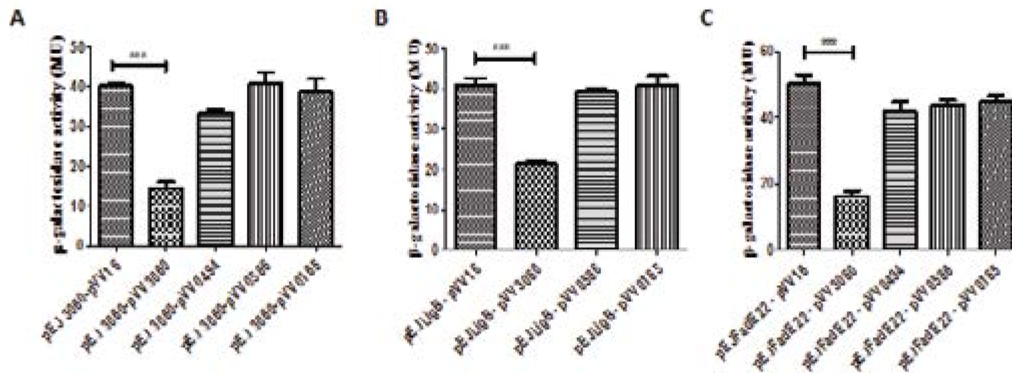

Figure 3. Ectopic expression of Rv3060c regulates its own promoter, *ligB,* and *fadE22*. (A) β-galactosidase activityof *Rv3060c* promoter in the presence and absence of ectopically overexpressed FadR proteins. Only Rv3060c reduced the expression of Rv3060c promoter by approximately three fold. **(B)** β-galactosidase activity of *ligB* promoter in the presence and absence of ectopically overexpressed FadR proteins. Only Rv3060c reduced the β-galactosidase activity from *ligB* promoter by approximately two fold. **(C)** β-galactosidase activity of *fadE22* promoter in the presence and absence of ectopically overexpressed FadR proteins. Only Rv3060c reduced the β-galactosidase activity from *lig*B promoter by approximately two fold.

oligomerization, the low complexity region (LCR) cause transition of annular oligomers to amorphous aggregates by charge interaction and helix-associated patch collapse. The unstructured N-terminal region of HYPK contains a negative charge-rich patch which loops back to interact and shield the LCR and prevent aggregation under physiological conditions. Not only does HYPK sequester toxic aggregates but it also reduces the total load of these proteins.

Details of progress made in the current reporting year (April 1, 2016 - March 31, 2017)

This year, we deciphered the mechanism by which HYPK reduces toxic aggregates at annulosome platform. HYPK augments a unique protein clearance pathway – 'Neddylation dependent autophagy'. While finding the scaffolding role of HYPK in autophagosome complex formation, we identified this novel phenomenon of neddylation dependent autophagy of aggregates. Huntingtin (Htt) can get poly-neddylated at all the three lysine residues (that are K6, K9 and K15) in the N-terminal region of exon1 (Figure 4). While the poly-neddylated Huntingtin can be degraded by proteasomal pathway, interestingly, we found that they can also be degraded by autophagic pathway. Huntingtin poly-neddylation show LC3 conversion and increase in Benclin-1 expression which are characteristic of autophagic induction. Poly-neddylated Huntingtin also show distinct co-localization with autophagy markers like LC3, ATG5, ATG12, and ATG16L1. While the K48 linkage in poly-neddylation cause proteasomal degradation, the K60 linkage of poly-neddylated Huntingtin drive autophagy. However, Huntingtin can also be neddylated by K27 linked Nedd8. Htt-K6 residue is marked for K48 linked neddylation and Htt-K15 is subject of K60 linked neddylation. In conclusion, our study revealed a novel pathway of Huntingtin aggregate clearance by poly-neddylation dependent autophagy.

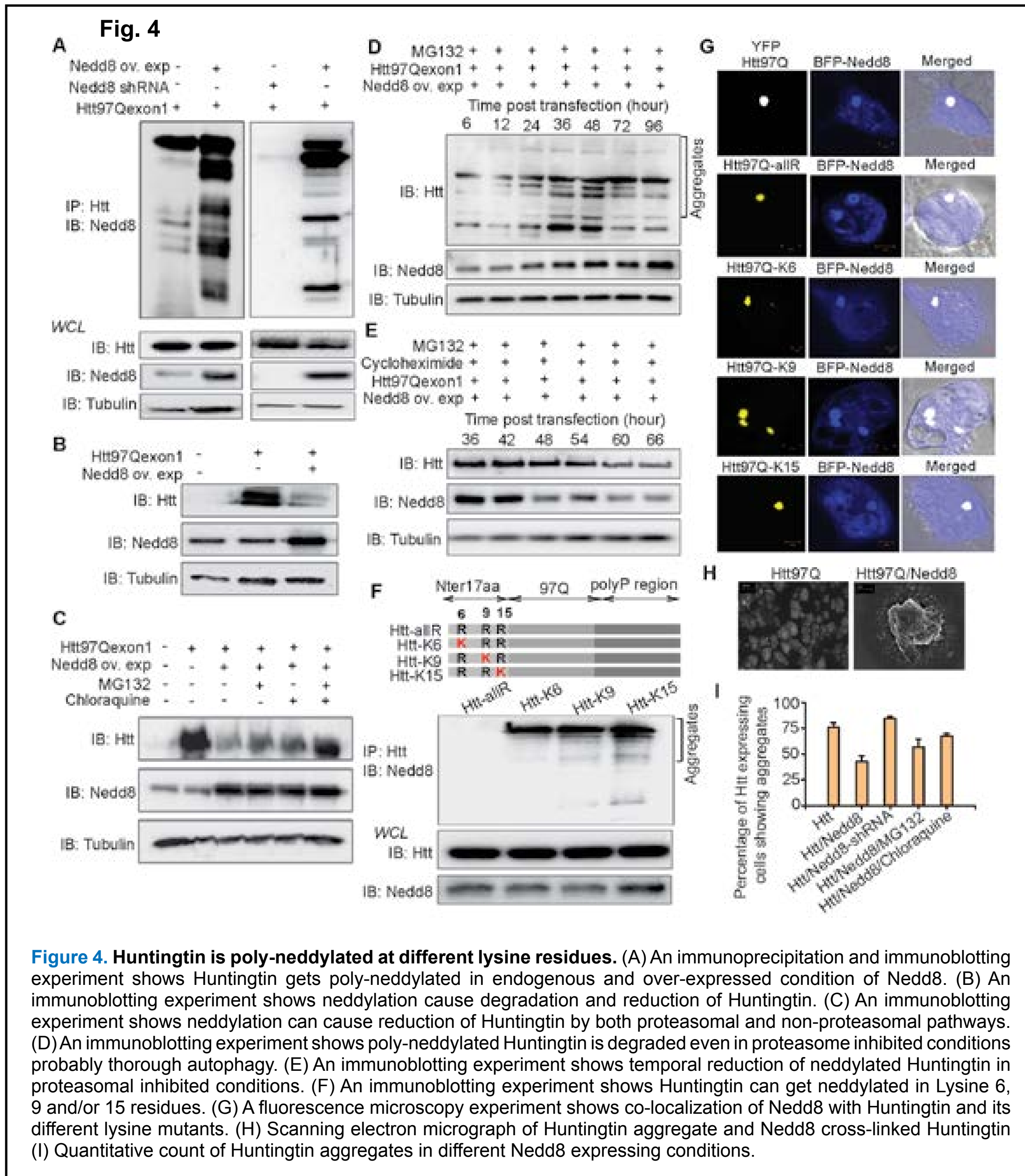

Figure 4. Huntingtin is poly-neddylated at different lysine residues. (A) An immunoprecipitation and immunoblotting experiment shows Huntingtin gets poly-neddylated in endogenous and over-expressed condition of Nedd8. (B) An immunoblotting experiment shows neddylation cause degradation and reduction of Huntingtin. (C) An immunoblotting experiment shows neddylation can cause reduction of Huntingtin by both proteasomal and non-proteasomal pathways. (D) An immunoblotting experiment shows poly-neddylated Huntingtin is degraded even in proteasome inhibited conditions probably thorough autophagy. (E) An immunoblotting experiment shows temporal reduction of neddylated Huntingtin in proteasomal inhibited conditions. (F) An immunoblotting experiment shows Huntingtin can get neddylated in Lysine 6, 9 and/or 15 residues. (G) A fluorescence microscopy experiment shows co-localization of Nedd8 with Huntingtin and its different lysine mutants. (H) Scanning electron micrograph of Huntingtin aggregate and Nedd8 cross-linked Huntingtin (I) Quantitative count of Huntingtin aggregates in different Nedd8 expressing conditions.

Publications

1. Roy A, Reddi R, Sawhney B, Ghosh DK, Addlagatta A, and Ranjan A. (2016) Expression, Functional Characterization and X-ray Analysis of HosA, A Member of MarR Family of Transcription Regulator from Uropathogenic Escherichia coli. ***Protein Journal***. 35(4):269-282.

2. Roy A, and Ranjan A. (2016) HosA, a MarR Family Transcriptional Regulator, Represses Nonoxidative Hydroxyarylic Acid Decarboxylase Operon and Is Modulated by 4-Hydroxybenzoic Acid. ***Biochemistry*** 55(7):1120-34.

Laboratory of *Drosophila* Neural Development

Understanding patterning and development of Central Nervous System using *Drosophila melanogaster*

Faculty	Rohit Joshi	Staff Scientist-Wellcome Trust DBT India Alliance Intermediate Fellow
Ph D Students:	Risha Khandelwal	Senior Research Fellow
	Neha Ghosh	Senior Research Fellow
	Ravi Ranjan	Senior Research Fellow
	Rashmi Sipani	Senior Research Fellow
	Asif Ahmad Bakshi	Junior Research Fellow
Other Members:	P. Kalyani	Technical Officer (till July 2016)
	Chandra Shekhar Singh	Technical Assistant (since Aug. 2016)
	Bijaylaxmi Swain	Project Assistant (since Jan. 2017)

Objective

The key objective of the lab is to understand how neural progenitor cells attain their positional identity in developing Central Nervous System **(CNS)** of an organism and how does this translate into generation of a variety of cell types found in CNS and their respective numbers (as represented in the Figure 1). Hox family of transcription factors are known to play an important role in execution of these features along the Anterior-Posterior (**AP**) axis of the CNS during development. Drosophila CNS comprise of two optic lobes, brain and ventral nerve cord (VNC). The molecular basis of role of Hox genes in patterning VNC of the CNS is not well investigated. Our lab is using *Drosophila melanogaster* as a model organism, to understand these phenomena by focusing mainly on early embryonic and larval stages of development. To this end, the specific aims of our lab are as follows:

1. Understanding the molecular function of Hox gene *Abdominal-A (Abd-A)* in larval CNS patterning.

Abdominal region of the *Drosophila* larval CNS has less number of neurons compared to its thoracic counterpart. This is because Hox gene *Abd-A* is known to cause programmed cell death (apoptosis) of neural progenitor cells (also called Neuroblasts-**NBs**) and therefore limit the number of neurons in abdominal region of CNS. The apoptosis is known to be mediated through activation of *reaper, hid and grim* (RHG) family of genes. The precise molecular details of how *Abd-A* cause NB apoptosis are unknown. Genetic evidence suggests a role for a helix-

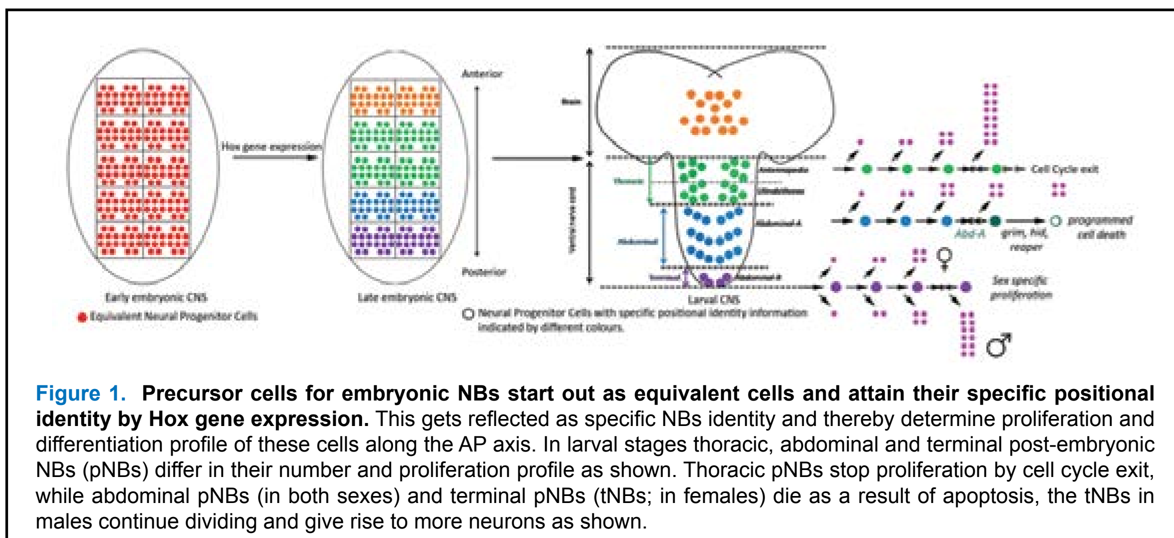

Figure 1. Precursor cells for embryonic NBs start out as equivalent cells and attain their specific positional identity by Hox gene expression. This gets reflected as specific NBs identity and thereby determine proliferation and differentiation profile of these cells along the AP axis. In larval stages thoracic, abdominal and terminal post-embryonic NBs (pNBs) differ in their number and proliferation profile as shown. Thoracic pNBs stop proliferation by cell cycle exit, while abdominal pNBs (in both sexes) and terminal pNBs (tNBs; in females) die as a result of apoptosis, the tNBs in males continue dividing and give rise to more neurons as shown.

loop-helix transcription factor Grainyhead (Grh) along with Abd-A in control of this apoptosis. Characterization of the molecular basis of this link is primary goal of this project. Furthermore, since Grh is involved in NB apoptosis and is not expressed in neuronal progeny refractory to this apoptosis, it is of interest to define *grh* regulation in these cells which keeps *grh* "on" in the pNBs and "off" in the neuronal progeny of pNBs.

2. Understanding the role of Hox gene *Deformed* (*Dfd*) in patterning of embryonic subesophageal ganglia.

Hox genes express in neural progenitor cells of CNS during embryonic stages of development (as represented in Figure 1) but how does their expression patterns the nervous system is not well understood. *Deformed (Dfd)* is known to express in the cells of maxillary (Mx) and mandibular (Mn) segments of subesophageal ganglion (SEG) of embryonic and larval CNS (Figure 2). This project focuses on understanding how Dfd patterns CNS. We study the auto-regulation of *Dfd* in the embryonic SEG region and role of Dfd in larval SEG to understand its role in CNS patterning. Former is being done by analyzing a 3.2kb CNS specific neural auto-regulatory enhancer for *Dfd* (NAE3.2), which recapitulates the expression of *Dfd* gene in developing embryonic CNS and latter is being investigated in context of Dfd mediated NB apoptosis in larval stages.

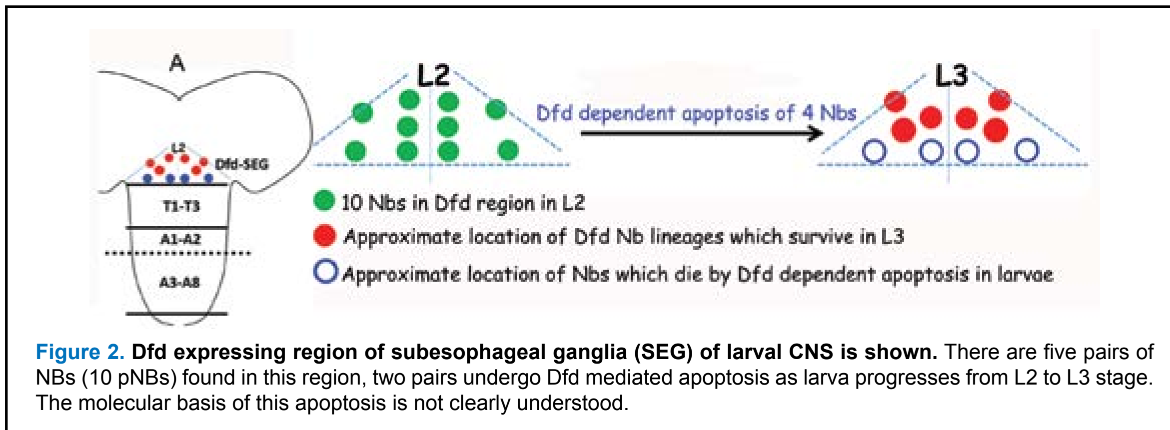

Figure 2. Dfd expressing region of subesophageal ganglia (SEG) of larval CNS is shown. There are five pairs of NBs (10 pNBs) found in this region, two pairs undergo Dfd mediated apoptosis as larva progresses from L2 to L3 stage. The molecular basis of this apoptosis is not clearly understood.

3. Investigating the role of *Abdominal-B (Abd-B)* and *Double-sex (Dsx)* in terminal CNS patterning.

AbdB expresses in the terminal region of VNC. There are 12 NBs in this region 8 of these stop dividing in both males and females at mid L3 stage of development. The remaining 4 NBs which we refer to as sex-specific terminal NBs (**tNBs**) express transcription factor Doublesex (Dsx). These Dsx+ tNBs die in females in early larval stages and continue dividing in males till late larval stages, giving rise to male specific neurons. Dsx is the most downstream member of sex specification hierarchy and has a male and female specific isoform. The hypothesis for this part of work is that Abd-B and Dsx play a role in sex specific proliferation and apoptosis of these tNBs. Although the role of the sex determining hierarchy and Hox gene Abd-B, in growth and differentiation of *Drosophila* genital discs, is well worked out, little is known about how sex determination hierarchy and Abd-B intersects with cell proliferation and survival behavior of tNBs in the larval VNC. We intend to test the interaction between Abd-B and Dsx in gender specific proliferation and apoptosis of these cells.

Summary of work done until the beginning of this reporting year (upto March 31, 2016)

1. Understanding the molecular function of Hox gene *Abd-A* in larval CNS patterning.

The relevant enhancer for the activation of RHG family of apoptotic genes in NBs lies within 23kb genomic region referred to as *NBRR-Neuroblast Regulatory Region*. The NBRR was divided into 5 overlapping genomic fragments (of 6-10kb). These genomic fragments were made into transgenic lines and were screened for their ability to drive pNB specific expression of lacZ reporter in late third instar larval **(LL3)** brain. The transgenic line analysis narrowed down the search to 3kb overlapping region of two 8kb fragments (NBRRF3 and F4) after analysis of all 5 *enhancer-lacZ* lines of *NBRR*. We generated a

smaller 2kb enhancer-lacZ from this overlapping region and found that it is expressed in pNBs of abdominal and terminal region of larval central nervous system.

We also genetically isolated the apoptotic enhancer by mobilizing a transposon inserted in *NBRR* to generate a smaller deletion (*NBRR-22*). This deletion in transheterozygotic combination with already existing deletion of NBRR gives ectopic pNBs in the abdominal region of CNS at LL3 stage. The finer PCR mapping indicates that 14.5kb region of the *NBRR* encompassing the relevant apoptotic enhancer has been deleted in this case.

The expression of 2kb enhancer in abdominal pNB and presence of ectopic pNBs in 14.5 kb deletion suggests that we have narrowed down the relevant apoptotic enhancer from 23kb NBRR to 2kb region of the genome. Next the putative Hox and Grh binding sites in the 2kb region were tested for respective transcription factor binding *in vitro* by EMSA. We tested closely placed Hox and Grh binding sites and found that both transcription factors bind on DNA, mutant oligo analysis indicated that these bindings were specific.

An indirect way to check for activation of RHG genes by AbdA and Grh in vivo was by checking *NBRRF3-lacZ* reporter expression in abdominal pNBs, in response to Abd-A and Grh downregulation in pNBs by RNA interference. We found that *NBRRF3-lacZ* line was down regulated in surviving abdominal pNBs in response to RNA interference for AbdA and Grh. Conversely the ectopic expression of Abd-A in thoracic pNBs where Abd-A is not normally expressed resulted in ectopic expression of *NBRRF3-lacZ* in thoracic region as well, indicating the responsiveness of enhancer for Abd-A.

Simultaneously a 4kb enhancer of *grainyhead* responsible for its expression in CNS was sub-fragmented to narrow down the relevant enhancer for the expression of *grainyhead* in CNS to 1kb region. Currently this region is being further analysed to identify transcriptional factors that could be regulating *grainyhead* differentially in NBs versus neurons.

2. Role of Hox gene *Deformed (Dfd)* in patterning of embryonic and larval subesophageal ganglia (SEG).

We found that Dfd auto-regulates itself only in Mn segment of embryonic subesophageal ganglia (SEG). Subsequently we tested the role of Hox cofactor Exd in neural autoregulation and Dfd expression in NBs of embryonic SEG by looking at Exd null mutant (*exd^{1}*). *exd^{1}* homozygous mutants showed no significant change in Dfd expression in NBs. This is due to the fact that Exd is known to be maternally contributed. In order to circumvent the problem of maternal contribution of Exd protein, we decided to analyze *hth^{P2}* a strong hypomorph of *hth* gene. Since Hth is a known partner of Exd, and plays an important role in its transport into the cell nuclei, we expected that *hth^{P2}* will mimic a phenotype similar to *exd* complete loss of function. We found a region specific role for *hth* in Dfd expression, wherein Dfd was completely missing in Mx NBs, while the expression in Mn NBs was dramatically down regulated, but low levels of Dfd could still be observed in these cells in *hth^{P2}* mutants. This suggest that Hth is critical for Dfd expression in Mx NBs but is important only for maintenance of the levels of Dfd protein in Mn NBs. We also found that Hth has no role in *Dfd* neural autoregulation in Mn segments.

Our subsequent experiments with homeodomain-less (HD-less) isoform of Hth (referred to as HM-Hth); show that HM-Hth is sufficient for maintaining *Dfd* expression levels in embryonic stages, and suggest that HD of Hth is not necessary for region specific role of Hth in CNS.

Since both Exd and HM-Hth are required only for regulating levels of Dfd expression in mandibular NBs, and neural autoregulation in these cells is independent of their roles, we propose a role for yet to be identified factor(s) in regulating core neural autoregulatory transcriptional loop. Identification of this/these factor(s) and characterization of their role in NBs and differentiated neurons of mandibular region are ongoing.

3. Investigating the role of *Abdominal-B (Abd-B)* and *Double-sex (Dsx)* in terminal CNS patterning.

It has been reported that female specific isoform of Dsx (DsxF) is responsible for the apoptosis of sex-specific tNBs in females while these cells continue dividing in males. The molecular mechanism behind the phenomenon of apoptosis in females and how Dsx^{M} play a role in tNB proliferation in males is not known so far. It also needs to be investigated how sex specific tNBs are different from other 8 NBs in the same region which stop dividing at mid L3 stage of development.

We find that Abd-B, Grh and Dsx express in tNBs in CNS of both male and female larvae. Since Grh is already known to play a role in apoptosis of pNB of abdominal segments,

grh mutants were analyzed, and we found ectopic pNB in the Abd-B region of female larval CNS compared to wild types where no pNBs are reported at the same stage. Interestingly none of these cells were found to be positive for Dsx which is a conclusive marker for tNBs. This suggests that apoptosis of Dsx+ tNBs in females is independent of Grh.

Analysis of *grim* gene mutants (a member of RHG family of apoptotic genes) showed ectopic NBs in Abd-B region of female larval CNS. On counterstaining of *grim* mutants with Dsx antibody and NB marker Dpn we observed that none of the ectopic NBs in female larval brains were Dsx positive. This suggest that *grim* doesn't play a role in tNB apoptosis and ectopic NBs are embryonic in origin, and some other RHG family member(s) play a role in tNB apoptosis.

In order to locate the enhancer for the apoptotic gene activation in tNBs, we analysed a previously reported 53kb genomic deletion (*MM3*). We find that larvae which are homozygous for this deletion show ectopic pNBs in Abd-B region which stained for Dsx antibody and for NB marker Dpn. This suggest that enhancer for tNB apoptosis lies in this 53kb region. Experiments for isolation of the minimal enhancer for tNB apoptosis are ongoing.

Summary of work done until the beginning of this reporting year (1 April, 2016-31 March, 2017)

1. Understanding the molecular basis of Hox gene Abdominal-A (Abd-A) in larval CNS patterning.

Notch signaling pathway is known for its role in helping cell make binary fate decision. It also been reported to play a role in abdominal NB apoptosis through activation of AbdA in these cells. We investigated the details of the role of Notch in abdominal NB apoptosis. We did find that Notch knockdown in the abdominal NB blocks the death and contrary to what has been reported earlier, we could not see a significant and consistent decrease in AbdA levels. This suggests that Notch signaling doesn't activate AbdA in these cells. Furthermore since Grh is known a play a role in apoptosis its expression was checked, and it was found to be unaffected in abdominal NBs. These results suggests that Notch perhaps has a more direct role to play in abdominal NB apoptosis.

Our subsequent analysis with 2kb enhancer narrowed us down to 1kb region of the genome. Potential Hox, Exd and Grh binding sites were identified and analyzed in this region. We identified 13 Grh binding sites conforming to variation of the known Grh binding consensus sequence (WCHGGTT) with AT rich sequences (potential AbdA and Exd binding sites) in 20bp flanking region. We classified 13 Grh binding sites into 2 categories; 7 were standalone individual Grh binding sites, while 6 Grh sites existed as 3 pairs and were in close vicinity (separated by 1 or 2 bps). We also found only one Hox-Exd consensus site (A/TGATNNATNN) in the entire 1kb region. We tested all these motifs by EMSA. We found that 6 out of 7 standalone Grh sites containing motifs show binding to Grh and two paired Grh sites was observed to bind Grh as well. All the motifs were also simultaneously checked for Exd and AbdA binding as well. The lone consensus Hox-Exd binding showed Hox-Exd binding but no Grh binding. Some of the Grh showed a good tetrameric complex formation with Grh, AbdA and Exd and are being analyzed in detail. The in vivo relevance of these sites will be assessed by testing the capacity of reporter expression by mutagenized enhancer.

Considering the importance of Grh in pNBs we are trying to identify *grh* regulators in pNBs. To this end an RNA interference screen is ongoing. In this screen a battery of 465 transcription factors selected based on their spatial and temporal expression pattern in developing CNS are being knocked down in abdominal and thoracic pNBs to identify regulator of *grh* gene by scoring for downregulation of Grh protein expression. Interestingly we could not identify any regulator or *grh* gene but we have been able to identify 23 genes which seems to play a role in abdominal NB apoptosis.

2. Role of Hox gene *Deformed* in patterning of larval subesophageal ganglia.

The subesophageal ganglia (SEG) of larval CNS (which expresses Dfd, Scr and Antennapedia) has been reported to have 36 NBs (18 segmental pairs) in second instar larval (L2) stage. Out of these 36 pNBs, 10 pNBs (5 pairs) are found in Dfd expressing region of SEG (also referred to as Dfd-SEG). Four out of these 10 pNBs undergo Dfd mediated apoptosis as larva progresses from L2

to L3 stage (Figure 2). The molecular mechanism of this Dfd mediated larval NB apoptosis in SEG region is also not characterized.

We tested whether Grh was expressed in pNBs found in Dfd-SEG. We consistently found all 10 pNBs to be Dpn^+/Grh^+ in EL2 stage. In late L3 stage of development, 4 out of 10 pNBs had undergone Dfd mediated apoptosis and only 6 pNB with associated lineages were remaining. In all of the 6 lineages we found that pNBs always expressed Grh. We also found that pNBs in the Dfd-SEG were Grh^+/Dfd^-, while on other hand, the progeny were Grh^-/Dfd^+. Interestingly Hox and Grh code for pNBs (Grh^+/Hox^-) and associated progeny (Grh^-/Hox^+) in a lineage was same in Dfd-SEG as well as in abdominal region of CNS. pNB specific Grh expression also suggests that like in abdominal pNBs Dfd-SEG apoptosis may be dependent on Grh, and is triggered by change in Hox^-/Grh^+ state of pNB to Hox^+/Grh^+ state. This prompted us to test the functional role of Grh in apoptosis of 4 pNBs in Dfd-SEG during development. These experiments are ongoing.

3. Investigating the role of *Abdominal-B (Abd-B)* and *Double-sex (Dsx)* in terminal CNS patterning.

Our results with mutant for apoptotic gene *grim* suggested that it was alone not responsible for Dsx+ tNB apoptosis in females. Hence we tested *reaper* (*rpr*) mutants and found that *rpr* alone was also not sufficient for Dsx+ tNB apoptosis. Since abdominal pNBs require both *grim* and *rpr* for their apoptosis, we checked *grim-rpr* double mutants and found many surviving NBs in females larval VNC. Four of these NBs expressed Dsx. This suggested that Dsx+ tNB apoptosis in females required both *grim* and *reaper* genes. Since 53kb genomic deletion had showed us Dsx+ NBs in AbdB expressing regions, we further tested the 14.5kb deletion genomic deletion in trans-heterozygotic combination with 53Kb deletion. Here as well we found ectopic NBs in AbdB expressing region of the female brain and four of these expressed Dsx. This suggests that enhancer for the Dsx+ tNB apoptosis lies within 14.5kb region of the genome like in case of abdominal NBs.

We interestingly also found that lacZ reporter lines (both 8kb *NBRRF3-lacZ* and *F4-lacZ* and *1kb-lacZ*) didn't express in Dsx+ tNB in males but express only in female Dsx+ tNB which are destined to undergo apoptosis. This suggested to us that the enhancer for the apoptosis of Dsx+ tNB is female specific and lies within 1 kb genomic region of the NBRR and is sex-specific in its expression.

Further analysis of the 2kb region in ongoing.

Simultaneously we are also testing the role of *Drosophila* cell cycle genes like *Cyclin, A, B, E* and E2F for their specific roles in continued sex specific proliferation of Dsx+ tNBs proliferation in male larval CNS.

LABORATORY OF FUNGAL PATHOGENESIS

Understanding the pathobiology of an opportunistic human fungal pathogen *Candida glabrata*

Faculty	Rupinder Kaur	Staff Scientist& Wellcome Trust -DBT India Alliance Senior Fellow
PhD Students	Vivek Kumar Srivastava	Senior Research Fellow (till June 2016)
	Vandana Sharma	Senior Research Fellow
	Mubashshir Rasheed	Senior Research Fellow
	Priyanka Bhakt	Senior Research Fellow
	Kundan Kumar	Junior Research Fellow
	Anamika Battu	Junior Research Fellow
	Fizza Askari	Junior Research Fellow (since July 2016)
	Mahima Sagar Sahu	Junior Research Fellow (since Feb. 2017)
Other Members	S Surya Vamshi	Technical Officer
	Reshma Chowdary Alokam	Research Associate
	T.S. Jalajaveronica Rani	Research Associate (since Dec. 2016)
	Rajaram Purushotham	Project JRF
	Deepak Kumar Choudhary	Project JRF
	Guru Govind Vaidu	Project JRF (During April to December 2016)
	Romilla Moirangthem	Project JRF (since May 2016)
Collaborators	Rajendra Prasad	JNU, New Delhi
	Naseem A Gaur	ICGEB, New Delhi

Candida species account for 70 to 80% of bloodstream fungal infections with *Candida glabrata* being the second most frequently isolated Candida species after *C. albicans*. Despite being a successful pathogen, *C. glabrata* lacks some of the key fungal virulence traits, and appears to rely primarily on alternative mechanisms to survive the nutrient-poor, hostile environment of the human host. Research in our laboratory is aimed at a better understanding of pathogenesis and antifungal drug resistance mechanisms of *C. glabrata*.

Project 1: Functional genomic analysis of *C. glabrata*-macrophage interaction

Objectives

1. Screening of a *C. glabrata* mutant library for altered survival profiles
2. Identification and analysis of genes required for survival *in vitro* and *in vivo*

Summary of the work done until the beginning of this reporting year

Using an *in vitro* system comprised of human monocytic cell line THP-1, we demonstrated that wild-type *C. glabrata* cells are able to impede phagolysosome acidification, survive the reactive oxygen species generated and replicate in THP-1 macrophages. We further screened a Tn*7* insertion mutant library, representing 50% of the *C. glabrata* genome, for altered survival in macrophages, and identified 53 novel genes required for intracellular survival and/or proliferation. These genes were implicated in diverse biological processes including chromatin and cell wall organization, signal transduction and Golgi vesicle transport. One of identified genes, CgVps15, codes for the regulatory subunit of the class III phosphoinositide 3-kinase (PI3K). By generation and characterization of deletion strains, *Cgvps15*Δ and *Cgvps34*Δ, which lack PI3K regulatory and catalytic subunits, respectively, we showed that CgVps15 and CgVps34 are essential for intracellular survival, vacuolar protein sorting, autophagy and virulence in *C. glabrata*. We also showed that CgVps34 catalyzes the conversion of phosphatidylinositol to phosphatidylinositol-3-phosphate.

Details of the progress made in the current reporting year (April 1, 2016 - March 31, 2017)

To examine the multiple stress-sensitive phenotype of the *Cgvps34*Δ mutant more closely,

we performed global transcriptome profiling of YPD-grown logarithmic (log)-phase wild-type (*wt*) and *Cgvps34*Δ cells using RNA Sequencing (RNA-seq) analysis.*CgVPS34* disruption led to differential regulation of 160 genes (≥1.5 fold change and a FDR-adjusted *p*-value of ≤ 0.05). Of these genes, 96 were up regulated and 64 were down regulated in the *Cgvps34*Δmutant. Gene Ontology (GO)-Slim Mapper analysis, using the Candida Genome Database (CGD; http://www.candidagenome.org), revealed genes involved in biological processes of "transport" and "response to stress" to be differentially expressed in the *Cgvps34*Δ mutant. Specifically, GO categories, iron ion transmembrane transport and cellular response to zinc ion starvation, were found to be significantly enriched in the down regulated gene list using the FungiFun2 analysis tool. A set of 13 iron homeostasis genes including genes encoding proteins involved in high-affinity iron uptake (CgFet3, a multi copper oxidase) and low-affinity ion transport (CgFet4, a low-affinity ion transporter) were differentially regulated with iron transport genes exhibiting down regulation while iron utilization/iron-sulfur (Fe-S) cluster-binding genes showing upregulation in the *Cgvps34*Δ mutant. We verified the RNA-Seq gene expression data by qPCR analysis and observed good correlation between these two analyses.

Consistent with the transcriptional profiling data, the *Cgvps34Δ* mutant,compared to *wt* cells, contained approximately 3.0-fold higher intracellular iron levels (Figure 1A) and 1.6-fold higher activity of the Fe-S cluster-containing mitochondrial aconitase enzyme (Figure1B) which were restored back to normal levels in the complemented-mutant strain (Figure1 A, B).These results are indicative of a significantly perturbed iron metabolism upon *CgVPS34* disruption, and raise the possibility that the higher iron content may lead to elevated Fe-S cluster generation, thereby, acting as a signal for transcriptional downregulation of the iron uptake machinery in the *Cgvps34Δ* mutant.

Next, due to elevated intracellular iron content, we hypothesized that the growth of the *Cgvps34Δ* mutant will be impaired in the high-iron environment. To test this, we checked the susceptibility of the *Cgvps34Δ* mutant to surplus iron as well as iron-limitation. Intriguingly, the *Cgvps34Δ* mutant was sensitive to both iron-replete (caused by $FeCl_3$ addition) and iron-deplete [caused by BPS (extracellular iron chelator) addition]conditions (Figure1C) which may imply that mutant cells are deficient in responding to variations in the environmental iron concentration. However, *Cgvps34*Δmutant cells, like *wt* cells, were able to up regulate and down regulate expression of the high affinity iron transport system in response to iron-limited and iron-excess conditions, respectively (Figure1D).

To address the question of why *Cgvps34Δ* cells, despite mounting an appropriate transcriptional response, could not grow in low- and high-iron medium, we sought to examine functioning of the iron transport machinery in the *Cgvps34Δ* mutant. The high-affinity iron uptake system in *C. glabrata* is composed of an iron permease (CgFtr1) and a copper ferroxidase (CgFet3) which are assumed to form a complex. The Ftr1 permease and Fet3 ferroxidase in *S. cerevisiae* are co-trafficked to and from the cell membrane. We first generated CgFtr1-GFP and CgFet3-GFP fusion proteins by inserting GFP (Green fluorescent protein) at the C-terminus of CgFtr1 and CgFet3 and confirmed their functionality followed by examination of their localization in *wt* cells. Under regular-iron log phase conditions, we found CgFtr1 to localize to both the plasma membrane and the vacuole, while CgFet3-GFP was primarily located on the plasma membrane and the membrane of an intracellular organelle in *wt* cells. Further, in response to iron limitation, CgFtr1-GFP did not localize to the vacuole as cellular fluorescence was limited only to the plasma membrane in both *wt* and *Cgvps34Δ* cells (Figure1E). Contrarily, the vacuolar localization and the cell membrane localization of CgFtr1-GFP was enhanced and diminished, respectively, in *wt* cells upon growth in the iron-surplus medium (Figure1E). Of note, localization of CgFtr1-GFP in the *Cgvps34Δ* mutant did not change in response to iron-replete conditions, and remained primarily confined to the plasma membrane in vast majority (95%) of cells (Figure1E). These data indicate that *Cgvps34Δ* cells are deficient in the retrograde transport of CgFtr1-GFP from the plasma membrane in the high-iron environment, which could partly account for elevated susceptibility of the *Cgvps34Δ* mutant to the surplus iron.

Similar to CgFtr1-GFP, approximately 90% of *wt* and *Cgvps34Δ* cells exhibited plasma membrane localization of CgFet3-GFP under iron-limiting conditions (Figure1F). Further, in the iron-excess medium, plasma membrane targeting of CgFet3-GFP was drastically reduced as

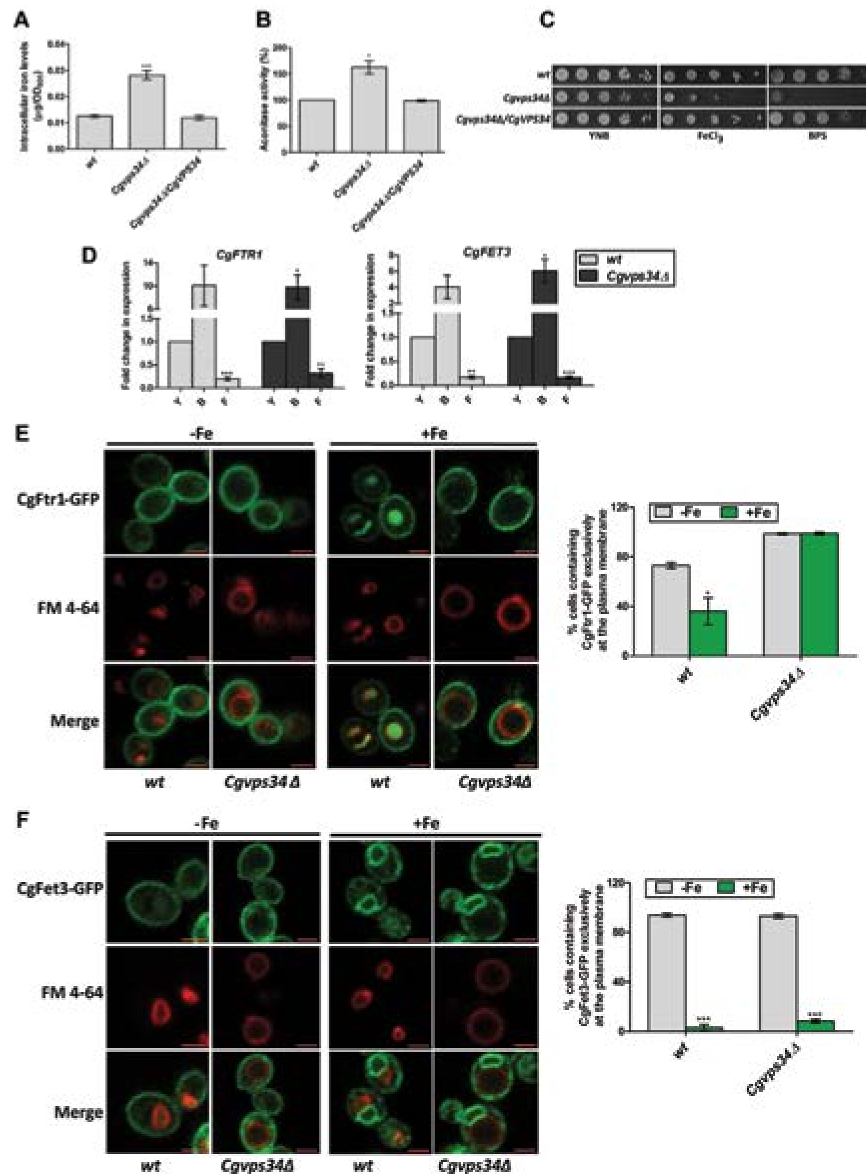

Figure 1. *CgVPS34* disruption results in perturbed iron homeostasis. (A) Intracellular iron levels of indicated, YPD medium-grown, log-phase *C. glabrata* cells, as determined by inductively coupled plasma-atomic emission spectrometry (ICP-AES). Data (mean ± SEM, n = 6) are presented as iron (μg) present in cells equivalent to one OD_{600}. Unpaired, two-tailed, Student's t test (***, p<0.0001). **(B)** Mitochondrial aconitase activity, as measured by the reduced nicotinamide adenine dinucleotide-coupled assay, in crude mitochondrial extracts of indicated YPD medium-grown, log-phase *C. glabrata* cells. Data represent mean ± SEM (n = 3). *, p<0.05; paired two-tailed Student's t-test. **(C)** Serial dilution spotting growth analysis of indicated *C. glabrata* strains in the YNB medium lacking or containing ferric chloride (3.5 mM) and BPS (100 μM). **(D)** qPCR analysis of *CgFTR1* and *CgFET3* gene expression in *wt* and *Cgvps34*Δ mutant upon 2 h growth in the YNB medium (Y) lacking or containing 50 μM BPS (B) or 500 μM ferric chloride (F). Data (mean ± SEM, n = 3-6) were normalized to an internal *CgACT1* mRNA control, and represent fold change in expression upon BPS and ferric chloride treatment compared to YNB-grown cultures. Paired, two-tailed, Student's t-test (*, p<0.05; **, p<0.005; ***, p<0.0001). **(E & F)** Overnight CAA medium-grown, *wt* and *Cgvps34*Δ cells expressing CgFtr1-GFP **(E)** and CgFet3-GFP **(F)** were inoculated in the CAA medium containing 50 μM BPS. After 12 h incubation at 30ºC, cells were collected, washed with CAA and inoculated to the CAA medium containing either 50 μM BPS (-Fe) or 500 μM ferrous ammonium sulfate and 1 mM sodium ascorbate (+Fe). Post 2 h growth at 30ºC, cells were imaged using the Zeiss LSM 700 META confocal microscope. Scale Bar = 2 μm. For each strain, a minimum of 160 cells displaying green fluorescence were counted, and data are presented as percentage of cells with CgFtr1-GFP **(E)** and CgFet3-GFP **(F)** at the plasma membrane on the right side of panels. Unpaired, two-tailed, Student's t-test (*, p<0.05; ***, p<0.0001). A.U., arbitrary units.

only 1-5% of *wt* and *Cgvps34Δ* cells contained CgFet3-GFP exclusively on the cell membrane (Figure1F). CgFet3-GFP was primarily confined to the intracellular organelle membrane in *wt* and *Cgvps34Δ* cells upon growth in the iron-rich medium (Figure1F), thereby, precluding involvement of CgVps34 in the retrograde transport of CgFet3-GFP.

To summarize these results, environmental iron content determines the recycling of CgFtr1 and CgFet3 from the cell membrane with high iron resulting in relocation to intracellular organelles, thereby, setting the stage for either recycling or degradation of CgFtr1 and CgFet3 proteins. Second, CgVps34 is dispensable for trafficking of CgFtr1 and CgFet3 to the cell membrane. Third, retrograde transport of CgFtr1 and CgFet3 probably occur independently of each other. Lastly, transport of CgFtr1 to the vacuole in response to surplus iron requires PI3-kinase.

Project 2: Characterization of glycosylphosphatidylinositol-linked aspartyl proteases in *Candida glabrata*: role in pathogenicity

Objectives

1. Molecular and biochemical characterization of *C. glabrata* yapsins
2. Identification and characterization of physiological substrates of *C. glabrata* yapsins

This is a new activity.

Details of the progress made in the current reporting year (April 1, 2016 - March 31, 2017)

Among known virulence factors of *C. glabrata*, a family of eleven putative glycosylphosphatidylinositol (GPI)-linked, cell surface-associated aspartyl proteases occupies a central position. These proteases, also referred as yapsins, are encoded by *CgYPS1-11* genes. Of these, eight *CgYPS* genes (*CgYPS3-6, 8-11*) are encoded in a unique cluster on chromosome E, and are referred to as '*CgYPS-C*'. Previous work from our laboratory has demonstrated the pivotality of *C. glabrata* yapsins to several pathobiological processes including pH and vacuole homeostasis, intracellular survival and virulence. A major goal of the current study is to delineate cellular processes regulated by the proteolytic activity of CgYapsins, and examine their centrality to Candida virulence.

Towards our goal, we have generated *C. glabrata* strains deleted for *CgYPS-C* (*CgYPS3*, *CgYPS4*, *CgYPS5*, *CgYPS6*, *CgYPS8*, *CgYPS9*, *CgYPS10* and *CgYPS11*) genes individually *via* a homologous recombination-based strategy using a cassette containing the *nat1* gene (imparts resistance to nourseothricin). We now have a panel of single deletion strains for all eleven *CgYPS* genes, as we already had single deletion mutants for three yapsin-encoding genes, *CgYPS1*, *CgYPS2* and *CgYPS7*. We also havemutants *Cgyps1Δyps7Δ,* *Cgyps2ΔypsCΔ* and *Cgyps1-11Δ*, that lacked two, nine and eleven aspartyl proteases, respectively. Growth profiling of generated strains under in vivo conditions is currently underway.

To delineate the role of yapsins in maintenance of the cell wall architecture in *C. glabrata*, we measured the cell wall chitin content of wild-type and *CgypsΔ* mutants via calcofluor white (CFW, a chitin-binding agent) staining-based flow cytometry assay. As shown in Figure 2A,*Cgyps1Δ*, *Cgyps7Δ*, *Cgyps1Δyps7Δ* and *Cgyps1-11Δ* mutants displayed 2.0- to 3.5-fold higher chitin levels compared to wild-type cells. In contrast, chitin levels were found to be similar between wild-type and the *Cgyps2ΔypsCΔ* mutant (Figure2A). These results point towards a role for CgYps1 and CgYps7 proteases in cell wall homeostasis. Currently, we are trying to create catalytically dead and GPI anchor-lacking versions of CgYps1 and CgYps7 enzymes to study the role of protease activity and localization in cell wall remodeling.

Next, to examine if altered cell wall composition of *Cgyps1Δ*, *Cgyps7Δ*, *Cgyps1Δyps7Δ* and *Cgyps1-11Δ* mutants affects the interaction of mutants with the abiotic surface, we assessed the ability of wild-type and mutants cells to form biofilm on polystyrene-coated plates. Of note, these *CgypsΔ* mutants are known to exhibit elevated adherence to Lec2 Chinese Hamster Ovary cells. Surprisingly, compared to wild-type cells, *Cgyps1Δ*, *Cgyps7Δ*, *Cgyps1Δyps7Δ* and *Cgyps1-11Δ* mutants displayed 2-6-fold lower biofilm-forming capacity (Figure2B). The *Cgyps2ΔypsCΔ* mutant produced biofilms similar to wild-type cells (Figure2B). These data indicate that despite increased expression of the Epa1 adhesin at the cell surface and increased adherence potential for Lec2 cells, *Cgyps1Δ*, *Cgyps7Δ*, *Cgyps1Δyps7Δ* and *Cgyps1-11Δ* mutants are impaired in their ability to form biofilms. Experiments are currently underway to elucidate whether the biofilm formation defect is due to diminished adherence or reduced growth rate of *CypsΔ* mutants.

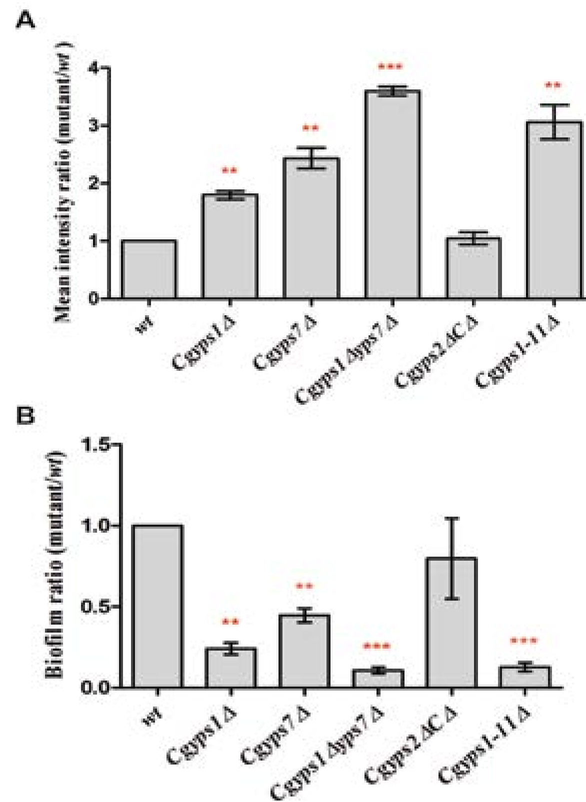

Figure 2. Cell wall analysis of *Cgyps*Δ mutants (A)Cell wall chitin measurement. Indicated *C. glabrata* strains were grown to logarithmic phase in the rich YPD medium and stained with 25 µg/ml calcofluor white (CFW) for 15 min at room temperature in the dark. Fluorescence intensity was measured by flow cytometry, and mean fluorescence intensity ratio was calculated by dividing the fluorescence intensity value of the mutant sample with that of the wild-type sample. **(B)** Biofilm formation assay. Indicated *C. glabrata* strains were grown in the RPMI 1640 medium containing 10% FBS for 48 h in a polystyrene 24-well plate. Cells were stained with 0.4% crystal violet for 45 min followed by destaining with 95% ethanol. Absorbance at 595 nm was recorded to measure the amount of the crystal violet stain in ethanol. Data represent mean ± S.E. of three independent experiments. Paired, two-tailed, Student's t test (**, p≤0.01; ***, p≤0.001). Statistically significant differences between wild-type and *Cgyps*Δ mutants are indicated.

Publications

Research papers published in the calendar year 2016

1. Khandelwal, NK, Kaemmer, P, Förster, TM, Singh, A, Coste, AT, Andes, DR, Hube, B, Sanglard, D, Chauhan, N, **Kaur, R,** d'Enfert, C, Mondal, AK and Prasad R. (2016) Pleiotropic effects of the vacuolar ABC transporter *MLT1* of *Candida albicans* on cell function and virulence. *Biochemical Journal* **473:**1537-52.

2. Gujjula, R[#], Veeraiah, S[#], Kumar, K, Thakur, SS, Mishra, K* and **Kaur, R.*** (2016) Identification of components of the SUMOylation machinery in Candida glabrata: Role of the deSUMOylation peptidase CgUlp2 in virulence. *Journal of Biological Chemistry* **291:**19573-89. [#]Equal contribution; *Corresponding authors.

3. Sharma, V, Purushotham, R and **Kaur, R** (2016) The phosphoinositide 3-kinase regulates retrograde trafficking of the iron permease CgFtr1 and iron homeostasis in *Candida glabrata*. *Journal of Biological Chemistry* **291:**24715-34.

Laboratory of Genomics and Profiling Applications

Faculty	Madhusudan Reddy Nandineni	Staff Scientist
PhD Students	Anujit Sarkar	Senior Research Fellow
	Soumya Rao	Senior Research Fellow
	Mugdha Singh	Senior Research Fellow
	Saphy	Junior Research Fellow (since March 2017)
Other Members	Vineesha Oddi	Project-JRF (till Dec. 2016)

Objectives:

1. Human genetic diversity studies among various population groups in India
2. Dissection of plant-fungal interactions in the chilli-*Colletotrichum*pathosystem

Project 1: Human genetic diversity studies among various population groups in India.

Summary of work done until the beginning of this reporting year (upto March 31, 2016)

With the aim of designing an single nucleotide polymorphism (SNP)-based panel for human identification (HID) in Indian populations, 384 SNPs were shortlisted from the publicly available SNP databases employing a stringent filtration procedure described previously. 206 SNPs which followed the Hardy-Weinberg equilibrium (HWE) and possessing high heterozygosity (Het ≥0.4), low Wright's F-statistics (F_{st} ≤0.02) and allele distribution required for HID purposes were further tested. 2-4 SNPs located >20Mb apart on each chromosome were selected to form a final panel of 70 SNPs. Post genotyping of these SNPs in ~400 individuals sampled from different geographical regions, the relevant forensic parameters were calculated using DNAView™ software. As mentioned in the previous report, the shortlisted 70-plex SNP panel demonstrated very high forensic parameters that are required for making unambiguous inferences in forensic casework analysis.

In another aspect of work, the expanded panel of autosomal short tandem repeats (STRs) present in PowerPlex® Fusion chemistry (PP Fusion) (Promega, Madison, WI, USA) were also evaluated for their forensic efficiency and performance in Indian populations. These loci were found to be highly polymorphic with an average informative index of 1.77 and demonstrated high forensic performance. Clustering analysis based on these STR loci revealed absence of any sub-structuring in Indian populations implying that there was no significant genetic distance among these populations.

Details of progress made in the current reporting year (April 1, 2016- March 31, 2017):

a) Association of genetic variants with human skin colour in Indian populations:

Skin colour variation is one of the most conspicuously visible attributes in humans. Considered as a polygenic quantitative trait, the skin colour varies widely, both within and between populations, all over the world. Among the environmental factors, the intensity of ultra violet radiation (UVR) at a given location strongly correlates with the phenotype, i.e., at regions with high UVR intensity, people tend to have darker skin colour,suggesting the role of adaptive evolution against UVR radiation. More than 100 genes were reported to affect skin pigmentation in mouse, with half of their homologues being identified in humans. Several genetic variants (particularly SNPs) in various world populations were reported to be strongly associated with the human skin colour. The role of genetic variants towards skin pigmentation was carried out in greater depth in the European populations, however, such studies for Indian populations have been relatively sparse.

In this context, as part of understanding the human genetic variation, the present study was aimed at determining the allelic distribution of SNPs,which were previously reported to be associated with the pigmentation phenotype in worldwide populations, and to test their association with the phenotype in Indian populations. Approx. 300 adult unrelated volunteers (232 males and 67 females) sampled from nine different sampling locations (States) from four geographic regions; viz., North India (N = 87), West India (N = 77), East India (N = 57) and South India

(N = 78), respectively, were genotyped for 30 SNPs which were reported to be associated with pigmentation phenotype. In order to quantitatively measure the skin colour, melanin index from the inner arm of each volunteer was measured using the using DSM Colorimeter II (Cortex Technology, Hadsund, Denmark). The shortlisted SNPs were genotyped using the Golden Gate® assay on Bead Xpress®(Illumina, Inc. USA) according to the manufacturer's instructions. From the genotype data, the allele frequency for each locus was calculated using the gene count method. The SNPs which were monomorphic or possessed very low minor allele frequency (MAF <0.05) were not included in the association analyses. Similarly, SNPs for which the percentage of missing data was high (>5%) were also excluded. Currently, the association of the SNPs with the melanin index, if any, is being analyzed to investigate the strength of association of each SNP and their corresponding effect on the skin colour phenotype in Indian populations. These studies are expected not only to help in validating the previously reported genotype-phenotype correlations but also would aid in better understanding the molecular mechanisms of the skin pigmentation phenotype in humans.

b) Human genetic variations studies in Indian populations based on expanded Y-chromosomal STRs:

To study the genetic relationship among the various sub-populations from different geographic regions and to evaluate the applicability of the expanded Y-STR loci in Power Plex® Y23 chemistry (Promega, Madison, WI, USA) for DNA profiling casework analysis in Indian populations, 346 individuals residing in 11 different regions of India were genotyped and the forensic efficacy of the panel was evaluated. A total of 341 unique haplotypes were obtained employing the above chemistry. The discrimination capacity (DC; DC = number of unique haplotypes/ total number of haplotypes) of 0.9855491329 was comparable with the values obtained with other worldwide populations. The decent value of match probability (MP) and haplotype diversity (HD) (0.003044349 and 0.999845377, respectively)showed the applicability of the tested Y-STR loci for forensic case work analysis in these populations as well. Locus wise analysis performed with GenALEx v6.5, showed that the loci DYS570 (0.837) and DYS391 (0.416) exhibited the highest and the lowest gene diversity (GD) values, respectively.

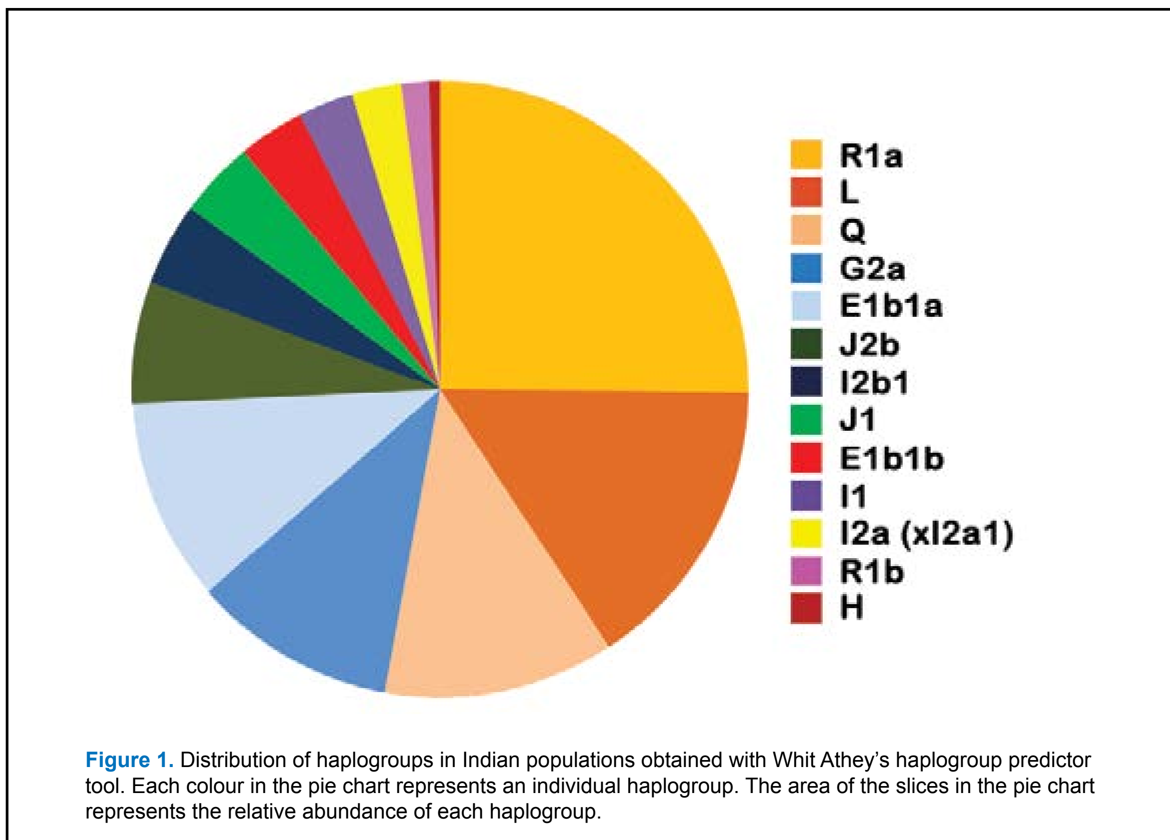

Figure 1. Distribution of haplogroups in Indian populations obtained with Whit Athey's haplogroup predictor tool. Each colour in the pie chart represents an individual haplogroup. The area of the slices in the pie chart represents the relative abundance of each haplogroup.

A total of 13 Y-STR based haplogroups were obtained for 346 male individuals employing Whit Athey's haplogroup predictor (Figure 1).

As can be seen in the pie chart in Figure 1; R1a was found to be the most abundant haplogroup in these populations followed by L, Q, G2a and E1b1a,whereas other haplogroups were present in less than 10% of abundance. Many studies in past had attested the fact that R1a is in fact the most prevalent haplogroup across Eurasia. Population specific analyses of the haplogroups are underway and expectedto provide useful insights to study correlation, if any, between geography and haplogroup abundance.

Project 2: Plant-fungal interaction studies in the Chilli - *Colleotrichum* pathosystem

Summary of work done until the beginning of this reporting year (up to March 31, 2016):

Colletotrichum truncatum (formerly called as *C. capsici*) is the most predominant species in India causing chilli anthracnose leading to both pre- and post-harvest losses. With the availabilityof whole genome sequence of chilli and six *Colletotrichum* speciesin public domain, the chilli - *C. truncatum* pathosystem offers an excellent system for studies on the infection process and molecular interactions between the host and fungal pathogen. The present study aims to identify and characterize pathogenicity genes in *C. truncatum* to get an insight into different aspects of its biology, life-style and host specificity through whole genome sequencing of *C. truncatum* and random insertional mutagenesis.

We had previously reported the *de novo* whole genome sequencing of *C. truncatum* employing Illumina HiSeq platform.The sequence assembly consisted of 81 scaffolds with a total length of 55.3 Mb. Phylogenetic analysis placed *C. truncatum* close to *C. gloeosporioides* and *C. orbiculare*, which helped in carrying out comparative genomics studies in later stages. The draft genome assembly of *C. truncatum* was assessed to be 100% completeby Core Eukaryotic Genes Mapping Approach (CEGMA) and TBLASTN based on coverage of orthologs of all 458 core eukaryotic genes (CEGs). The preliminary annotation was carried out using MAKER based on *ab initio* predictions and homology with the proteomes of *Colletotrichum* species.

Details of progress made in the current reporting year (April 1, 2016- March 31, 2017):

a) Whole genome *de novo* sequence analysis

In order to obtain evidence for the identification of transcribed genes and annotation based on accurate exon structure, RNA-sequencing (RNA-Seq) analysis was carried out for three *in vitro* and three *in planta* samples of *C. truncatum*. The raw reads from each of the samples were cleaned up to remove adapters, low quality sequences, rRNA contamination and PCR duplicates from each library. The *in vitro* reads were mapped to the *C. truncatum* genome that was previously sequenced in our laboratory and the mapped reads (~89%) were used for genome-guided assembly. The pre-processed reads from *in planta* samples were mapped to both the published chilli genomes. Around 80% of the reads mapped to *C. annuumcv*. CM334, while ~88% mapped to *C. annuumcv*. Zunla. The unmapped reads were retrieved and mapped to *C. truncatum* genome. All *in vitro* reads and the *in planta* reads which mapped to *C. truncatum* genome were used for *de novo* transcript assembly, which along with genome-guided assembly, formed a transcriptome with 93,000 contigs. Itenabled in predicting the protein coding ORFs used to train *ab initio* gene prediction tools, viz; SNAP and AUGUSTUS. 13,724 consensus gene models were predicted by combining RNA-Seq evidence with homologues from other *Colletotrichum* species as well as SwissProt database and predictions from different *ab initio* tools. ~77% of the predicted genes had homologues in SwissProt database and/or a known protein family domain.

Secretome is the most important category of genes in the pathogenic fungi, which includes genes encoding secreted proteins that play a role at the host-pathogen interphase to establish a successful infection. The secreted proteins were predicted by using a battery of tools based on the presence of signal peptide and, absence of transmembrane domains, GPI anchors and ER retention signals (Figure 2).

This stringent pipeline of tools returned 1,257 proteins that were highly likely to be secreted. The *C. truncatum* secretome was rich in FAD-domain containing oxidoreductases, subtilisin-like serineproteases, carbohydrate metabolizing enzymes, carbohydrate binding modules, effector-like proteins etc. A total of 59 of these

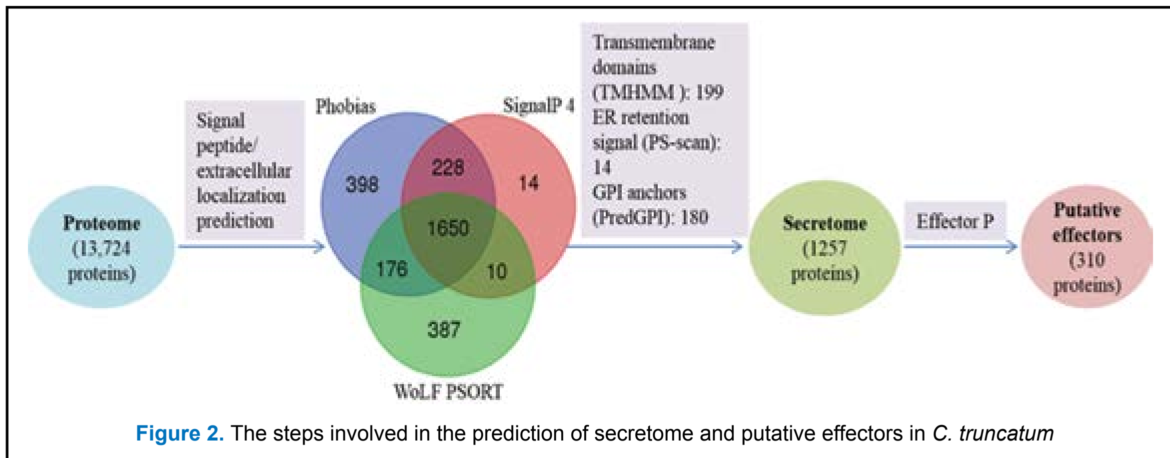

Figure 2. The steps involved in the prediction of secretome and putative effectors in *C. truncatum*

secreted proteins were predicted to contain the nuclear localization signals which may modulate the host cellular dynamics by localizing to the host nuclei and controlling the expression of genes involved in defence responses.

Approx. 310 effectors were predicted through a bioinformatics tool, EffectorP. The effectors are typically small, secreted, cysteine-rich proteins that suppress plant defense responses and modulate the plant physiology to facilitate the host colonization during pathogen attack. In *C. truncatum* secretome, 125 cysteine-rich proteins (a minimum of 3 cysteine residues and at least 3% cysteine-content) that were <300 amino acid long (Figure 3) were considered as putative effectors, of which 109 were in common with EffectorP predicted proteins. The candidate effectors for further studies were selected based on the absence of homology to any known proteins in Swiss Prot database or any known domains. The proteome and secretome would be mined in future for other gene categories relevant for pathogenicity, like cell wall degrading enzyme (carbohydrate active enzymes and proteases) and secondary metabolism associated genes.

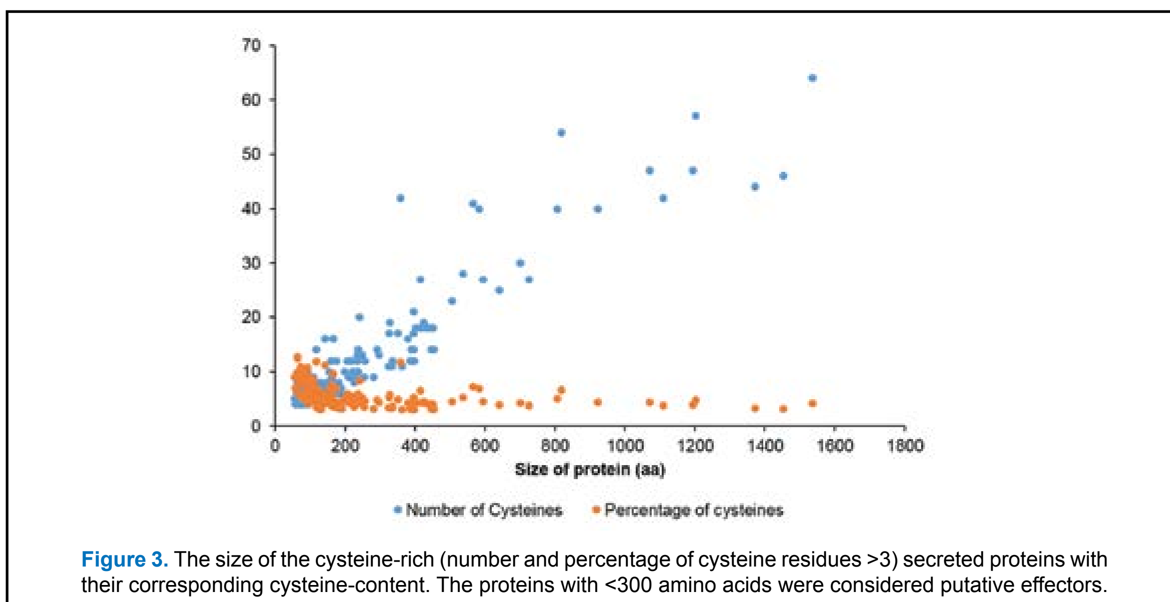

Figure 3. The size of the cysteine-rich (number and percentage of cysteine residues >3) secreted proteins with their corresponding cysteine-content. The proteins with <300 amino acids were considered putative effectors.

Publications:

1. Sarkar A and Nandineni MR(2017). Development of a SNP-based panel for human identification for Indian populations. ***Forensic Science International: Genetics*** *27* , 58-66.

2. Singh M and Nandineni MR(2017). Population genetic analyses and evaluation of 22 autosomal STRs in Indian populations. ***International Journal of Legal Medicine****131*, 971-973.

LABORATORY OF IMMUNOLOGY

Understanding the melanoma tumorigenesis and its regulation

Faculty	Sunil K Manna	Staff Scientist
PhD Students	Adeel H Zaidi	Senior Research Fellow (till Dec. 2016)
	Neeharika Verma	Senior Research Fellow (till Dec. 2016)
	Raveendra Babu M	Senior Research Fellow
	Pankaj Gupta	Senior Research Fellow
	Shashank Saurav	Junior Research Fellow
	Aher Abhishek Taterao	Junior Research Fellow
Other Members	T Navaneetha	Technical Assistant
Collaborators	Biswadev Bishayi	Calcutta University, Kolkata
	Tushar Basu Baul	NEHU, Shilong

Objectives

1. Understanding the mechanism of melanoma tumorigenesis and its regulation for better therapeutics
2. Understanding and regulation of advanced glycation endproducts (AGE)-mediated lipogenesis and autophagy.
3. Understanding and regulation of inflammatory and tumorigenic responses.
4. Understanding the role of Profilin in regulation of tumorigenesis.

Summary of work done until the beginning of this reporting year (April 1, 2015 - March 31, 2016)

Advanced glycation end products (AGE) accumulate in diabetic patients and aging people due to high amounts of 3- or 4-carbon sugars derived from glucose and thereby causing multiple consequences including inflammation, apoptosis, obesity and age-related disorders. It is important to understand the mechanism of AGE-mediated signaling leading to activation of autophagy (self-eating) that might negatively assist in developing obesity and its consequences. We have detected AGE as one of the potent inducers of autophagy compared to doxorubicin and TNF. AGE-mediated autophagy is inhibited by suppression of PI3 kinase (upon wortmanin treatment) and potentiated by autophagosome maturation blocker, bafilomycin. AGE-mediated autophagy is suppressed partially by inhibitor of NF-κB, ERK, or PKC alone and significantly in combination. Subsequently, *IκBα-DN* (IκBα dominant negative) transfected cells, even when stimulated by AGE showed reduction in autophagy markers suggesting the important role of NF-κB in AGE-mediated autophagy.

AGE stimulation increases both lipogenesis as determined by Oil Red O stained cells and autophagy as determined by MDC stained cells in time dependent manner. To validate the probable role of autophagy in lipogenesis, Oil Red O staining is again done in presence of autophagy inhibitors and mangiferin which shows dramatic drop in lipid droplets. AGE increases SREBP DNA binding kinetically. AGE-mediated lipid accumulation is inhibited to almost 50% by PKC I or SB and PD. BAY (NF-κB inhibitor) or SR (Raf Kinase inhibitor) inhibited almost 80% of lipid accumulation in AGE-stimulated cells. Inhibiting autophagy upon Atg7 and Atg12 shRNA transfection and subsequent stimulation with AGE resulted in the increase in accumulation of lipid droplets in cells. Almost complete inhibition of lipid accumulation was observed in AGE-stimulated cells pretreated with novastatin (inhibitor of HMG CoA pathway) or SR and BAY. These data suggest that NF-κB and Raf kinase pathways are involved in AGE-mediated lipid accumulation. We have detected the AGE and glucose mediated autophagy and lipogenesis follow different pathways and AGE-mediated autophagy machinery initiates prior to lipogenesis which probably helps cells with supply of energy and other building blocks to assist lipogenesis and hence shifts the balance from lipolysis to lipid accumulation.

Details of progress in the current reporting year (April 1, 2016 - March 31, 2017)

1) MITF inhibition is the main cause of resveratrol mediated cell death but not NF-κB.

Resveratrol (3,5,4' trihydroxystilbene) is polyphenolic compound, which is natural component of grapes, peanuts, berries and especially red wine. It is known as an anti-oxidant and for its cardio-protective functions. Recent researches were focused on its anti-cancerous properties. Here we investigated mechanism of its anti-melanoma activity. Resveratrol significantly activated cell death in A375 melanoma cells, compared to other natural and synthetic compounds like azadiachtin and thiadiazolidine derivative (P_3-25), respectively. But its effectiveness at 72 hours was less than therapeutic drug vemurafenib, which is a specific inhibitor of ^{V600E}B-Raf (Figure 1A). Resveratrol induces more cell death in melanoma, compared with PC3, HT29 and MDA MB-231 (Figure 1B). This suggests resveratrol is potent melanoma inhibitor than other types of cancers. The mechanism of cell death was further confirmed as apoptosis. Interestingly, resveratrol is more effective than vemurafenib at 24 hours (Figure 1C). We further wanted to study melanoma specific mechanism of resveratrol. MITF is the most important transcription factor for melanoma survival, proliferation and differentiation. Resveratrol inhibited melanoma DNA binding activity (Figure 1D). It can be due to its inhibition of MITF's activation or due to downregulation of its levels. Decreased protein levels upon treating with resveratrol suggest the latter mechanism (Figure 1E). Overexpressed MITF inhibited resveratrol mediated cell death, which further strengthened this view (Figure 1F). Previous literature analysis shows that resveratrol can inhibit cancer cell proliferation by inhibiting NF-κB. We did further experiments to understand its role in this mechanism and used its specific inhibitor BAY 11-7082. Resveratrol inhibited both the transcription factors, whereas BAY 11-7082 inhibited only partially. As expected, MITF is not present in a non-melanoma cell line MDA MB-231 (Figure 1G). This suggests that inhibition of NF-κB is the reason for general cancer cell death, but MITF inhibition must be the main reason or giving additive effect for melanoma specific cell death. Knock down of RelA, did not induce PARP cleavage and did not enhance PARP cleavage done by resveratrol, concluding that NF-κB has little or no role in mechanism of resveratrol mediated melanoma cell death (Figure 1H). MITF knock down induced PARP cleavage and increased resveratrol treated PARP cleavage (Figure 1I). Overall, resveratrol induced potent melanoma cell death by inducing apoptosis. The primary reason for this melanoma specific cell death is because of resveratrol's ability to inhibit MITF. These data warrants further study of mechanism, upstream of MITF, in order to improve resveratrol based chemotherapy for melanoma.

2) Role of ERK and p53 in resveratrol mediated melanoma cell death

We were interested in studying the signaling intermediates that are modulated by resveratrol, leading to inhibition of MITF and activation of melanoma cell death. As MAPK pathway with gain of function mutations in B-Raf (especially ^{V600E}B-Raf) is the most activated signaling mechanism in melanoma, we hypothesized that resveratrol might be inhibiting MAPK pathway similar to vemurafenib. To our surprise, it activated phosphorylation of many kinases such as ERK1/2, Akt and AMPKα. It also activated p53, showing its role in the apoptosis induced by resveratrol (Figure 2A). Apoptosis activation by ERK, in p53 dependent manner was previously reported by many studies. To find out the upstream MAPK component (either B-Raf or MEK1/2) responsible for resveratrol mediated ERK activation and MITF inhibition, specific inhibitors (vemurafenib for B-Raf and PD98059 for MEK1/2) were used. Both of them were unable to inhibit p-ERK1/2 or MITF downregulation (Figure 2B). Kinase assay for ERK using MBP as substrate also confirmed the same findings (Figure 2C). Specific ERK inhibitor, SCH772984 was used to deplore the mechanism of cell death downstream of ERK. SCH772984 is a dual inhibitor, where it inhibits activity of p-ERK as well as its phosphorylation by upstream MEK1/2. This compound partially inhibited ERK phosphorylation and p53 activation, but not PARP cleavage (Figure 2D). Even the co-treatment of SCH772984 with resveratrol did not decrease the cell death caused by resveratrol, suggesting there is more in the mechanism than that meets the eye (Figure 2E). We further wanted to explore the role of p53. We made stable cell lines expressing shRNA for p53. Knock down of p53 rescued approximately 25% of resveratrol mediated cell death, indicating p53's need for

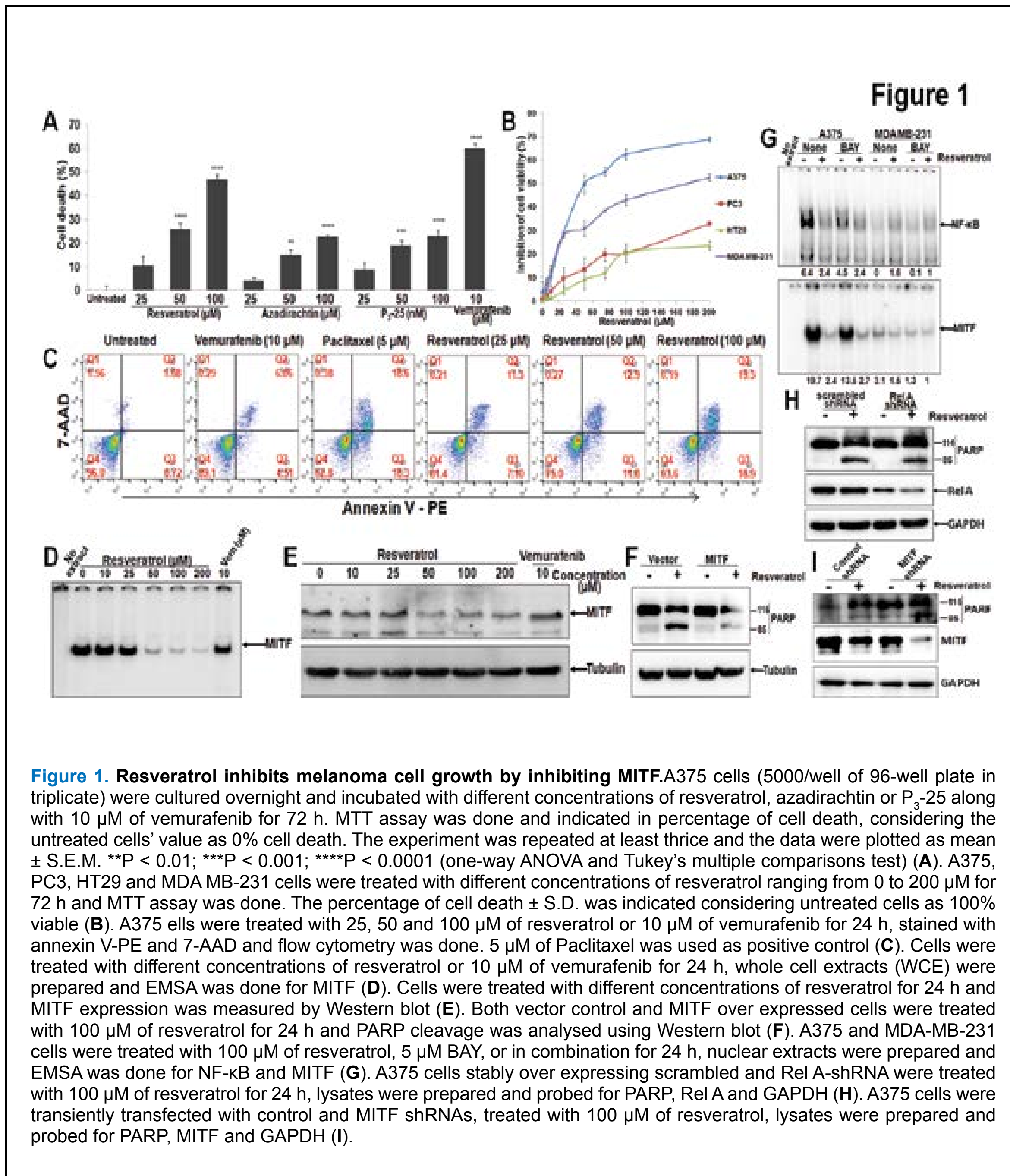

Figure 1. Resveratrol inhibits melanoma cell growth by inhibiting MITF. A375 cells (5000/well of 96-well plate in triplicate) were cultured overnight and incubated with different concentrations of resveratrol, azadirachtin or P_3-25 along with 10 µM of vemurafenib for 72 h. MTT assay was done and indicated in percentage of cell death, considering the untreated cells' value as 0% cell death. The experiment was repeated at least thrice and the data were plotted as mean ± S.E.M. **P < 0.01; ***P < 0.001; ****P < 0.0001 (one-way ANOVA and Tukey's multiple comparisons test) (**A**). A375, PC3, HT29 and MDA MB-231 cells were treated with different concentrations of resveratrol ranging from 0 to 200 µM for 72 h and MTT assay was done. The percentage of cell death ± S.D. was indicated considering untreated cells as 100% viable (**B**). A375 ells were treated with 25, 50 and 100 µM of resveratrol or 10 µM of vemurafenib for 24 h, stained with annexin V-PE and 7-AAD and flow cytometry was done. 5 µM of Paclitaxel was used as positive control (**C**). Cells were treated with different concentrations of resveratrol or 10 µM of vemurafenib for 24 h, whole cell extracts (WCE) were prepared and EMSA was done for MITF (**D**). Cells were treated with different concentrations of resveratrol for 24 h and MITF expression was measured by Western blot (**E**). Both vector control and MITF over expressed cells were treated with 100 µM of resveratrol for 24 h and PARP cleavage was analysed using Western blot (**F**). A375 and MDA-MB-231 cells were treated with 100 µM of resveratrol, 5 µM BAY, or in combination for 24 h, nuclear extracts were prepared and EMSA was done for NF-κB and MITF (**G**). A375 cells stably over expressing scrambled and Rel A-shRNA were treated with 100 µM of resveratrol for 24 h, lysates were prepared and probed for PARP, Rel A and GAPDH (**H**). A375 cells were transiently transfected with control and MITF shRNAs, treated with 100 µM of resveratrol, lysates were prepared and probed for PARP, MITF and GAPDH (**I**).

resveratrol (Figure 2F). Overexpression of MITF in p53 knock down background rescued it even further (Figure 2G). These data conclude that both inhibition of MITF and activation of p53 can have role in mechanism of cell death. But knock down of MITF in p53 knock down background, brought the cell death equal to just MITF knock down levels (Figure 2H). This allocates more importance to MITF inhibition as p53 knock down cannot rescue. Our findings conclude that, resveratrol activates many signaling intermediates. ERK1/2 is one of them, which could be involved in p53 mediated apoptosis. We need further evidence to establish role of ERK1/2 in resveratrol mediated p53 activation, as this is necessary for melanoma cell death. Mechanism needs to be explored further until identifying the direct targets of resveratrol in melanoma.

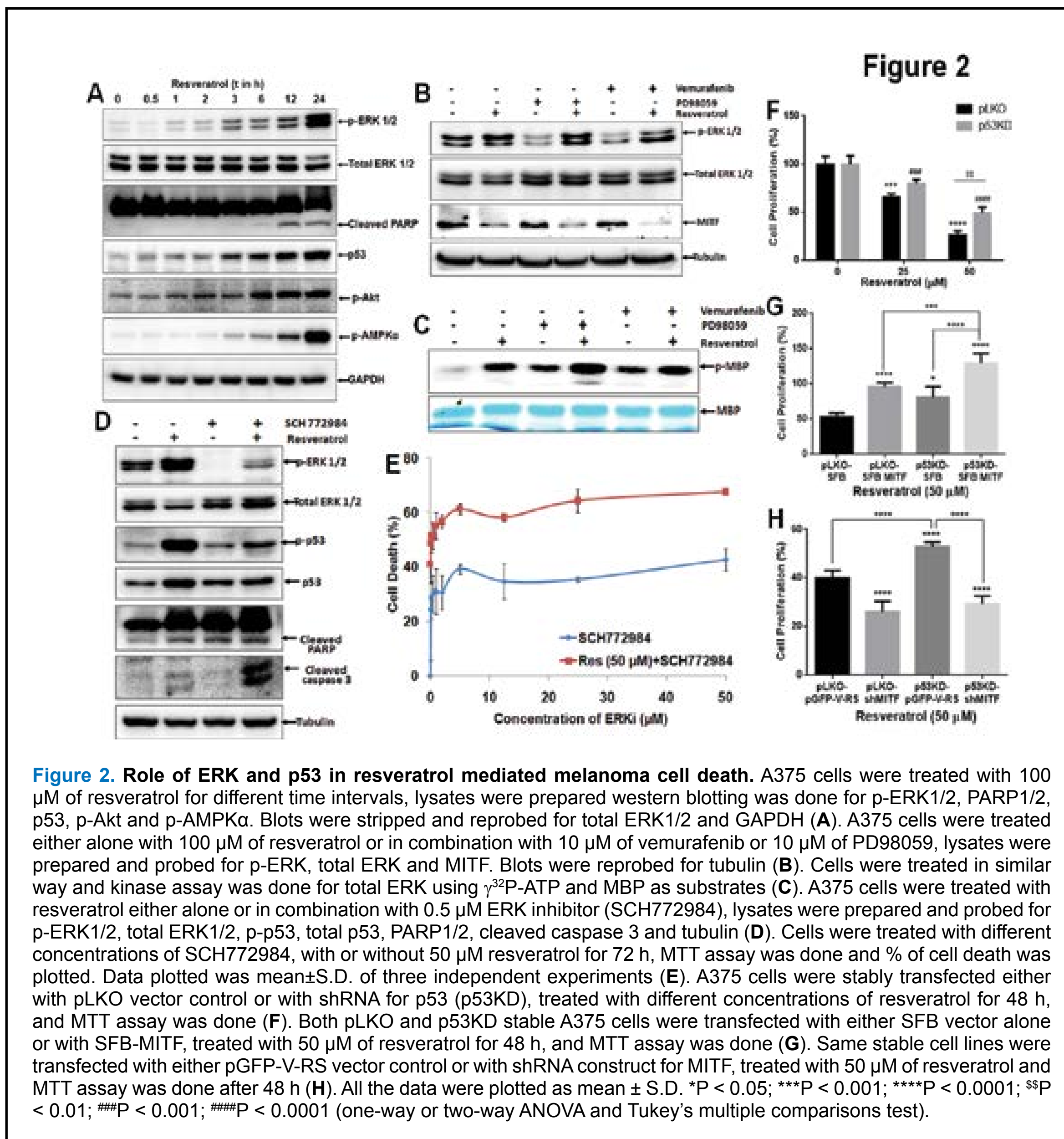

Figure 2. Role of ERK and p53 in resveratrol mediated melanoma cell death. A375 cells were treated with 100 μM of resveratrol for different time intervals, lysates were prepared western blotting was done for p-ERK1/2, PARP1/2, p53, p-Akt and p-AMPKα. Blots were stripped and reprobed for total ERK1/2 and GAPDH (**A**). A375 cells were treated either alone with 100 μM of resveratrol or in combination with 10 μM of vemurafenib or 10 μM of PD98059, lysates were prepared and probed for p-ERK, total ERK and MITF. Blots were reprobed for tubulin (**B**). Cells were treated in similar way and kinase assay was done for total ERK using γ^{32}P-ATP and MBP as substrates (**C**). A375 cells were treated with resveratrol either alone or in combination with 0.5 μM ERK inhibitor (SCH772984), lysates were prepared and probed for p-ERK1/2, total ERK1/2, p-p53, total p53, PARP1/2, cleaved caspase 3 and tubulin (**D**). Cells were treated with different concentrations of SCH772984, with or without 50 μM resveratrol for 72 h, MTT assay was done and % of cell death was plotted. Data plotted was mean±S.D. of three independent experiments (**E**). A375 cells were stably transfected either with pLKO vector control or with shRNA for p53 (p53KD), treated with different concentrations of resveratrol for 48 h, and MTT assay was done (**F**). Both pLKO and p53KD stable A375 cells were transfected with either SFB vector alone or with SFB-MITF, treated with 50 μM of resveratrol for 48 h, and MTT assay was done (**G**). Same stable cell lines were transfected with either pGFP-V-RS vector control or with shRNA construct for MITF, treated with 50 μM of resveratrol and MTT assay was done after 48 h (**H**). All the data were plotted as mean ± S.D. $^{*}P < 0.05$; $^{***}P < 0.001$; $^{****}P < 0.0001$; $^{\$\$}P < 0.01$; $^{\#\#\#}P < 0.001$; $^{\#\#\#\#}P < 0.0001$ (one-way or two-way ANOVA and Tukey's multiple comparisons test).

Publications

1. Zaidi AH, and Manna SK. (2016) Profilin-PTEN interaction suppresses NF-kappa B activation via inhibition of IKK phosphorylation. **Biochemical Journal.** 473: 859-872

2. Zaidi AH, Raviprakash N, Mokhamatam RB, Gupta P, and Manna SK. (2016) Profilin potentiates chemotherapeutic agents mediated cell death via suppression of NF-kappa B and upregulation of p53. **Apoptosis** 21: 502-513

3. Verma N, and Manna SK. (2016) Advanced Glycation End Products (AGE) Potently Induce Autophagy through Activation of RAF Protein Kinase and Nuclear Factor κB (NF-κB). **Journal of Biological Chemistry** 291: 1461-1491

4. Mokhamatam RB, Sahoo B, and Manna SK. (2016) Suppression of microphthalmia-associated transcription factor, but not NF-kappa B sensitizes melanoma specific cell death. **Apoptosis** 21: 928-940.

5. Basu Baul TS, Kehie P, Duthie A, Guchhait N, Raviprakash N, Mokhamatam RB, Manna SK, Armata N, Scopelliti M, Wang R, and Englert U (2017) Synthesis, photophysical properties and structures of organotin- Schiff bases utilizing aromatic amino acid from the chiral pool and evaluation of the biological perspective of a triphenyltin compound. **Journal of Inorganic Biochemistry** 168: 76-89.

6. Basu Baul TS, Dutta D, Duthie A, Guchhait N, Rocha BGM,Guedes da Silva MFC, Mokhamatam RB, Raviprakash N, and Manna SK (2017) New dibutyltin(IV) ladders: Syntheses, structures and, optimization and evaluation of cytotoxic potential employing A375 (melanoma) and HCT116 (colon carcinoma) cell lines in vitro. **Journal of Inorganic Biochemistry** 166: 34-48.

In Press

Verma N, and **Manna SK**. (2017) AGE potentiates cell death in p53 negative cells via upregulaion of NF-kappaB and impairment of autophagy. **Journal of Cellular Physiology** (2017 Jan 27. doi: 10.1002/jcp.25828).

LABORATORY OF MAMMALIAN GENETICS

Epigenetic mechanisms underlying developmental pathways

Faculty	Sanjeev Khosla	Staff Scientist
Ph D Students	Rachana Roshan Dev	Senior Research Fellow
	Imtiyaz Yaseen	Senior Research Fellow
	Thushara Thamban	Senior Research Fellow
	Ramisetti Rajeev	Senior Research Fellow
	Viplove Agarwaal	Senior Research Fellow
	Ambey Prasad Dwivedi	Junior Research Fellow
	Anunay Sinha	Junior Research Fellow
Other Members	Archana Tomar	Bioinformatician
	M Srilalitha	Technical Officer
	Prakruti Singh	Research Associate
	Mohammed Hassan	Laboratory Assistant
Collaborators	Gayatri Ramakrishna	ILBS, New Delhi
	Shekhar Mande	NCCS, Pune
	Rakesh Mishra	CCMB, Hyderabad
	Vinay K. Nandicoori	NII, New Delhi
	V Nagaraja	JNCASR&IISc, Bangalore
	Sharmistha Banerjee	UoH, Hyderabad
	Manjula Sritharan	UoH, Hyderabad

Project 1: *Dnmt2* and RNA processing

Summary of work done until the beginning of this reporting year (up to March 31, 2016)

DNMT2 has been categorized as a DNA methyltransferase but studies have failed to show significant DNA methylation activity under *in vitro* and *in vivo* conditions. Previous studies from our laboratory has shown the involvement of Dnmt2 in RNA processing especially during cellular stress.

Details of progress made in the current reporting year (April 1, 2016- March 31, 2017)

Previous work from our laboratory had shown DNMT2 to be a component of stress granules. DNMT2 not only relocalized to cytoplasmic stress granules (SG) in response to oxidative and endoplasmic reticulum (ER) stress but it was also found to be interacting and colocalizing with established stress granule markers like G3BP and TTP. Our results also showed this relocalization was not just restricted to oxidative and endoplasmic reticulum (ER) stress as we found relocalisation of DNMT2 to the cytoplasmic stress granules even under other stress conditions including low pH and osmotic shock.

Since infection by a pathogen also causes stress to the cell, we investigated whether infection of a cell by a virus could also cause DNMT2 relocalization. CEMx174 cells were infected with HIV-1 and the localization of the endogenous DNMT2 was observed at different time intervals by immunofluorescence, 12 hrs to 72 hrs post infection. We observed dynamic relocalization of the DNMT2 protein from the nucleus to the cytoplasmic stress granules. DNMT2 was found to be predominantly nuclear in uninfected cells (Figure 1A, topmost panel). By 12 hrs, DNMT2 was found to be present both in the cytoplasm and the nucleus (Figure 1A, second panel from top). Twenty four hours after infection, DNMT2 was completely relocalized to the cytoplasmic stress granules. The predominant cytoplasmic localization persisted till 36 hrs post infection and by 72 hrs, DNMT2 was found both in the cytoplasm and the nucleus. (Figure 1A). The quantitation of DNMT2 signal in uninfected and HIV-1 infected CEMx174 cells also confirmed significant localization of DNMT2 in cytoplasm after HIV-1 infections (Figure 1B). As a control, to confirm that the DNMT2 relocalisation was correlated with HIV-1 infection, the cells were incubated with heat-killed HIV viral particles. No

relocalisation of DNMT2 was observed in these cells infected with heat-killed HIV virus particles. Thus, the DNMT2 protein responds to multiple cellular stresses including HIV-1infection and gets localized to the stress granules. Further work to characterize the role of DNMT2 during HIV infection is being undertaken in the laboratory.

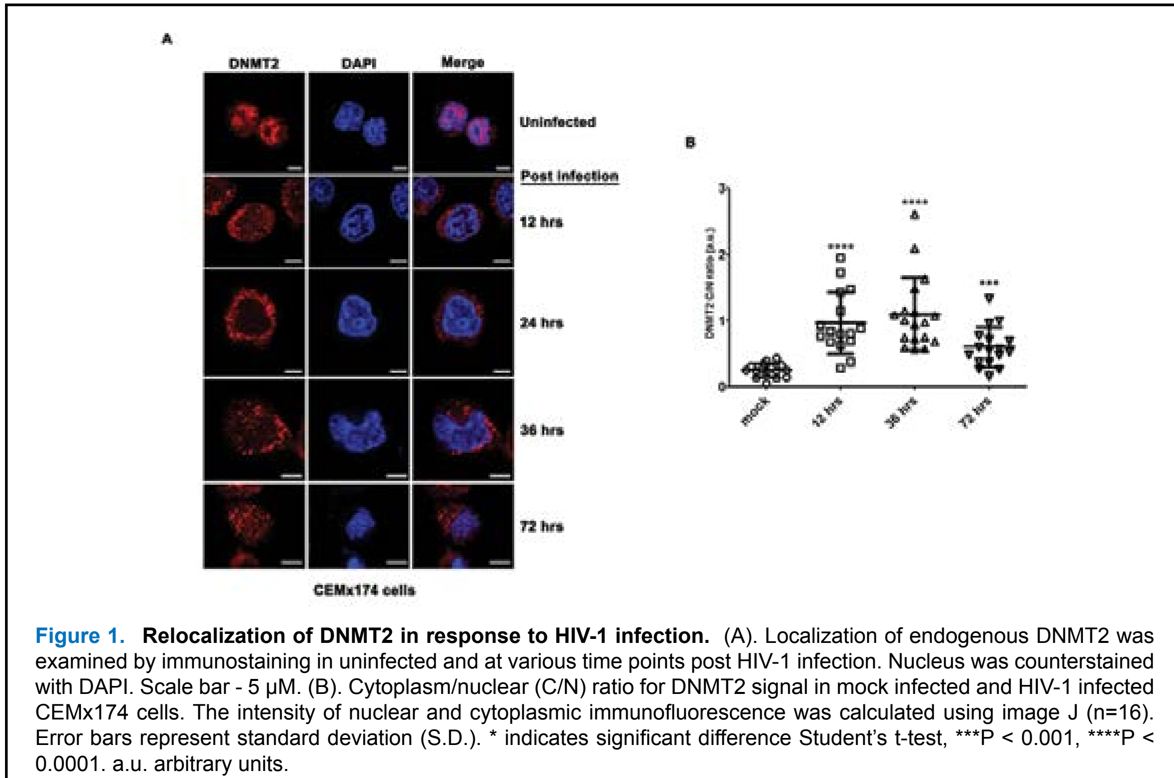

Figure 1. Relocalization of DNMT2 in response to HIV-1 infection. (A). Localization of endogenous DNMT2 was examined by immunostaining in uninfected and at various time points post HIV-1 infection. Nucleus was counterstained with DAPI. Scale bar - 5 μM. (B). Cytoplasm/nuclear (C/N) ratio for DNMT2 signal in mock infected and HIV-1 infected CEMx174 cells. The intensity of nuclear and cytoplasmic immunofluorescence was calculated using image J (n=16). Error bars represent standard deviation (S.D.). * indicates significant difference Student's t-test, ***P < 0.001, ****P < 0.0001. a.u. arbitrary units.

Project 2: Host epigenetic response to infection

Summary of work done until the beginning of this reporting year (up to March 31, 2016)

We have previously identified mycobacteria encodedDNA methyltransferase (Rv2966c)and a histone methyltransferase (Rv1988) which have the ability to methylate cytosinesand histone H3 in the host genome in a non-canonical manner. This methylation ability was found to be correlated with change in the expression of specific host genes.

Details of progress made in the current reporting year (April 1, 2016- March 31, 2017)

We have previously shown that mycobacterial species have devised efficient epigenetic mechanisms by which they try to directly control host cell gene expression. Rv2966c and Rv1988 help mycobacteria hijack the epigenetic circuitry by directly interacting with the host chromatin and methylating cytosines in the host DNA and a novel non-tail arginine in histone H3 respectively. Moreover, we showed genome-wide changes in the DNA methylation of the host during mycobacterial infection. To (i) activate genes involved in immune response, (ii) prevent the mycobacteria from making changes to its epigenetic profile or (iii) reverse the epigenetic modifications made by the mycobacterial proteins, it is conceivable that the host cell also brings about changes in the expression of epigenetic effector proteins like DNA and histone methyltransferases that are involved in establishing the epigenetic modifications. Therefore, we wanted to identify host epigenetic effector proteins that play a role in host response to mycobacterial infection and also characterize the downstream changes in epigenetic modifications that ensue.

In a preliminary experiment, where we examined the expression profile of several histone methyltransferases and demethylases in PMA treated THP1 cells (THP1 macrophages) upon *M. bovis* BCG infection, we found increase in the expression of SUV39H1 (KMT1A), the histone H3K9 methyltransferase. SUV39H1 expression

level, a protein that is normally expressed at very low levels in THP1 macrophages, was markedly increased during *M. bovis*BCG infection (Figure 2A). The increase in this expression was gradual and specific to infection by mycobacterial species (*M. bovis*BCG;Figure 2B); *M. smegmatis* and *M. tuberculosis*. THP1 macrophages infected with *E. coli* or *Candida glabrata*did not show any increase in SUV39H1 expression level.

In addition to being overexpressed in infected cells, SUV39H1 was also found to be predominantly localised in the cytoplasm (Figure 2B, upper two panels) as compared to uninfected or heat-killed *M. bovis* BCG infected THP1 macrophages where it was present in the nucleus (Figure 2B, lower two panels). We also noticed two different localization profiles of SUV39H1 in the cytoplasm of infected THP1 macrophages. As seen in the uppermost panel of Figure 2B, the localisation of SUV39H1 in the cytoplasm was found to be speckled in most cells. However, we also observed in some fields that cells not showing the SUV39H1 speckles were stained at the cell surface for SUV39H1 (Figure 2B, second panel from top).

Cytoplasmic localization of SUV39H1 during *M. bovis* BCG infection was also confirmed by western blotting proteins corresponding to cytoplasmic and nuclear fraction of *M. bovis* BCG infected THP1 macrophages and probing for the presence of SUV39H1. The purity of the subcellular fractions (Figure 2C) were confirmed by localisation of histone H3 (nucleus) and Tubulin (cytoplasmic). As compared to uninfected

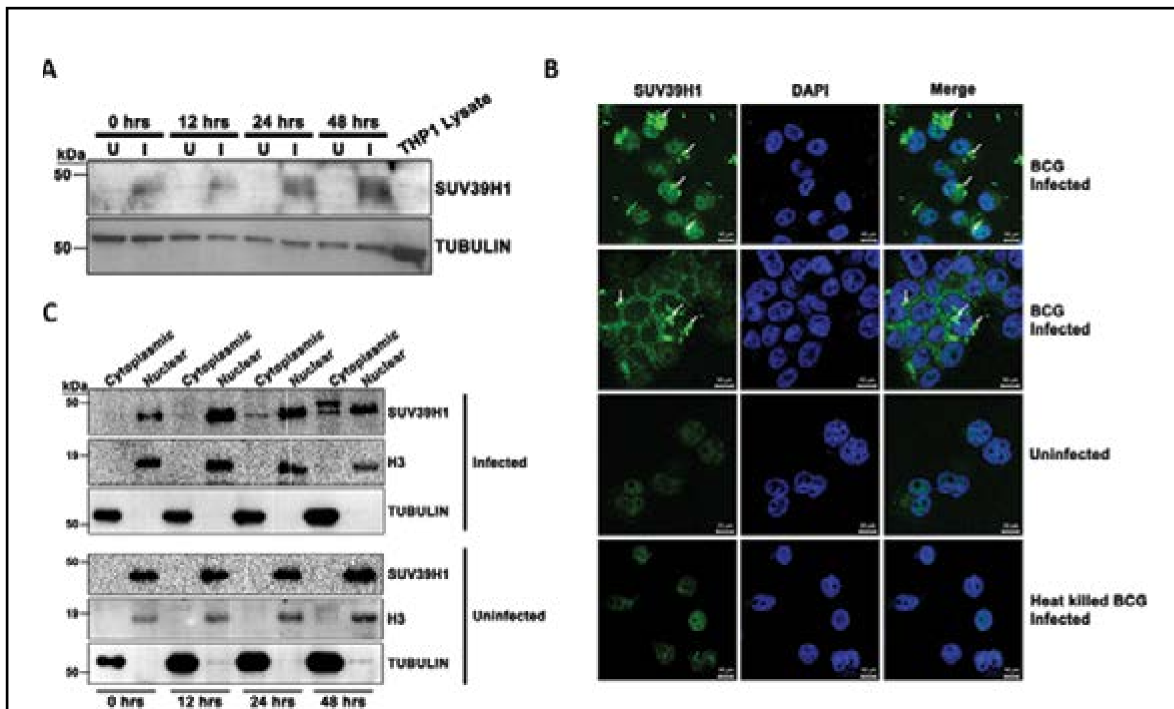

Figure 2. SUV39H1 is over expressed during mycobacterial infection. A.) Uninfected (U) and *M. bovis* BCG infected (I) THP1 macrophages were examined for the expression level of SUV39H1 (top panel) by western blotting at different time points post infection (indicated above the panels). TUBULIN was used as a control (bottom panel).B.) Uninfected (second panel from below), *M. bovis* BCG infected (upper two panels) and heat-killed *M. bovis* BCG infected (lowermost panel) THP1 macrophages were immuno-stained for SUV39H1and visualised by confocal microscopy. Note the speckled loci of SUV39H1 in the cytoplasm (marked by arrows in upper two panels) and on the cell surface (second panel from top) in infected THP1 macrophages in contrast to uninfected cells where the staining was predominantly in the nucleus. Nuclei were counter stained with DAPI. Scale bar - 10 μM. C. Western blot showing presence of SUV39H1 in the cytoplasm during mycobacterial infection. Nuclear and cytoplasmic fractions of uninfected (lower panel) and *M. bovis* BCG infected (upper panel) THP1 macrophages at different time points after infection (indicated below the panels) were examined for the presence of SUV39H1 by western blotting. As a control, the blots were also probed with H3 (nuclear) and TUBULIN (cytoplasmic) antibodies.

THP1 macrophages where it was detected only in the nuclear fraction (Figure 2C, bottom panel), SUV39H1 was detected in both nuclear and cytoplasmic fractions of *M. bovis*BCG infected THP1 macrophages (Figure 2C, upper panel). While the level of SUV39H1 increased in both fractions, there was a substantial increase in its level in the cytoplasmic fraction with increasing time, post infection. Further work to characterize the role of SUV39H1 during infection is underway in the laboratory.

Publications:

1. Basu A,Tomar A, Dasari V, Mishra RK*, and **Khosla S*** (2016) DNMT3L enables accumulation and inheritance of epimutations in transgenic Drosophila. **Scientific Reports** 6:19572.* *corresponding authors*
2. Sharma G, Sowpati DT, Singh P, Khan MZ, Ganji R, Upadhyay S, Banerjee S, Nandicoori VK, and **Khosla S.** (2016) Genome-wide non-CpG methylation of the host genome during M. tuberculosis infection. **Scientific Reports** 6: 25006.
3. Anwar T, **Khosla S** and Ramakrishna G (2016) Increased expression of SIRT2 is a novel marker of cellular senescence and is dependent on wild type p53 status. **Cell Cycle** 15: 1883-1897.

Other Publications

1. **Khosla S***, Sharma G and Yaseen I (2016). Learning epigenetic regulation from mycobacteria. **Microbial Cell**3: 92-94.* *corresponding author*

LABORATORY OF MOLECULAR CELL BIOLOGY

Signal transduction pathways in macrophages and host-pathogen interaction in tuberculosis

Faculty	Sangita Mukhopadhyay	Staff Scientist
PhD Students	Atul Udgata	Senior Research Fellow (till May 2016)
	Gourango Pradhan	Senior Research Fellow
	Parul Singh	Senior Research Fellow
	Vishwanath Jha	Senior Research Fellow
	Komal Dolasia	Senior Research Fellow
	Shruti Srivastava	Senior Research Fellow
	KM Rohini	Senior Research Fellow
	Ravi Pal	Junior Research Fellow
	Manoj Kumar	Junior Research Fellow (since July 2016)
Other Members	R. Nagender Rao	DST-SERB Project Investigator
	Niteen Pathak	Senior Technical Officer
	Philip Abraham	ICMR Research Associate
	Madhu Babu Battu	DST-National Postdoctoral Fellow (since Sept. 2016)
	Rahila Qureshi	ICMR Senior Research Fellow
	Faiza Nazar	Project Assistant
Collaborators	Prof. Seyed E Hasnain	Jamia Hamdard (Hamdard University), New Delhi
	Dr. G. Narahari Sastry	IICT, Hyderabad
	Dr. Sudip Ghosh	NIN, Hyderabad
	Dr. Sanjeeva Srivastava	IIT-B, Mumbai
	Dr. JRC Reddy	IICT, Hyderabad
	Prof. Anand Kondapi	University of Hyderabad, Hyderabad
	Dr. Gaddam Sumanlatha	Osmania University, Hyderabad
	Dr. Vijaya Laxmi Valluri	Bhagwan Mahavir Medical Research Centre, Hyderabad
	Dr. Vinay Nandikoori	NII, New Delhi

Objectives

Signal transduction pathways in macrophages regulating its innate-effector functions and how various candidate proteins of *Mycobacterium tuberculosis* (Mtb) interfere with macrophage signaling cascades to modulate host's protective responses against the bacilli.

Project I: Role of PPE2 of *M. tuberculosis* as a virulent factor

Details of progress made in the current reporting year (April 1, 2016 - March 31, 2017)

In response to infection, one of the initial reactions of macrophages is to produce bursts of toxic reactive oxygen species and nitric oxide (NO) and its intermediates in order to kill the invading pathogens. In mice, production of NO is found to be one of the essential components for antimycobacterial resistance. Abrogation of *inos* gene, which catalyzes production of NO, could severely compromise the virulence of Mtb. However, the role of NO in human tuberculosis is controversial. But we need to keep in mind why do Mtb care to retain several genes like *noxR1*, *noxR3*, *ahpC* to neutralize toxic effects of NO and its intermediates? Indeed, several lines of evidences suggest that NO do play a significant contributory role in human host defense against Mtb infection. The Mtb PE/PPE proteins are now emerging as the key components of complex mycobacterial virulence mechanisms that can modulate the host cellular machinery for its survival and persistence *in vivo*. Microarray studies have shown that expression of PPE2 (Rv0256c) is upregulated in Mtb during hypoxia and NO stress and is also upregulated in *DosS*-null mutants upon exposure to NO. In

both laboratory and clinical strains, expression of *ppe2* is increased when Mtb is exposed to the macrophage environment indicating that PPE2 may play a role in protecting the bacilli from NO and/or oxidative stress. We found that PPE2 is a secretory protein and *ppe2*-null mutants allowed higher production of nitric oxide in macrophages when compared with the wild-type strains. These observations suggest that PPE2 may help the bacteria to inhibit NO production and could be a virulent factor. The sequence analysis of PPE2 predicted presence of a strong monopartite nuclear localization signal as well as a leucine zipper motif at the C-terminal region of PPE2 with 100% probability of nuclear transport (characteristic of many eukaryotic transcription factors). Though rare in animal bacteria, several plant pathogenic bacteria possess NLS-containing effector proteins that are known to be targeted to the nucleus. Nuclear targeting of effector proteins and subsequent pathology of the host cells appears to be an emerging pathogenic mechanism in bacteria.

a. PPE2 mimics eukaryotic transcription factors: We found that the monopartite NLS present in PPE2 is biologically functional, since transiently expressed GFP-tagged PPE2 in RAW 264.7 macrophages could be localized into the nucleus, whereas truncated mutants without the NLS signal (ΔNLS-PPE2) failed to do so (Figure1A). When the positively charged arginine residues in the monopartite NLS were replaced by neutral alanine residues, the mutant PPE2 (MutNLS-PPE2) also failed to be localized inside the nucleus. Nuclear import of PPE2 involved classical importin α/β since ivermectin (a specific inhibitor of importin α/β–mediated nuclear import) was able to block its nuclear import and PPE2 with intact NLS sequence was able to interact with importin α/β but not the ΔNLS-PPE2 or MutNLS-PPE2.

b. Nuclear translocation of PPE2 is important to inhibit *iNOS* transcription and NO production: It is now interesting to know whether nuclear entry of PPE2 is crucial for inhibition of NO production.We observed that macrophages expressing wild-type PPE2 could significantly inhibit formation of LPS-stimulated nitrite, but not the cells transfected with ΔNLS-PPE2 or MutNLS-PPE2 (Figure 1B). Since NO is predominantly produced by the inducible nitric oxide synthase (iNOS) in macrophages, semi-quantitative RT-PCR was performed to compare LPS-induced *inos* transcript levels in these groups and PPE2 was found to strongly inhibit *inos* gene transcription (Figure 1C). When a luciferase reporter gene driven by the *inos* promoter was transfected to RAW 264.7 macrophages stably expressing wild-type PPE2(pCX4Neo-PPE2), luciferase activity was found to be significantly inhibited upon stimulation with LPS as compared to those cells harboring truncated PPE2 (pCX4Neo-Δ NLS-PPE2), suggesting a role of PPE2 in inhibiting transcription from the *inos* promoter. PPE2 appears to be a secretory protein as it can be detected in the culture supernatants of a clinical strain of Mtb and in the cytoplasm and nucleus of macrophages infected with *PPE2*-expressing *M. smegmatis* (a non-pathogenic surrogate bacterium which naturally lacks PPE2).

c. Translocated PPE2 binds to GATA-1 elements to inhibit *inos* transcription: Expression of iNOS is known to be predominantly regulated at the level of transcription. As PPE2 was predicted to contain a leucine zipper DNA-binding motif, we speculated that PPE2 probably binds to some crucial regulatory element of the promoter important for *inos* gene transcription. In addition to major role played by NF-κB and IRF-1, the GATA transcription factors are known to play an important role in driving transcription from the promoter of the inos gene. Using Alibaba 2.1 (http:// wwwiti.cs.uni-magdeburg.de/-grabe/alibaba2), we found at least five putative GATA-1 binding sites in the 5′-upstream region of the transcriptional start site. Interestingly, one of the putative sequences (− 16 to − 25) was found to be overlapping with the TATA box close to the transcription initiation site (Figure 1D). We observed a specific binding of recombinant PPE2 protein to the GATA-1-binding oligonucleotide proximal to the TATA box of inos promoter but not with the cognate NF-κB or IRF-1-binding oligonucleotides (Figure1 E).Since a GATA-1 consensus sequence was present overlapping with *inos* TATA box, we speculated that PPE2 protein possibly sterically inhibit recruitment of transcription machinery by directly competing with binding of TATA binding protein. Alternative mechanisms in which PPE2 may inhibit transcription by binding to non-overlapping GATA-1 sites present in the upstream region of the *inos* promoter cannot be rule out. Mtb lacks classical virulence factors unlike other typical bacterial pathogens e.g. toxins produced by *Corynebacterium diptheri, Shigella*

dysenteriae or *Vibrio cholerae*. Therefore, in case of mycobacteria virulence is broadly defined as factors that are important for the progression of the tuberculosis disease, usually measured in terms of mortality as wells as increased bacterial load following infection. PPE2 was found to confer significant survival advantages both *in vitro* and *in vivo* to *M. smegmatis* which naturally lacks this protein. Bacterial loads were significantly higher in mice infected with *M. smegmatis* expressing PPE2 and were well correlated with decreased levels of *inos* transcripts

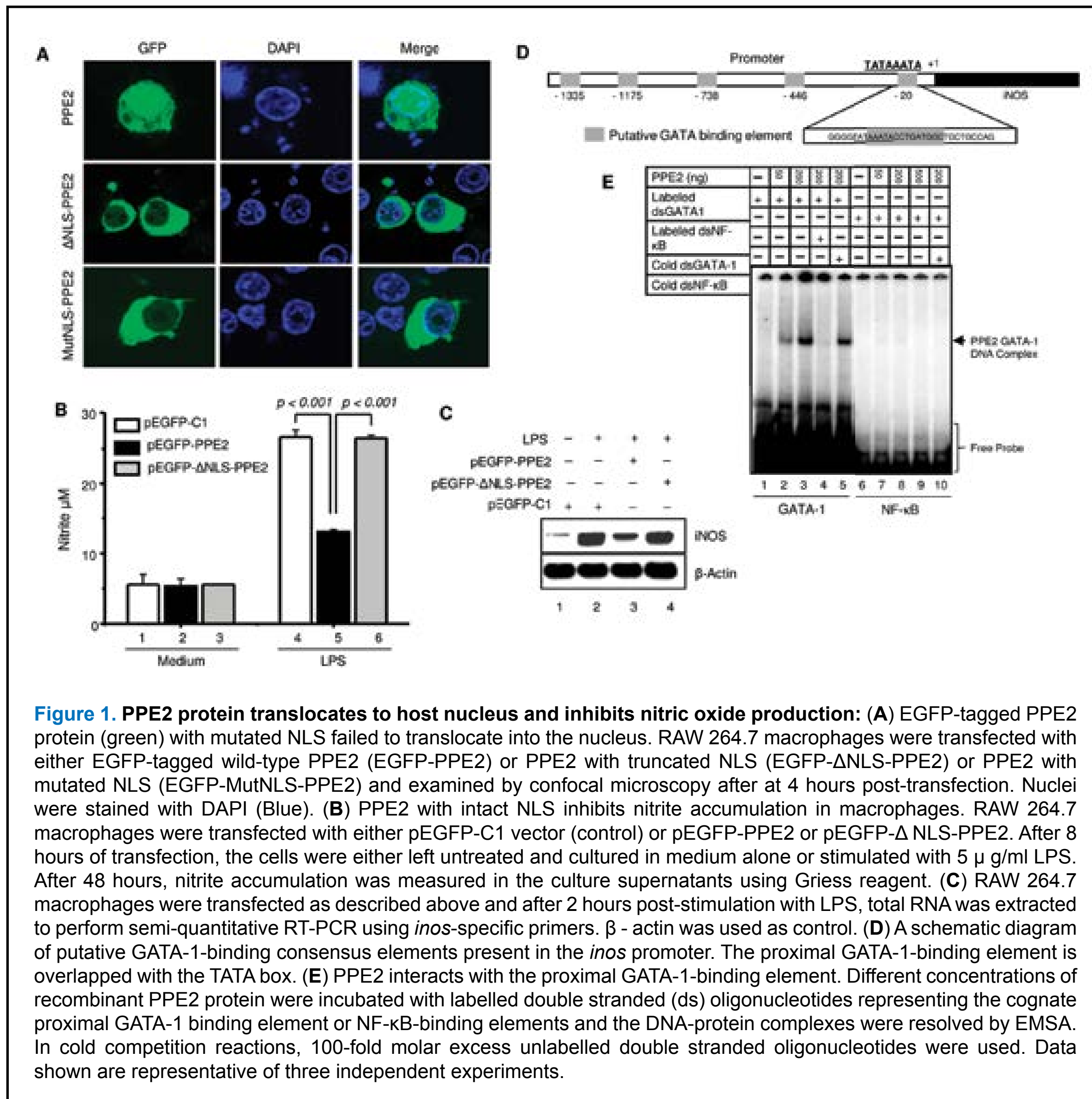

Figure 1. **PPE2 protein translocates to host nucleus and inhibits nitric oxide production:** (**A**) EGFP-tagged PPE2 protein (green) with mutated NLS failed to translocate into the nucleus. RAW 264.7 macrophages were transfected with either EGFP-tagged wild-type PPE2 (EGFP-PPE2) or PPE2 with truncated NLS (EGFP-ΔNLS-PPE2) or PPE2 with mutated NLS (EGFP-MutNLS-PPE2) and examined by confocal microscopy after at 4 hours post-transfection. Nuclei were stained with DAPI (Blue). (**B**) PPE2 with intact NLS inhibits nitrite accumulation in macrophages. RAW 264.7 macrophages were transfected with either pEGFP-C1 vector (control) or pEGFP-PPE2 or pEGFP-Δ NLS-PPE2. After 8 hours of transfection, the cells were either left untreated and cultured in medium alone or stimulated with 5 μ g/ml LPS. After 48 hours, nitrite accumulation was measured in the culture supernatants using Griess reagent. (**C**) RAW 264.7 macrophages were transfected as described above and after 2 hours post-stimulation with LPS, total RNA was extracted to perform semi-quantitative RT-PCR using *inos*-specific primers. β - actin was used as control. (**D**) A schematic diagram of putative GATA-1-binding consensus elements present in the *inos* promoter. The proximal GATA-1-binding element is overlapped with the TATA box. (**E**) PPE2 interacts with the proximal GATA-1-binding element. Different concentrations of recombinant PPE2 protein were incubated with labelled double stranded (ds) oligonucleotides representing the cognate proximal GATA-1 binding element or NF-κB-binding elements and the DNA-protein complexes were resolved by EMSA. In cold competition reactions, 100-fold molar excess unlabelled double stranded oligonucleotides were used. Data shown are representative of three independent experiments.

Future studies

PPE2 may be a novel drug target [US Patent (US-8603739B2) granted, December 10, 2013]. Our future studies are aimed at i) What are other host genes targeted by the DNA-binding domain of mycobacterial PPE2 and ii) Whether the molecules targeting the nuclear import of PPE2 be used as novel anti-mycobacterial therapeutics.

Project II: Studying the structural and molecular dynamics of ESAT-6:β2M interaction

Early secretory antigenic target (ESAT)-6 or Rv3875, an abundantly secreted protein of *Mycobacterium tuberculosis* is an important virulence factor. Inactivation of ESAT-6 leads to reduced virulence of *M. tuberculosis*. In our previous study, we demonstrated that

ESAT-6 protein alone or in complex with CFP-10 interacts with the host protein Beta-2-microglobulin (β2M), and deletion of the last 6 amino acids (VTGMFA) at the C-terminal end of ESAT-6 could disrupt the interaction of ESAT-6 with β2M indicating that the C-terminal (90-95) residues of ESAT-6 protein are important for its interaction with β2M. ESAT-6 was shown to interact and sequester β2M in the endoplasmic reticulum (ER) and thereby reduced the amount of β2M available for MHC-I-peptide complex formation resulting in downregulation of class I antigen presentation function of macrophages and $CD8^+$ T-cell responses (Sreejit et al., PLoS Pathogens, 2014). β2M is also non-covalently associated with several non-classical MHC-I proteins, like human hemochromatosis protein (HFE) and CD1. Thus, it is assumed that ESAT-6 by interacting and sequestering β2M could play an important role in modulating host immune environment and offers favorable conditions for advancement of infection. Therefore, it is crucial to gain insights into the molecular mechanism of ESAT-6:β2M complexation and the biophysical parameters governing this interaction.

Details of progress made in the current reporting year (April 1, 2016 - March 31, 2017)

a. Physico-chemical evaluation of ESAT-6:β2M complexation: In order to understand the mechanism of ESAT-6:β2M complexation, in the current study we defined the parameters governing the complexation process. We observed that ESAT-6 and β2M complex formation was an endothermic reaction with moderate strength of dissociation constant (K_d = 6.9 μM) and stoichiometry of interaction as 1:1. The energetic values for binding isotherm of ESAT-6:β2M indicates that ESAT-6 binding is positively stabilized by entropic factor. However, the strength of binding of ESAT-6:β2M is comparatively lesser than HLA:β2M ($1×10^{-8}$ M) and ESAT-6:CFP-10 ($1.1 × 10^{-8}$ M) (Figure 2A). Moreover, in the physiological condition, the concentration of ESAT-6 is decidedly regulated, which is probably high. Thus, ESAT-6 is able to bind with free β2M and manipulate macrophage responses as described earlier (Sreejit et al., PLoS Pathogens, 2014). We also observed that ESAT-6:β2M complex is stable at higher salt concentration and is possibly stabilized by hydrophobic non-covalent interactions (as indicated by fluorescence based ANS binding assay) (Figure 2B). The stability (calculating the ellipticity and the mid-point of the thermal transition as a function of temperature) study suggests that ESAT-6 in the complex is probably stabilized by β2M (Figure 2C). The interaction of the thermally stable β2M with ESAT-6 probably contributes to the stabilization of ESAT-6 in the complex at physiological condition.

b. Asp53 residue of β2M is important to form complex with ESAT-6: Interestingly, C-terminus of ESAT-6 (residues 84-95) is free and is not involved in interaction with CFP-10. Earlier, we have established that the last 6 amino acids (VTGMFA) of C-terminal region of ESAT-6 are crucial for interaction with free β2M which are not associated with HLA (Sreejit et al., PLoS Pathogens, 2014). In normal cells, β2M is non-covalently linked with the α chain polypeptide of MHC-I like molecules (MHC-I/HLA, CD1 and HFE) and makes extensive contacts with all three domains of the α chain to form complex. Association of β2M with the α chain of MHC-I, CD1 and HFE is a prerequisite for the cell-surface expression of these receptors and number of residues at the points of contact with β2M are shared among MHC-I like molecules, suggesting a common contact among these molecules. The residues of β2M that are critical for interaction with ESAT-6 are not identified previously which is an important point-of-consideration for future discovery of novel drugs. The molecular dynamics simulation studies followed by yeast two hybridization assay was therefore carried out to identify the β2M regions that are crucial for interaction with ESAT-6. Human β2M protein structure containing seven aspartate residues, Asp53, Asp59, Asp76, Asp96 and Asp98 are almost 100% conserved in all the sequences analyzed, while Asp34 and Asp38 are found substituted mostly by glutamate or by other polar-uncharged amino acids. Asp53 residue of β2M is shown to be vital for the stabilization of MHC class I heavy chain and β2M complex, however, in the isolated β2M, it is totally solvent exposed and devoid of interactions with neighboring residues. Asp53 lies in the middle of the β2M D-strand, one of the edgiest strands of the four-stranded β-sheet, creating a structural flexibility to harbor MHC class I heavy chain. Our computational and site directed mutagenesis studies clearly suggested that mutation of Asp53Ala in β2M can significantly affects the affinity of ESAT-6 to form complex with β2M (Figure 2D-F). Also, our previous results clearly indicated that ESAT-6 can suppress the levels

of HLA:β2M complex and thereby interfere with class I antigen presentation, eventually by binding to portions of the available free β2M pool before it forms complex with the HLA heavy chain. This suggests that Asp53 region of β2M is bargained by both the MHC-I and ESAT-6 molecules and ESAT-6 competitively hijacks the Asp53 site of β2M to prevent HLA:β2M complex formation.

Future Studies

Small molecules/chemical inhibitors will be screened targeting ESAT-6, and the lead molecules that inhibit interaction of ESAT-6 protein with β2M, will be tested for upregulation of class I antigen presentation function of macrophages.

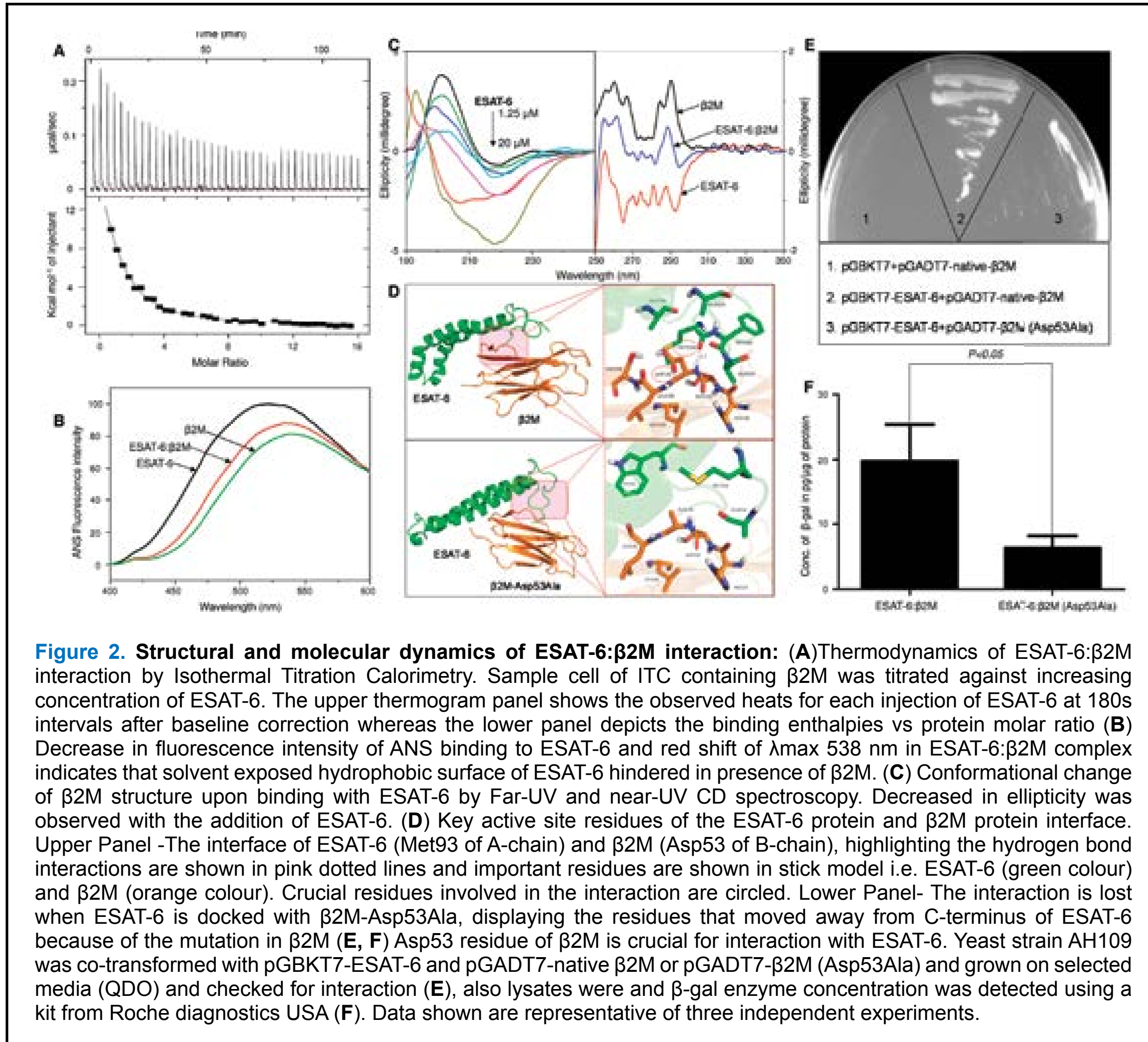

Figure 2. Structural and molecular dynamics of ESAT-6:β2M interaction: (**A**)Thermodynamics of ESAT-6:β2M interaction by Isothermal Titration Calorimetry. Sample cell of ITC containing β2M was titrated against increasing concentration of ESAT-6. The upper thermogram panel shows the observed heats for each injection of ESAT-6 at 180s intervals after baseline correction whereas the lower panel depicts the binding enthalpies vs protein molar ratio (**B**) Decrease in fluorescence intensity of ANS binding to ESAT-6 and red shift of λmax 538 nm in ESAT-6:β2M complex indicates that solvent exposed hydrophobic surface of ESAT-6 hindered in presence of β2M. (**C**) Conformational change of β2M structure upon binding with ESAT-6 by Far-UV and near-UV CD spectroscopy. Decreased in ellipticity was observed with the addition of ESAT-6. (**D**) Key active site residues of the ESAT-6 protein and β2M protein interface. Upper Panel -The interface of ESAT-6 (Met93 of A-chain) and β2M (Asp53 of B-chain), highlighting the hydrogen bond interactions are shown in pink dotted lines and important residues are shown in stick model i.e. ESAT-6 (green colour) and β2M (orange colour). Crucial residues involved in the interaction are circled. Lower Panel- The interaction is lost when ESAT-6 is docked with β2M-Asp53Ala, displaying the residues that moved away from C-terminus of ESAT-6 because of the mutation in β2M (**E, F**) Asp53 residue of β2M is crucial for interaction with ESAT-6. Yeast strain AH109 was co-transformed with pGBKT7-ESAT-6 and pGADT7-native β2M or pGADT7-β2M (Asp53Ala) and grown on selected media (QDO) and checked for interaction (**E**), also lysates were and β-gal enzyme concentration was detected using a kit from Roche diagnostics USA (**F**). Data shown are representative of three independent experiments.

Publications

i) Research papers published in the calendar year 2016 (in print with final page numbers)

1. Udgata A, Qureshi R and Mukhopadhyay S. (2016). Transduction of functionally contrasting signals by two mycobacterial PPE proteins downstream of TLR2 receptors.

Journal of Immunology197:1776-87.

2. Abraham PR, Udgata A, Latha GS and Mukhopadhyay S. (2016). The *Mycobacterium tuberculosis* PPE protein Rv1168c induces stronger B cell response than Rv0256c in active TB patients. **Infection, Genetics and Evolution** 40:339-345.

(ii) Research papers published in the calendar year 2017

1. Bhat KH, Srivastava S, Kotturu SK, Ghosh S and Mukhopadhyay S. (2017). The PPE2 protein of *Mycobacterium tuberculosis* translocates to host nucleus and inhibits nitric oxide production. **Scientific Reports** 7:39706.doi: 10.1038/srep39706.

(iii) Other Publications

1. Mukhopadhyay S and Ghosh S. (2017). *Mycobacterium tuberculosis*: what is the role of PPE2 during infection? **Future Microbiology** (Invited Editorial Article) (In Press).

2. Rameshwaram NR, Shrivastava R, Pradhan G, Singh P and Mukhopadhyay S. Phagosome-lysosome fusion hijack - An art of intracellular bacteria. **Proceedings of the Indian National Academy of Sciences** (In Press).

LABORATORY OF MOLECULAR GENETICS

(Laboratory of Molecular Genetics)

(Explanatory note: The Laboratory of Molecular Genetics, which also includes the Centre of Excellence (CoE) in Silkmoth Genetics and Genomics, was headed by CDFD's faculty member Dr. J Nagaraju who unfortunately passed away in December 2012. Subsequently, the activities of his erstwhile group have been continued by Dr K P Arun Kumar and Dr V V Satyavathi with their respective colleagues, whose individual reports are given below. The Director of CDFD is the designated coordinator of the CoE)

Faculty	KP Arun Kumar	Scientist
PhD Students	Asha Minz	Senior Research Fellow
	S Suresh Kumar	Senior Research Fellow
	G Gopinath	Senior Research Fellow (till Feb 2017)
	Ch. Gangi Reddy	Junior Research Fellow
Other Members	S Annapurna Bhavani	Technical Officer
	R Lakshmi Vaishna	Technical Assistant (till Feb 2016)
	Matta Divya	Technical Assistant (from Aug 2016)
	Saikat Chakraborty	Project JRF (till Sep 2016)
	Vidya T	Project JRF (till Oct 2016)

Objectives

1. Identification and characterization of novel antiviral proteins in *Bombyx mori*
2. Transcriptome analysis of sexed embryonic stages and larval heads of *Bombyx mori* to identify genes involved in sex determination and differentiation

The progress made in the projects related to sex determination and immune response in *B. mori* is reported here.

Summary of the work done until the beginning of this reporting year (upto March 31, 2016)

- We reported that an autosomal CCCH type zinc finger protein, Bmznf-2 induces masculinisation by promoting male type of *Bmdsx* splicing in the domesticated silkworm *B. mori*. The Bmznf-2 also induces differential splicing of *Bmtra-2* gene in BmN cells. Similar to the recently discovered *masc* gene, Bmznf-2 also appears to be a redundant masculinisation factor in the mechanism of *B. mori* sex determination. Presence of more than one upstream factor governing the sex specific splicing of *Bmdsx* pre-mRNA indicates the complexity behind evolution of sexual differentiation in *B. mori*.
- In the quest of addressing the immunological function of DmNoduler (a *Drosophila* homolog of *Noduler* - also known as putative ferric-chelate reductase 1 homolog - DmSDR2) we deciphered its vital role as a regulator of NF-κB/Rel transcription factors in both Toll and IMD immune pathways of *Drosophila*. With this study, we introduce a new factor to immune response cascades, which is unique as it regulates both pathways by affecting translocation of NF-κB factors.

Details of progress made in the current reporting year (April 1, 2016 - March 31, 2017)

Objective 1: Identification and characterization of novel antiviral proteins in *Bombyx mori*

Unlike vertebrates, insects lack antibody based adaptive immunity and mainly relies on innate immune response as first line of defense against pathogens. Innate immune system evolutionarily conserved in metazoans, involves recognition of conserved pathogen-associated molecular patterns (PAMPs) on the surface of invading organisms by host encoded pattern recognition receptors (PRRs). In insects, recognition of PAMPs by PRRs activates the Toll, the Immune Deficiency (Imd) and the Janus kinase (JAK)-signal transducer and activator of transcription (STAT) pathways, which induce humoral (antimicrobial peptides synthesis, coagulation

and melanization) and cellular (phagocytosis, nodulation and encapsulation) responses.

Innate immunity serving as a primary defense mechanism in animals involves recognition of PAMPs by the host. The host molecules that recognize PAMPs are pattern recognition molecules, and Calcium dependent lectins (C-type) constitute one such type. C-type lectins with carbohydrate recognition domains bind to various PAMPs and initiate cellular and humoral immune responses to protect the host. Lectins also mediate attachment and binding of bacteria and viruses, as well as mediate the first line of defense against invading microorganisms with MBL, the mannan binding lectin in the innate immune system.

Bombyx mori nucleopolyhedrovirus (BmNPV), a baculovirus, is the devastating pathogen of the domesticated silkworm *B. mori.* However, the molecular mechanism underlying the host resistance to virus remains elusive. To identify genes involved in immune responses of *B. mori,* two different strains - resistant SBNP1 and susceptible CSR2 were chosen. Uninfected and BmNPV infected fat body tissues of both strains were subjected to Next Generation Sequencing. Analysis of the data showed pronounced increase in the transcript level of certain immune genes such as odorant binding protein, Gloverin, C-type lectin, Juvenile hormone diol kinase and Muscle LIM upon infection, thereby suggesting possible role of these genes in BmNPV infection. C-type lectin was chosen for the present study because though the antibacterial role is well established, the role of lectin in antiviral immune response is unclear. Semi-quantitative and quantitative real time RT-PCR was done with fat body RNA of both strains. In the resistant strain, expression of lectin was significantly higher upon infection than its uninfected control, which is consistent with the NGS analysis results. However, in the susceptible strain there was no change in lectin expression in uninfected and infected RNA. Therefore resistant strain was selected for further studies. Expression of lectin was still further checked in *Bombyx mori* ovarian cell line, BmN. Again the expression of lectin was found to be highly up-regulated upon BmNPV infection.

In this study, we have shown that CTL-5 (*B. mori* encoded CTL-5) mediated JAK-STAT signaling pathway is crucial for defense against BmNPV in *B. mori*. Our results demonstrate that, CTL-5 functions as PRR to recognize BmNPV and thereby restrict viral replication. CTL-5 promotes viral resistance by triggering four AMPs or immune elicitors such as Ser1, Ser2, OBP 6 and MLP via JAK-STAT pathway. CTL-5 interacted with BmNPV virions, and this recognition is required for the activation of JAK-STAT pathway. Loss of STAT repressed the immune elicitors and was lethal to the host. These findings suggest that JAK-STAT immune pathway is a key player in anti-BmNPV defense of *B. mori*. Collectively our results provide strong evidence that CTL-5 is an important PRR that acts upstream of JAK-STAT pathway to induce immune elicitors for defense against BmNPV.

Based on our findings, we propose a hypothetical model for CTL-5 as PRR to evade BmNPV by immune elicitors induced through JAK-STAT pathway in *B. mori* (Figure 1).

BmNPV, a member of baculovirus enters the cell by endocytosis and might be mediated by GP64 envelope fusion protein as described for AcMNPV. The existence of signal peptide in N terminus of CTL-5 and the mode of viral entry made us to assume that CTL-5 acts as a cytoplasmic PRR, though evidence is lacking. Upon binding with PAMPs, numerous PRR driven signaling pathways are activated to induce cytokines. Future research should delineate the mechanism by which the cytokines are induced and subsequent activation of JAK-STAT signaling cascade in *B. mori*. In *Drosophila,* JAK-STAT pathway is found to be activated by cytokine ligands Upd1, Upd2 and Upd3. However homologs of these ligands are not found in *B. mori* indicating that the pathway is activated by unknown cytokines. Cytokine mediated JAK-STAT cascade, then transcriptionally upregulates PRR (CTL-5) and antiviral genes (Ser1, Ser2, OBP 6 and MLP). Thus JAK-STAT pathway can feed back and regulate the transcription of PRR, thereby providing a bi-directional regulatory loop between cytokines and PRRs. Our study uncovers the principle underlying the host resistance to BmNPV, which may be amenable to effective silk production. The findings reveal the essence of JAK-STAT pathway in viral immunity, thus paving way for a better understanding of host pathogen interaction and to further improve the viral resistance in economically important insects.

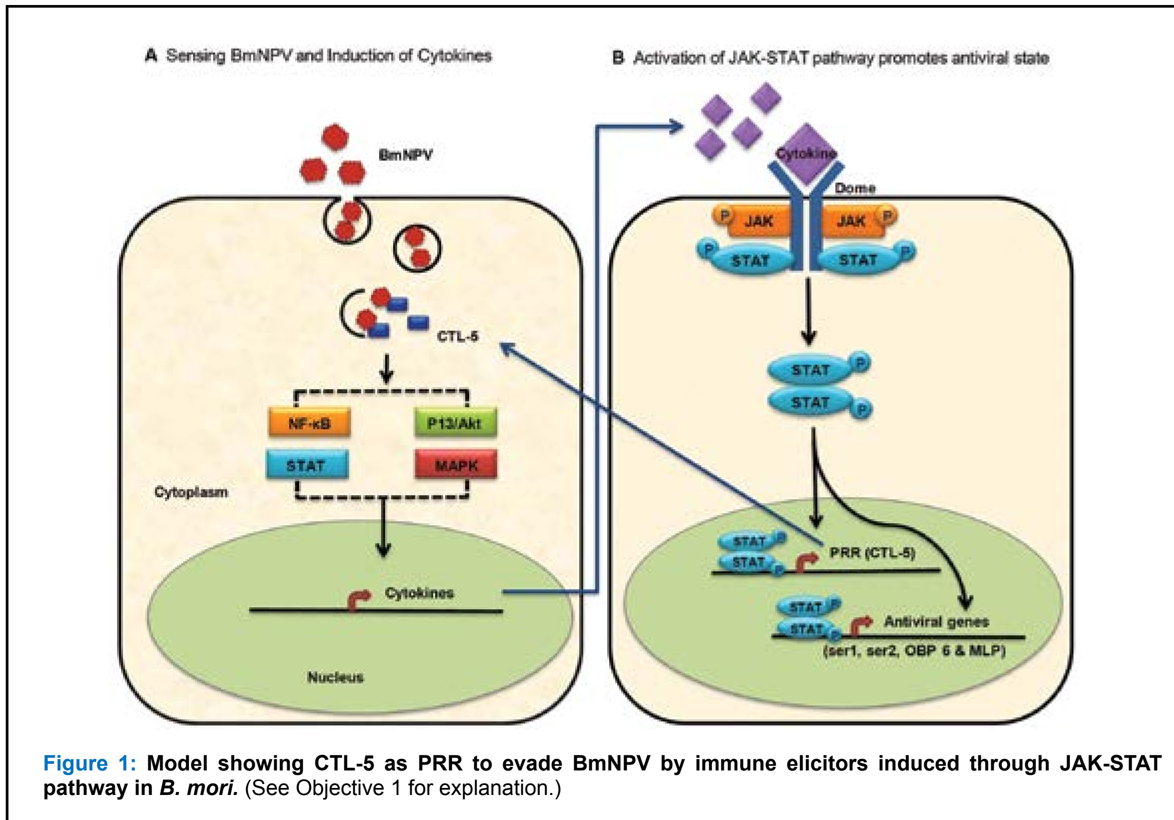

Figure 1: Model showing CTL-5 as PRR to evade BmNPV by immune elicitors induced through JAK-STAT pathway in *B. mori*. (See Objective 1 for explanation.)

Objective 2: Transcriptome analysis of sexed embryonic stages and larval heads of *Bombyx mori* to identify genes involved in sex determination and differentiation

"How sex is determined in species?" this puzzling aspect of biology had resulted in a pursuit, nearly a century ago to study the molecular mechanism behind this process. This has revealed an array of genetic cascades mostly determined by sex chromosomes. Studies on understanding the mechanism of sex determination in various taxa have led to the proposal of bottom-up theory by Adam Wilkins, where the bottom most player of the cascade is highly conserved but the top players are diverse. In insects, sex is not influenced by hormones and every cell maintains its own sex, hence gynandromorphs are possible. The sex determination cascade involves a primary signal mostly genetical, coming from sex chromosomes that activates a "key gene", which in turn takes control of sub-ordinate control genes - finally driving the double switch (*dsx* gene). The striking differences between male and female originate from the differential splicing of *dsx* pre-mRNA, producing sex-specific proteins that are antagonistic in the process of sexual differentiation and development. In most of the insects studied for sex determination, there is conservation to some extent at the level of "key gene" (tra), whereas this gene is not found in *B. mori* by homology search. Additionally there seems to be many regulatory factors involved in the sex specific differential splicing of *B. mori dsx* pre-mRNA (*Bmdsx*) Eg., Bmpsi, Bmimp, Masc and recently identified Bmznf-2. These two observations make the cascade of sex determination in *B. mori*, remarkably different from that of other insects.

In an attempt to identify possible new players of sex determination and W-encoded genes, RNA-sequencing was performed for early embryonic stages. The embryonic stages were selected based on the observation that *dsx* gene exhibits sex specific differential splicing at 96h. Hence, a stage before (78h) and a stage after (120h) 96h were selected for analysis. Analysis of these three stages suggested an early male biased expression at 78h and 96h stages, which gets normalized at 120h stage.

The differential gene expression analysis has revealed a set of male biased and female biased genes at 78h, 96h and 120h stages. For the identification of W-derived fragments, the genome unmapped reads were subjected to de novo-assembly. This resulted in thousands of

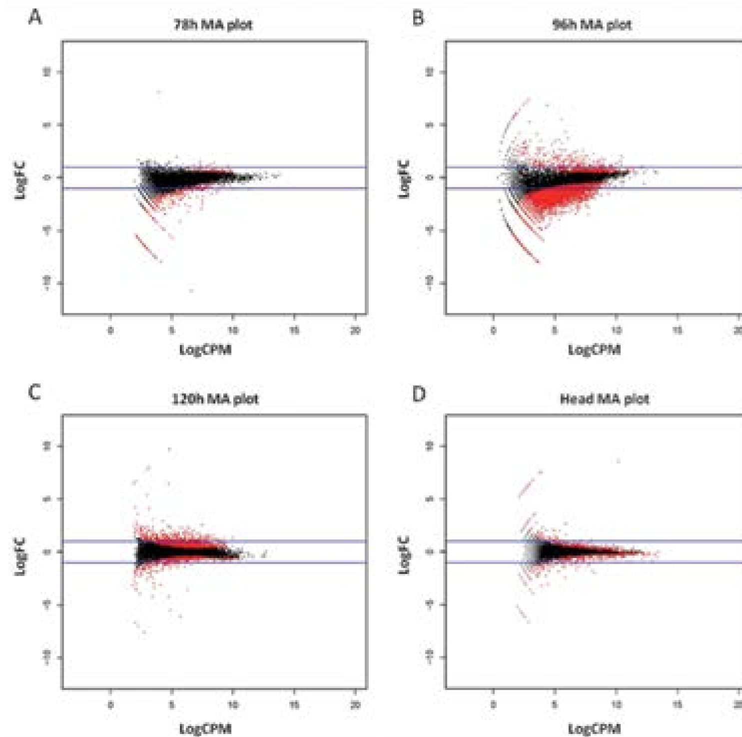

Figure 2: The MA plots [M(log ratios) and A (mean average)] of embryonic and head samples in male to female comparison. In these plots, logFC denotes the biased expression, female biased (+ve y-axis) and male biased (–ve y-axis) and logCPM represents the average expression strength of genes. A, B, C and D represents the MA plots for 78h, 96h, 120h and head samples respectively.

unmapped transcripts with ~200bp length (from male samples = 5726; female = 4667). BLAST analysis showed that nearly 50% of these transcripts (male = 2596; female = 2365) could be the precursor transcripts for the reported ovarian small RNAs in *B. mori*. These transcripts were further subjected to various levels of filtering, which resulted in 862 novel transcripts in which 225 were identified only in female samples and 423 were identified only in male samples. Out of the 225 female specific transcripts, 62 transcripts were predicted to be of W-origin based on the BLAST analysis against the W-chromosome derived BAC clones. Unfortunately no protein coding transcripts were identified among them and all the transcripts were non-coding in nature.

Several important genes involved in various metabolisms exhibited a high male biased expression in the embryonic stages, especially many zinc finger motif encoding genes and transcription factors. It is interesting to note that the zinc finger motif encoding genes that exhibited male biased expression at 78h and 96h stage are unique and none of them are male biased at 120h stage. At 120h stage, almost no zinc finger motif encoding gene exhibited a profound male biased expression, instead many zinc finger genes showed a female biased expression suggesting the dynamic expression profile of these zinc finger motif encoding genes which may be crucial in the development and sustainability of the embryos.

In the early stage of development, i.e., at 78h, hundreds of genes (520) showed a differential expression. This number surge to thousands at 96h (4068) and it decreases at 120h (2596). The DGE analysis suggested a very high male biased expression of many important genes of silk composition, developmental, transcription factors and many zinc finger genes, which must have crucial roles in the process of development and sexual differentiation. In addition, the analysis of unmapped transcripts yielded thousands of precursors for the *B. mori* small RNAs and many non-coding transcripts that are presumably W-chromosome derived. Further analysis of these unmapped transcripts may help in uncovering the W-transcriptome and thus aid in a comprehensive understanding of the role of W-chromosome in *B. mori* sex determination.

Publications

1. Gopinath G, Arunkumar KP, Mita K and Nagaraju J (2016). Role of *Bmznf-2*, a *Bombyx mori* CCCH zinc finger gene, in masculinisation and differential splicing of *Bmtra-2*. ***Insect Biochemistry and Molecular Biology*** 75: 32-44.
2. Sawanth SK, Gopinath G, Sambrani N and Arunkumar KP (2016). Autoregulatory loop: a common mechanism of regulation of key sex determining genes in insects. ***Journal of Biosciences*** 41: 283-294.
3. Shantibala T, Victor TH, Luikham R, Arunkumar KP, Sharma HD, Lokeshwari RK and Kim I (2016). Complete mitochondrial genome of the wild eri silkworm, *Samia canningi* (Ledpidoptera: Saturniidae). ***Mitochondrial DNA*** 27: 844-845.
4. Guo H, Cheng T, Chen Z, Jiang L, Guo Y, Liu J, Li S, Taniai K, Asaoka K, Kadono-Okuda K, Arunkumar KP, Wu J, Kishino H, Zhang H, Seth RK, Gopinathan KP, Montagne N, Jacquin-Joly E, Goldsmith MR, Xia Q and Mita K (2017). Expression map of a complete set of gustatory receptor genes in chemosensory organs of *Bombyx mori*. ***Insect Biochemistry and Molecular Biology*** 82: 74-82.

Other publications

1. Chakraborty S and Arunkumar KP (2016) Book review of the *Annual Review of Genetics* 2015, Bonnie Bassler et al., (eds) ***Current Science*** 111: 933-935

LABORATORY OF MOLECULAR ONCOLOGY

Genomics and molecular genetics of cancer and genetic disorders

Faculty	Murali D Bashyam	Staff Scientist
PhD Students	Raju Kumar	Senior Research Fellow
	Animireddy Srinivas	Senior Research Fellow
	Pratyusha Bala	Senior Research Fellow
	Ashmala Naz	Senior Research Fellow
	Sara Anisa George	Junior Research Fellow
Other Members	Mithu Raychaudhuri	DST woman Scientist
	A Sita Rama Raju	Research Associate
	Ajay K Chaudhary	Technical Officer
	K Viswakalyan	Project SRF (till Dec. 2016)
	K Padmavathi	Project JRF
Collaborators	A Dalal	CDFD, Hyderabad
	H A Nagarajaram	CDFD, Hyderabad
	Ravi Gupta	Med Genome Pvt Ltd, Bengaluru
	Saumyadipta Pyne	IIPH, PHFI, Hyderabad
	Ramana Davuluri	Northwestern University, USA
	Suresh Ramakrishna	Hanyang University, Korea
	G Swarnalata	Apollo Hospitals, Hyderabad
	T Subramanyeshwar Rao	BIACHRI, Hyderabad
	KVVN Raju	BIACHRI, Hyderabad
	Sujit C Patnaik	BIACHRI, Hyderabad
	M Srinivasulu	MNJ Hospital, Hyderabad
	Mohana Vamsy	Omega Hospitals, Hyderabad
	RA Sastry	KIMS, Hyderabad
	I Satish Rao	KIMS, Hyderabad
	Sankar V Hariharan	Medical College, Trivandrum

Objectives

1. Identification and characterization of important deregulated genes/pathways in cancers prevalent in India.
2. Identification and characterization of disease causing mutations in genetic disorders.

Summary of work done until the beginning of this reporting year (upto March 31, 2016)

Tongue and Esophageal Cancer: Genome-wide mRNA profiling revealed *TP53* and *SMARCD1* as the only two up-regulated transcripts in tongue cancer samples harbouring a mutant p53.

Colorectal Cancer (CRC): Computational analysis of transcriptome data generated from Wnt- and Wnt+ rectal cancer samples revealed several differentially expressed 'gene sets'. We further extracted a differentially expressed 12 gene signature; the constituent genes were validated in independent set of samples.

Details of progress made in the current reporting year (April 1, 2016 - March 31, 2017)

Tongue and Esophageal Cancer: Transcript elevation of *TP53* correlated significantly with that of *ZMAT3* in tongue cancer samples (Figure 1A); *ZMAT3* itself was transcriptionally activated by wild type as well as mutant p53 when ectopically expressed in CRC cells (Figure 1B). Thus, the *TP53-ZMAT3* positive feedback loop appears to contribute towards stabilization of the *TP53* transcript in tongue cancer. *SMARCD1* was confirmed to be a transcriptional target of non-hotspot mutant p53 using ectopic expression followed by RT-QPCR (Figure 1B), chromatin immunoprecipitation (Figure 1C) and luciferase assays (Figure 1D).

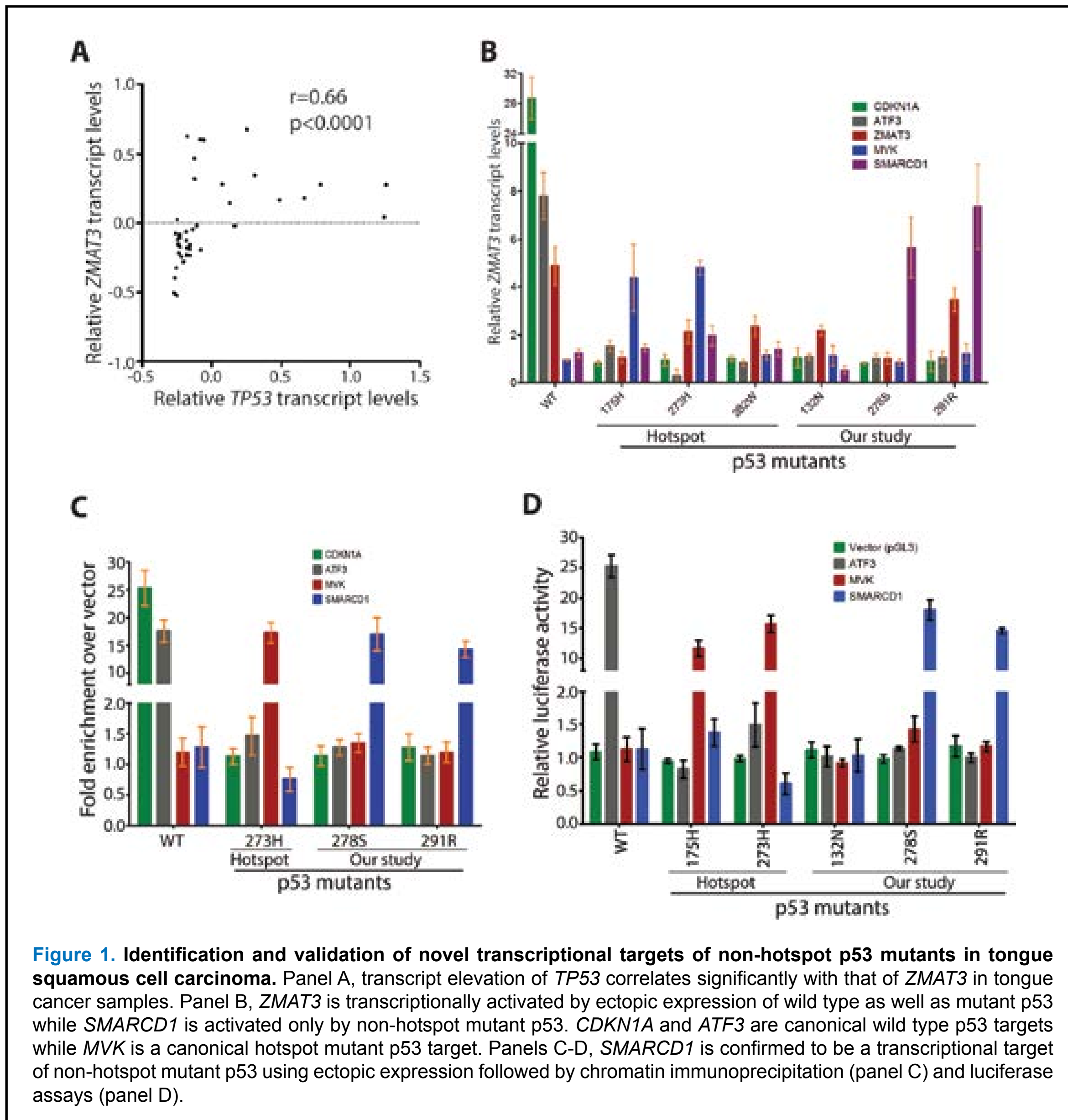

Figure 1. Identification and validation of novel transcriptional targets of non-hotspot p53 mutants in tongue squamous cell carcinoma. Panel A, transcript elevation of *TP53* correlates significantly with that of *ZMAT3* in tongue cancer samples. Panel B, *ZMAT3* is transcriptionally activated by ectopic expression of wild type as well as mutant p53 while *SMARCD1* is activated only by non-hotspot mutant p53. *CDKN1A* and *ATF3* are canonical wild type p53 targets while *MVK* is a canonical hotspot mutant p53 target. Panels C-D, *SMARCD1* is confirmed to be a transcriptional target of non-hotspot mutant p53 using ectopic expression followed by chromatin immunoprecipitation (panel C) and luciferase assays (panel D).

A similar microarray-based gene expression screen performed on esophageal squamous cell carcinoma (ESCC) samples revealed other novel transcriptional targets of mutant p53 (Figure2A) of which *ARF6*,*TRIM23* and *C1QBP* were validated in additional tumor samples (Figure 2B) and confirmed by ectopic expression of wild type and various mutant forms of p53 (Figure 2C). Further, ARF6 was confirmed to be highly expressed in p53 mutant vs wild type samples based on immunohistochemistry performed on an ESCC tissue microarray. Thus, our work has revealed novel transcriptional targets for non-hotspot mutant p53 relevant for squamous cell carcinoma.

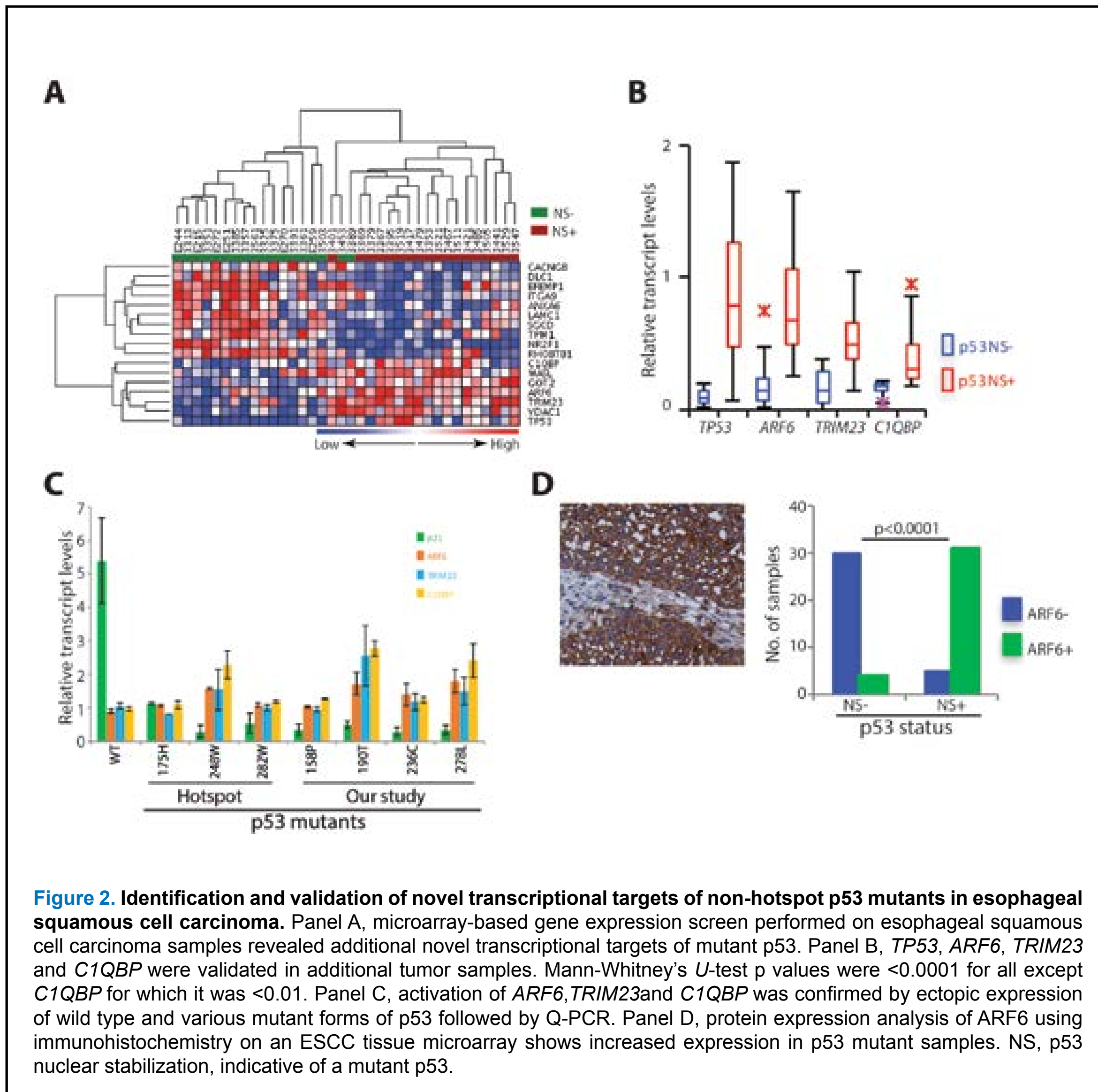

Figure 2. Identification and validation of novel transcriptional targets of non-hotspot p53 mutants in esophageal squamous cell carcinoma. Panel A, microarray-based gene expression screen performed on esophageal squamous cell carcinoma samples revealed additional novel transcriptional targets of mutant p53. Panel B, *TP53*, *ARF6*, *TRIM23* and *C1QBP* were validated in additional tumor samples. Mann-Whitney's *U*-test p values were <0.0001 for all except *C1QBP* for which it was <0.01. Panel C, activation of *ARF6*,*TRIM23*and *C1QBP* was confirmed by ectopic expression of wild type and various mutant forms of p53 followed by Q-PCR. Panel D, protein expression analysis of ARF6 using immunohistochemistry on an ESCC tissue microarray shows increased expression in p53 mutant samples. NS, p53 nuclear stabilization, indicative of a mutant p53.

CRC: Computational analysis of genome-wide gene expression data generated for rectal cancer samples revealed an enrichment of Ca^{2+}/NFAT signalling in samples devoid of canonical Wnt/β-catenin signalling. In addition, NFAT family was the most significantly enriched transcription factor class in genes differentially expressed between Wnt+ and Wnt- samples. Seven (of the total forty nine) differentially expressed genes in addition to *NFATC1* were validated in a set of rectal cancer samples not subjected to transcriptome profiling (Figure 3A). The seven genes included six putatively involved in Ca^{2+} signalling (*CDH19*, *GPC6*, *GSN*, *IRAK3*, *LRRK2* and *RUNX2*) and one in canonical Wnt signaling (*AXIN2*). More importantly, all eight validated genes alone could distinguish Wnt+ and Wnt- samples in hierarchical clustering analysis. Ectopic expression followed by RT-QPCR (Figure 3B), chromatin immunoprecipitation (Figure 3C) and luciferase assays (Figure 3D) confirmed the six differentially expressed genes to be transcriptional targets of NFATc1. These six genes in addition to *NFATC1* predicted worse survival in the TCGA CRC expression data set (Figure. 3E). Finally, we confirmed significantly elevated expression of gene coding for a non-canonical Wnt ligand namely *WNT9A*, previously suggested to activate Ca^{2+}/NFAT signalling, in Wnt- (as compared to Wnt+) tumor samples (Figure 3A).

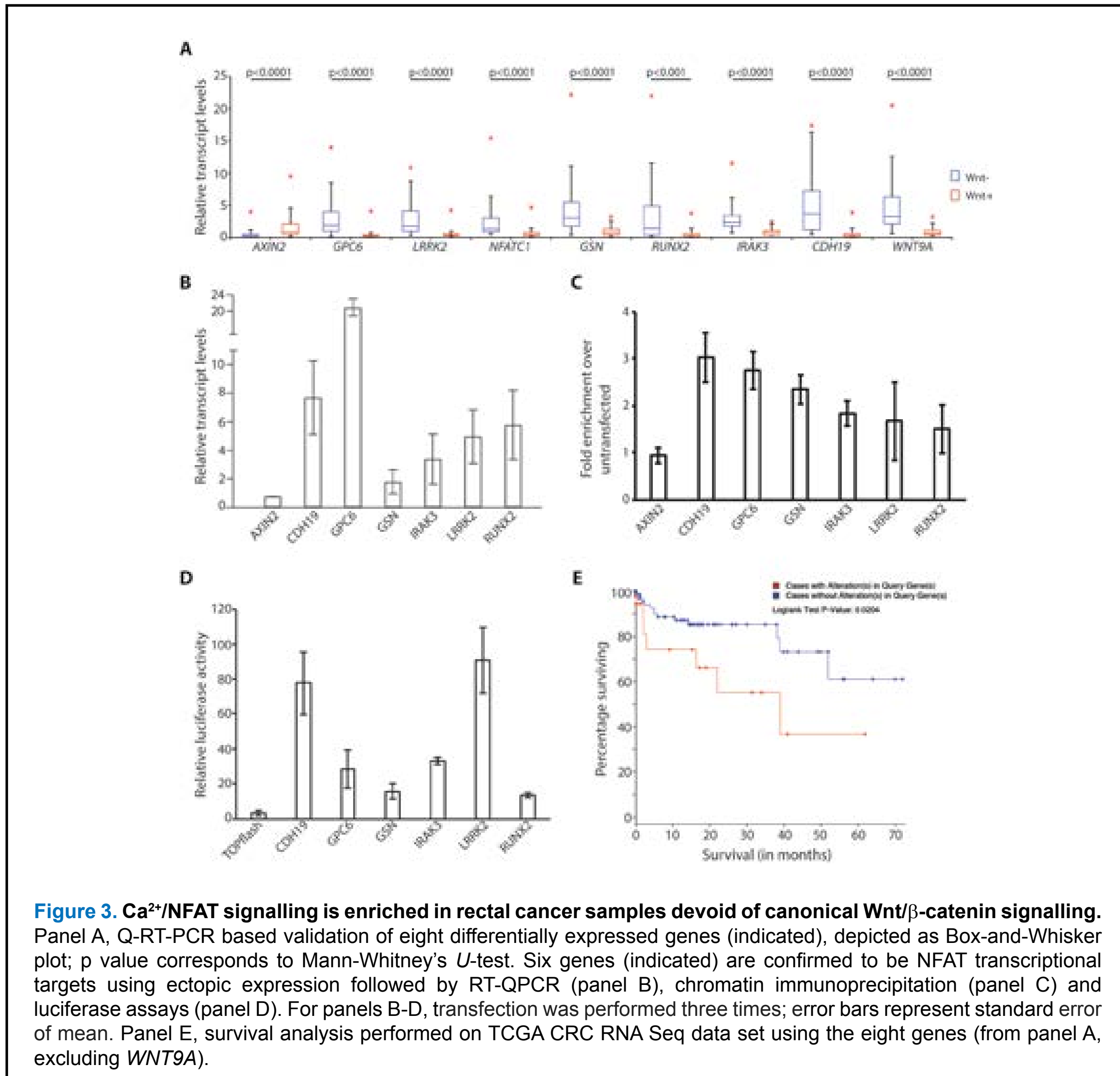

Figure 3. **Ca^{2+}/NFAT signalling is enriched in rectal cancer samples devoid of canonical Wnt/β-catenin signalling.** Panel A, Q-RT-PCR based validation of eight differentially expressed genes (indicated), depicted as Box-and-Whisker plot; p value corresponds to Mann-Whitney's *U*-test. Six genes (indicated) are confirmed to be NFAT transcriptional targets using ectopic expression followed by RT-QPCR (panel B), chromatin immunoprecipitation (panel C) and luciferase assays (panel D). For panels B-D, transfection was performed three times; error bars represent standard error of mean. Panel E, survival analysis performed on TCGA CRC RNA Seq data set using the eight genes (from panel A, excluding *WNT9A*).

Thus, Ca^{2+}/NFAT target genes appear to be activated in rectal cancer in the absence of canonical Wnt signalling. The transcriptome screen also revealed *XPNPEP3* as a novel putative transcriptional target of canonical Wnt/β-catenin signalling which was validated using RT-QPCR in tumor samples (Figure 4A). Induction of *XPNPEP3* upon activation of canonical Wnt/β-catenin signalling was further confirmed in three separate cell lines using RT-QPCR (Figure 4B) and luciferase assays (Figure 4C).

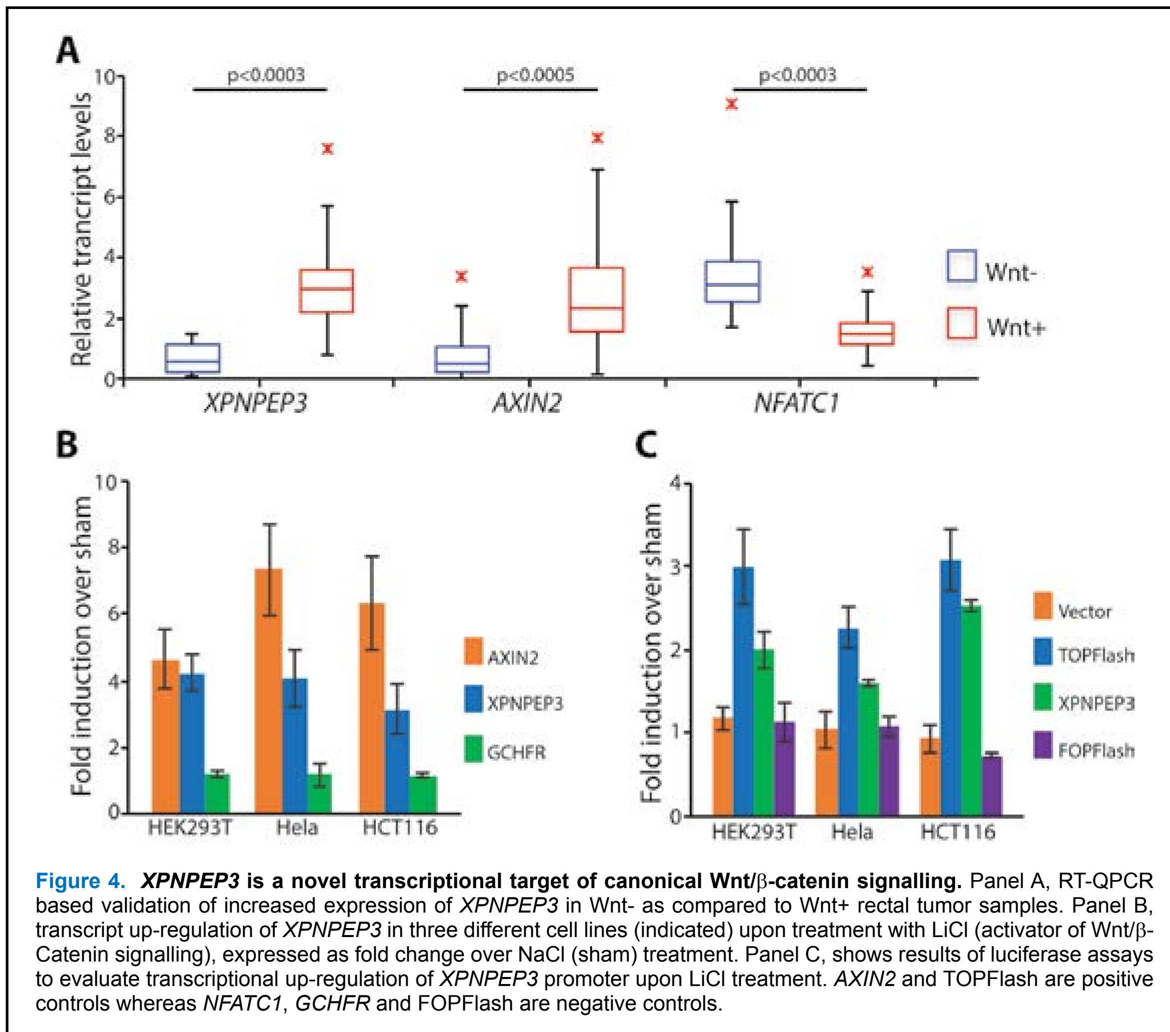

Figure 4. *XPNPEP3* is a novel transcriptional target of canonical Wnt/β-catenin signalling. Panel A, RT-QPCR based validation of increased expression of *XPNPEP3* in Wnt- as compared to Wnt+ rectal tumor samples. Panel B, transcript up-regulation of *XPNPEP3* in three different cell lines (indicated) upon treatment with LiCl (activator of Wnt/β-Catenin signalling), expressed as fold change over NaCl (sham) treatment. Panel C, shows results of luciferase assays to evaluate transcriptional up-regulation of *XPNPEP3* promoter upon LiCl treatment. *AXIN2* and TOPFlash are positive controls whereas *NFATC1*, *GCHFR* and FOPFlash are negative controls.

Future plans and directions

1. Characterization of novel transcriptional targets of mutant p53.
2. Characterization of Ca^{2+}/NFAT signalling pathway driving Wnt- rectal cancer.

Publications

1. Chaudhary AK, Sankar VH and Bashyam MD (2016). A novel large deletion that encompasses *EDA* and the downstream gene *AWAT2* causes X-linked hypohidrotic/ anhidrotic ectodermal dysplasia. ***Journal of Dermatological Science*** 84:105-107.
2. Chaudhary AK, Girisha K and Bashyam MD (2016). A novel EDARADD 5’-splice site mutation resulting in activation of two alternate cryptic 5’-splice sites causes autosomal recessive Hypohidrotic Ectodermal Dysplasia. ***American Journal of Medical Genetics-A*** 170:1639-1641.
3. Chaudhary AK, Mohapatra R, Nagarajaram HA, Ranganath P, Dalal A, Dutta A, Danda S, Girisha KM and Bashyam MD (2017). The novel EDAR p.L397H missense mutation causes autosomal dominant hypohidrotic ectodermal dysplasia. ***Journal of the European Academy of Dermatology and Venereology*** 31:e17-e20.

LABORATORY OF NEUROSPORA GENETICS

Novel findings on meiotic silencing by unpaired DNA, and on ascospore partitioning in Neurospora.

Faculty	Durgadas P. Kasbekar	Haldane Chair
PhD student	Dev Ashish Giri	SRF
Other Members	A. Sheeba	Technical Officer
	S. Rekha	Technical Assistant
	K. Sreethi Reddy	Technical Assistant

Project 1: Meiotic silencing by unpaired DNA (MSUD) is atypically robust in the *Neurospora crassa* Oak Ridge (OR) genetic background.

Objective: To understand why MSUD is stronger in *tester^{OR}* x OR than *tester^{OR}* x wild-strain crosses. In *Neurospora*, allelic sequences misaligned ("unpaired") in meiosis get silenced via an RNAi-mediated process called meiotic silencing by unpaired DNA (MSUD). The unpaired sequences are transcribed into 'aberrant RNA' that is made double-stranded and then processed into single-stranded MSUD-associated small interfering RNA (masiRNA) for use by a silencing complex to degrade complementary mRNA. The MSUD tester strains *::act*, *::asm-1*, *::Bml^{r}*, *::mei-3*, and *::r^{+}* contain an additional copy of the *act* (*actin*), *asm-1^{+}* (*ascospore maturation-1*), *Bml* (*β-tubulin*), *mei-3*, or *r^{+}* (*round ascospores*) gene inserted at an ectopic location. In *tester*-heterozygous crosses the unpaired ectopic copy instigates the production of masiRNA to silence its complementary mRNA, and the resulting deficit of actin, ASM-1, β-tubulin, MEI-3, or R protein results in striking ascus or ascospore phenotypes. In contrast, MSUD does not occur in homozygous *tester A* x *tester a* crosses, and ascus and ascospore development is normal. Most genetic studies in *Neurospora crassa* have used strains of the Oak Ridge (OR) genetic background, and *tester^{OR}* x OR crosses in which the *tester^{OR}* is OR-derived were used to study MSUD. Unexpectedly, MSUD was not always as robust when the *tester^{OR}* strains were crossed with wild-isolated *N. crassa* strains. One hypothesis (model 1) to explain this difference is that sequence heterozygosity between the *tester^{OR}* and wild strain genomes might cause a natural asynapsis and a consequent self-silencing of one or more "MSUD gene". An alternative hypothesis (model 2) is that natural populations harbor wide genetic variation in MSUD strength and that the OR strains represent the MSUD-conducive extreme. If the latter were the case, then the use of OR strains for genetic studies fortuitously facilitated MSUD discovery. Our results obtained in the past year support model 2.

Summary of work done until the beginning of the reporting year (upto March 31, 2016). of 80 wild-isolated strains tested in crosses with the *::Bml^{r}* and *::mei-3* testers, only eight, designated as the "OR" type wild strains, showed silencing phenotypes comparable to those in the corresponding *tester^{OR}* x OR crosses. Crosses with four wild strains designated as the "Sad" type failed to silence *bml* and *mei-3^{+}*, and the remaining 68 strains showed an intermediate phenotype, in that, their crosses silenced *bml* but not *mei-3^{+}*, and they were designated the "Esm" type. Deletion alleles of genes encoding MSUD proteins often act as dominant suppressors of MSUD, presumably because they cause the wild-type homologue to become unpaired, trigger its autogenous silencing, and thereby decrease the encoded protein's level to below the threshold required for MSUD in other loci. The *sad-1*Δ and *sad-2*Δ deletions (i.e. *Sad-1* and *Sad-2* (*Suppressor of ascus dominance-1* and *-2*) are strong dominant suppressors whereas the other gene deletions were less effective, possibly because of their high expression or long protein half-life. *Sad-1* and *Sad-2* also suppressed the barren phenotype of duplication-heterozygous crosses (i.e., *Dp* x *N*). *Dp(EB4)* and *Dp(IBj5)* strains contain duplicated segments bearing, respectively, 35 and 115 genes, and their crosses with the OR type wild strains were barren, with the Sad type were fertile, and with the Esm type, respectively, fertile and barren.

We used two Sad type wild-isolated strains, Bichpuri-1 *a* (B) and Spurger *A* (S), to construct a novel pair of isogenic *mat A* and *mat a* strains. New MSUD testers were made in this B/S background (*tester$^{B/S}$*), and close to isogenic

tester$^{B/S}$ x B/S crosses were tested for MSUD. The f1 progeny from a B x S cross were used to make four f1 *a* x f1 *A* sib-pair crosses, and thereby initiate the formation of recombinant inbred lines. Within a line, in each generation sibling progeny of opposite mating type were crossed to produce the next generation (i.e., sibling f1 *a* x f1 *A* to produce the f2, then sibling f2 *a* x f2 *A* to produce the f3, etc). We were able to reach the f10 generation in two lines. Since in each successive generation the residual heterozygosity is halved, crosses between sibling f10 strains of a line would be < 1% heterozygous. The *mat A* and *mat a* strains of the f10 generation of B/S line 1, referred to henceforth as B/S1 *A* and B/S1 *a*, were used in the subsequent studies.

We employed RIP-mutagenesis to induce a *mus-51* mutant in the B/S1 background. Strains mutant in *mus-51* are defective for non-homologous end joining, consequently, any transforming DNA can integrate only via homologous recombination. A DNA construct bearing a 1683 bp *mus-51* segment and the hygromycin-resistance (*hph*) cassette was transformed by electroporation into B/S1 *A* conidia, and ectopic integration of the transforming DNA created the *Dp(mus-51)* transgenic duplication. The *Dp(mus-51)* primary transformant was then crossed to B/S1 *a*, and the progeny were used to make a *Dp(mus-51)*-homozygous cross. Of 40 progeny examined from the late harvested ascospores, one was found to contain several RIP-induced mutations, including in-frame stop codons (Genbank accession number KM025239), in the endogenous *mus-51* gene and from it we derived the B/S1 *mus-51* *A* and *a* strains, whose transformation would produce the *tester*$^{B/S1}$ strains (below).

Progress made in the current reporting year (April 1, 2016 - March 31, 2017)

The r^{+} gene on chromosome 1 is 3.3 kb long. A 2.3 kb fragment (r^{ef}) from its 3' end was joined to the *hph* cassette by double-joint PCR to create a 4.1 kb r^{ef}-*hph* fusion construct that also included flanking sequences to enable homologous recombination for its precise insertion into the sites used by Tom Hammond and colleagues to construct the $::r1^{OR}$ and $::r3^{OR}$ testers (Samarajeewa *et al*., Genetics, 2014). The DNA construct was transformed by electroporation into B/S1 *mus-51* conidia and transformants were selected for on hygromycin-medium. Since the transforming DNA can integrate only via homologous recombination the insertions obtained were exactly analogous to those in the $::r1^{OR}$ and $::r3^{OR}$ testers. Since the primary transformants were potentially heterokaryotic, they were crossed with B/S1 *a* to segregate out the *mus-51* mutation and homokaryotic $::r1^{B/S1}$ *A* and $::r3^{B/S1}$ *a* tester strains were obtained.

The $::r1^{OR}$ *A* x OR *a* and $::r3^{OR}$ *A* x OR *a* crosses produced > 95% round ascospores, whereas the $::r1^{B/S1}$ *A* x BS1 *a* and $::r3^{B/S1}$ *A* x BS1 *a* crosses produced < 60% round ascospores, and reassuringly, the $::r3^{B/S1}$ *A* x $::r3^{OR}$ *a* crosses produced < 5% round ascospores. These results supported model 2 and allow us to reject model 1. Interestingly, the round ascospores were found to be dispersed significantly less efficiently than their wild-type "American football" shaped counterparts. A manuscript describing these findings is under preparation.

Project 2: Evidence for the occasional uncoupling of ascospore partitioning from post-meiotic mitosis.

Objective: To understand the significance of the rare eight-spored asci found bearing heterokaryotic ascospores.

The partitioning of ascospores in *Neurospora* occurs at the eight-nucleus stage that follows meiosis and the post-meiotic mitosis. Consequently, the ascospores in eight-spored asci are usually homokaryotic (i.e., contain initially a single nucleus from which all the nuclei of the mycelium derived from the ascospore are mitotically descended). By introgressing *N. crassa* insertional translocations into *N. tetrasperma* we had created T^{Nt} strains. Although crosses of the T^{Nt} strains with opposite mating type derivatives of the standard *N. tetrasperma* strain 85 (viz., T^{Nt}*a* x 85*A* or T^{Nt}*A* x 85*a*) produced mostly four-spored asci bearing heterokaryotic [*mat A* + *mat a*] ascospores, as is normal in this species, a few rare eight-spored asci also were produced, and to our surprise a subset of ascospores in the eight-spored asci was found to be heterokaryotic. Eight-spored asci with heterokaryotic ascospores were never previously reported from any Neurospora species therefore we wanted to understand the significance of this finding.

Summary of work done until the beginning of the reporting year (upto March 31, 2016): Introgression is the transfer of genes or genomic regions from one species into another via hybridization and back-crosses. By introgressing

N. crassa insertional and quasiterminal translocations into *N. tetrasperma* we generated hybrid translocation strains (designated as T^{Nt}) whose genome was nominally from *N. tetrasperma*, except at the *N. crassa*-derived translocation breakpoint junctions. In *T* x *N* crosses (*T* = translocation, *N* = normal sequence strain), the chromosomes can segregate either by alternate (ALT) or adjacent-1 (ADJ) segregation. In an *N. crassa T* x *N* cross, ALT produces eight viable parental-type progeny (i.e., 4*T* + 4*N*), and for insertional and quasiterminal translocations (but not for reciprocal translocations), ADJ produces four progeny with a viable duplication and four with its complementary inviable deficiency (i.e., 4*Dp* + 4*Df*). Since ALT and ADJ are equally likely, *T* x *N* crosses yield equal numbers of viable homokaryotic *T*, *N*, and *Dp* progeny. In an *N. tetrasperma* T^{Nt} x *N* cross, ALT produces four viable heterokaryotic [T^{Nt} + *N*] ascospores, whereas ADJ produces four viable heterokaryotic [*Dp* + *Df*] ascospores. Significantly, [*Dp* + *Df*] type heterokaryons were never previously made in any species. [*Dp* + *Df*] and [*T* + *N*] heterokaryons share the same genes and hence should have the same phenotype. Any difference in phenotype would flag the absence of one or more 'nucleus-limited' gene from the *Df* nuclei. A nucleus-limited gene is one for which nuclei bearing its deletion allele (Δ) fail to be complemented by the wild type nuclei (*WT*) in a [*WT* +Δ] heterokaryon. No nucleus-limited genes have yet been reported in the literature, but the phenotype of some fungal mutants suggests that they may be caused by mutations in such genes. Additionally, the T^{Nt} x *N* crosses produced rare eight-spored asci, and a subset of their ascospores was found to be heterokaryotic. Obtaining heterokaryotic ascospores from eight-spored asci is incommensurate with the supposition that ascospore partitioning occurs strictly at the eight-nucleus stage.

Progress made in the current reporting year (April 1, 2016 - March 31, 2017)

We crossed the T^{Nt} strains with opposite mating type derivatives of *N. tetrasperma* strain 85, and harvested the progeny ascospores on water agar as well-separated clumps of 4-8 ascospores, each clump representing an individual ascus. Although a majority of asci were four-spored, we also obtained decreasing fractions of five-, six-, seven-, and eight-spored asci, and the eight-spored asci were 1-2% of the total. The T^{Nt} x 85 crosses behaved largely like crosses in the *N. tetrasperma* strain 85 genetic background, although in 85 *A* x 85 *a* the frequency of non-4-spored asci is typically < 3%. Ascospores from the eight-spored asci were carefully picked to sterile water, germinated, and genomic DNA from the resulting mycelia was used for genotype determination by PCR. Ordinarily, eight-spored asci are expected to yield *T*, *N*, or *Dp* homokaryotic progeny. While this expectation was fulfilled by a subset of the progeny tested, a number of progeny had genotypes that were inconsistent with the expectation. Indeed, some were found to be [*T* + *N*] or [*Dp* + *Df*] heterokaryons whose constituent nuclei had both mating types.

We suggest that in a small subset (~1-2%) of asci one or more nucleus from the post-meiotic mitosis undergoes an additional mitosis and forms supernumerary nuclei whose partitioning leads to formation of heterokaryotic ascospores. In some heterokaryotic ascospores the different nuclear types were of the same mating type. This can happen if crossover occurs proximal to a translocation breakpoint, and the *mat* locus undergoes first-division segregation whereas the breakpoint undergoes second-division segregation (Figure 1). Ascospores receiving a pair of "first-cousin" nuclei can be homoallelic for first-division segregation markers and heteroallelic for second-division segregation markers, whereas those receiving a pair of "second-cousin" nuclei can be homoallelic for second-division segregation markers and heteroallelic for first-division segregation markers (Figure 1). Our findings probably reflect the background level of uncoupling between ascospore partitioning and the post-meiotic mitosis.

Why was such uncoupling not previously detected? It is possible that most normally developing asci in the T^{Nt} x 85 crosses are four-spored, whereas the dysgenic ones are enriched among the eight-spored asci. In *N. crassa* the *tol* (*tolerant*) gene on chromosome 4R would render any mating type heterokaryon unstable, but if the normal tol^C allele is replaced by the recessive mutant allele *tol*, then the [*tol mat A* + *tol mat a*] heterokaryons are stable provided that they are also homokaryotic for the other *het* incomptatibility loci. The *N. tetrasperma* tol^T allele resembles the *N. crassa* mutant *tol* allele. Further, *N. crassa* heterokaryons homoallelic for mating type are difficult to distinguish from a

homokaryon, since the only difference between the two genotypes is that the heterokaryon is heteroallelic for markers that underwent second-division segregation (Figure 1). In *N. tetrasperma*, any heterokaryotic ascospores from eight-spored asci would be vastly outnumbered by heterokaryons from the four- to seven-spored asci, and rare heterokaryons that are homoallelic for mating type would be difficult to distinguish from the significant number of homokaryons from five- to seven-spored asci. Our findings were published in J. Biosci. (2017a). These results also allowed us to account for an exceptional strain of unexpected phenotype (the DA phenotype) reported by D. D. Perkins (Genetics, 1972) that for want of an explanation were attributed to technical error. Our explanation was published in J. Biosci. (2017b).

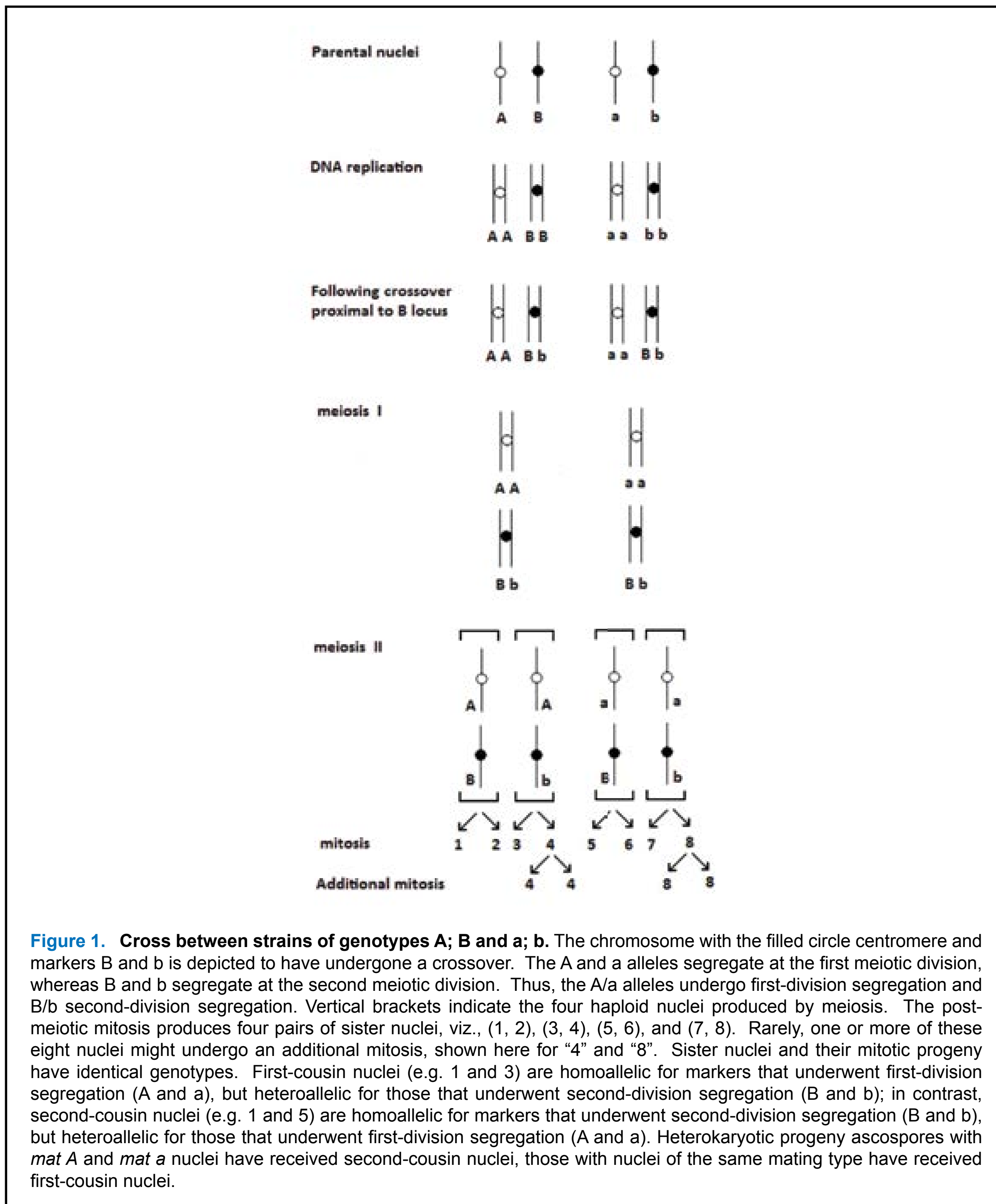

Figure 1. Cross between strains of genotypes A; B and a; b. The chromosome with the filled circle centromere and markers B and b is depicted to have undergone a crossover. The A and a alleles segregate at the first meiotic division, whereas B and b segregate at the second meiotic division. Thus, the A/a alleles undergo first-division segregation and B/b second-division segregation. Vertical brackets indicate the four haploid nuclei produced by meiosis. The post-meiotic mitosis produces four pairs of sister nuclei, viz., (1, 2), (3, 4), (5, 6), and (7, 8). Rarely, one or more of these eight nuclei might undergo an additional mitosis, shown here for “4” and “8”. Sister nuclei and their mitotic progeny have identical genotypes. First-cousin nuclei (e.g. 1 and 3) are homoallelic for markers that underwent first-division segregation (A and a), but heteroallelic for those that underwent second-division segregation (B and b); in contrast, second-cousin nuclei (e.g. 1 and 5) are homoallelic for markers that underwent second-division segregation (B and b), but heteroallelic for those that underwent first-division segregation (A and a). Heterokaryotic progeny ascospores with *mat A* and *mat a* nuclei have received second-cousin nuclei, those with nuclei of the same mating type have received first-cousin nuclei.

Publications

1. Giri DA, Rekha S, and Kasbekar DP. (2016) Crosses heterozygous for hybrid Neurospora translocation strains show transmission ratio distortion disfavoring homokaryotic ascospores made following alternate segregation. ***G3: Genes Genomes Genetics*** 6: 2593-2600.
2. Kasbekar DP and Rekha S (2017a) *Neurospora tetrasperma* crosses heterozygous for hybrid translocation strains produce rare eight-spored asci bearing heterokaryotic ascospores. ***Journal of Biosciences*** 42: 15-21.

Other Publications.

1. Kasbekar DP (2016) History and Development of Genetics Research in India: Three case studies. ***Indian Journal of History of Science*** 51.2.2: 423-430.
2. Kasbekar DP (2016) Obaid Siddiqi's study of the *PABA1* gene of the fungus *Aspergillus nidulans*. ***Biographical Memoirs of Fellows of the Indian National Science Academy Special*** 42: 16-24.
3. Kasbekar DP (2016) RNA-Seq, and ye shall find: Sexual-stage-specific A-to-I RNA editing in fungi. ***Journal of Biosciences*** 41: 171–172.
4. Kasbekar DP (2016) Neurospora deficiencies: The long and short of it. ***Cell Biology Newsletter*** 35: 1-6.
5. Kasbekar DP (2017b) Sherlock Holmes, David Perkins, and the missing Neurospora inversions. ***Journal of Biosciences*** 42: 5-10.

LABORATORY OF PLANT-MICROBE INTERACTIONS

Understanding virulence mechanisms of *Xanthomonas* plant pathogens and interaction with host plants

Faculty	Subhadeep Chatterjee	Staff Scientist
PhD Students	Sheo Shankar Pandey	Senior Research Fellow
	Akanksha Kakkar	Senior Research Fellow
	Raj Kumar Verma	Senior Research Fellow
	Biswajit Samal	Senior Research Fellow
	Prashantee Singh	Senior Research Fellow
	Yasobanta Padhi	Junior Research Fellow
Other Members	Binod Bihari Pradhan	Technical officer
	Krishnamurty	Tradesman

Objectives

1. Identification and characterization of virulence factors of *Xanthomonas*
2. Role of cell-cell communication in *Xanthomonas* colonization and virulence
3. Function of protein secretion system in *Xanthomonas* and role in virulence
4. Role of PAMP in pathogen recognition and plant defense response

Summary of work done until the beginning of this reporting year (April 1, 2015 – March 31, 2016)

Cell–cell communication mediated by diffusible signal factor (DSF) plays an important role in virulence of several *Xanthomonas* group of plant pathogens. In the bacterial pathogen of rice, *Xanthomonas oryzae* pv. *oryzicola*, DSF is required for virulence and *in planta* growth. Our results also indicate that requirement of iron uptake strategies to utilize either Fe^{3+} or Fe^{2+} form of iron for colonization may vary substantially among closely related members of the *Xanthomonas* group of plant pathogens. Apart from iron, we have identified novel role of DSF in regulating Type III secretion system which is required for pathogenicity of *Xanthomonas*. DSF deficient *rpfF* mutant are exhibit reduced Hypersensitive Response (HR) and reduced expression of Type III secretion components and effectors. In future, we want to study the mechanism of DSF sensing which controls iron uptake and regulatory mechanisms, which are involved in DSF regulated traits such as Type III secretion, attachment and biofilm formation. We have shown that Xanthomonas group of phytopathogens produce xanthoferrin, the α-hydroxy carboxylate type siderophore. Our study reveals that the siderophore xanthoferrin is an important virulence factor of *X. campestris* pv. *campestris* which promote *in planta* growth by sequestering ferric iron. We have shown that bacteria exhibit reversible non gebnetic heterogeneity in QS. We have proposed a model based on our studies and evolutionary theory, which predicts that maintaining phenotypic heterogeneity in performing social tasks is advantageous as it can serve as a bet-hedging survival strategy. Our results have shown that bacteria maintain stochastic reversible phenotypic heterogeneity during a widely conserved QS-response that is involved in coordinating multiple social behaviors. We are now addressing the role of cell-cell signaling in adaptation to stationary phase and role of heterogeneity in bet-hedging.

Details of the progress made in the current reporting year (April 1, 2016 – March 31, 2017)

Project 1: Role of cell-cell signaling and cyclic Di-GMP in coordination of virulence associated functions in *Xanthomonas*.

Bacteria integrate extracellular cell-cell signalling or quorum sensing with intracellular signalling mediated by c-di-GMP to co-coordinately regulate diverse cellular processes. Although quorum sensing and c-di-GMP regulate diverse functions including motility, biofilm formation and production of virulence associated functions, their interplay and functional diversification of c-di-GMP turnover effectors in regulation of diverse functions remains undefined. In phytopathogen *Xanthomonas oryzae*, quorum

sensing is mediated by diffusible signal factor (DSF), a fatty acid like signalling molecule which is involved in the regulation of several virulence associated functions including modulation of c-di-GMP effectors. However, it is still unclear how the c-di-GMP network regulates these traits. In an attempt to delineate the entire range of c-di-GMP functionality in *Xanthomonas oryzae* we constructed a deletion mutant library of 15 in-frame deletion mutants, targeting genes predicted to be involved in c-di-GMP metabolism (biosynthesis or degradation) to understand the interplay between QS and complex c-di-GMP signalling network (Figure 1).

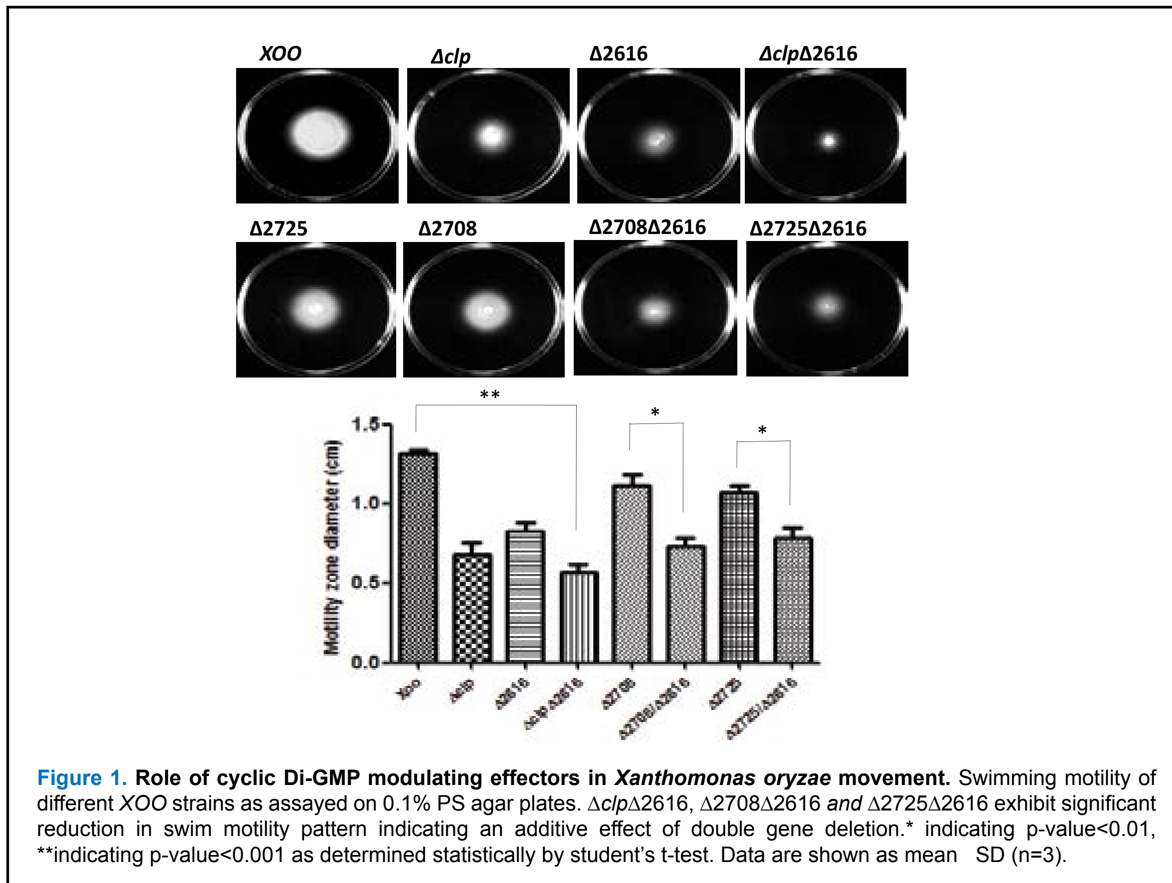

Figure 1. Role of cyclic Di-GMP modulating effectors in *Xanthomonas oryzae* movement. Swimming motility of different *XOO* strains as assayed on 0.1% PS agar plates. Δ*clp*Δ2616, Δ2708Δ2616 *and* Δ2725Δ2616 exhibit significant reduction in swim motility pattern indicating an additive effect of double gene deletion.* indicating p-value<0.01, **indicating p-value<0.001 as determined statistically by student's t-test. Data are shown as mean SD (n=3).

Our results indicate that putative c-di-GMP turnover protein encoding genes, Xoo2563, Xoo2616, Xoo2331 and Xoo2330 are required for optimal swimming motility pattern and biofilm formation. Interestingly, Δ*Xoo2563* and Δ*Xoo2331* also exhibit increased secretion of Type II cell wall hydrolyzing enzymes and siderophore production under iron starvation conditions. Δ*Xoo2563* and Δ*Xoo2725* are significantly deficient in virulence and host colonization, whereas Δ*Xoo2616*, Δ*Xoo2708*, Δ*Xoo2331* and Δ*Xoo2330* are partially reduced disease development. *in vitro* biochemical analysis of their enzymatic activities by HPLC, correlated with the *in vivo* c-di-GMP levels in mutants defective in c-di-GMP turnover. Furthermore, we over expressed the c-di-GMP metabolizing enzymes in wild type *Xoo* to elucidate a direct role of c-di-GMP in virulence and growth inside host. Interestingly, Xoo2563, Xoo2616, Xoo2331 and Xoo2725 gene deletions in the quorum sensing DSF-deficient mutant could rescue the growth defect of Δ*rpfF* under iron starvation condition. Our phenotypic analysis of QS pathway deletion mutants showed that Δ*rpfC*, Δ*rpfG* and Δ*clp* do not phenocopy the growth defect of Δ*rpfF* in the presence of 2,2'-dipyridyl and streptonigrin, indicating a phenotype specific dissection of cell-cell signalling network unlike in *Xcc*. In this study we identified potential candidates that could have a regulatory role in maintenance of optimal c-di-GMP levels in *Xoo* and also coordinate with the DSF signalling system to fine tune this complex network (Figure 2).

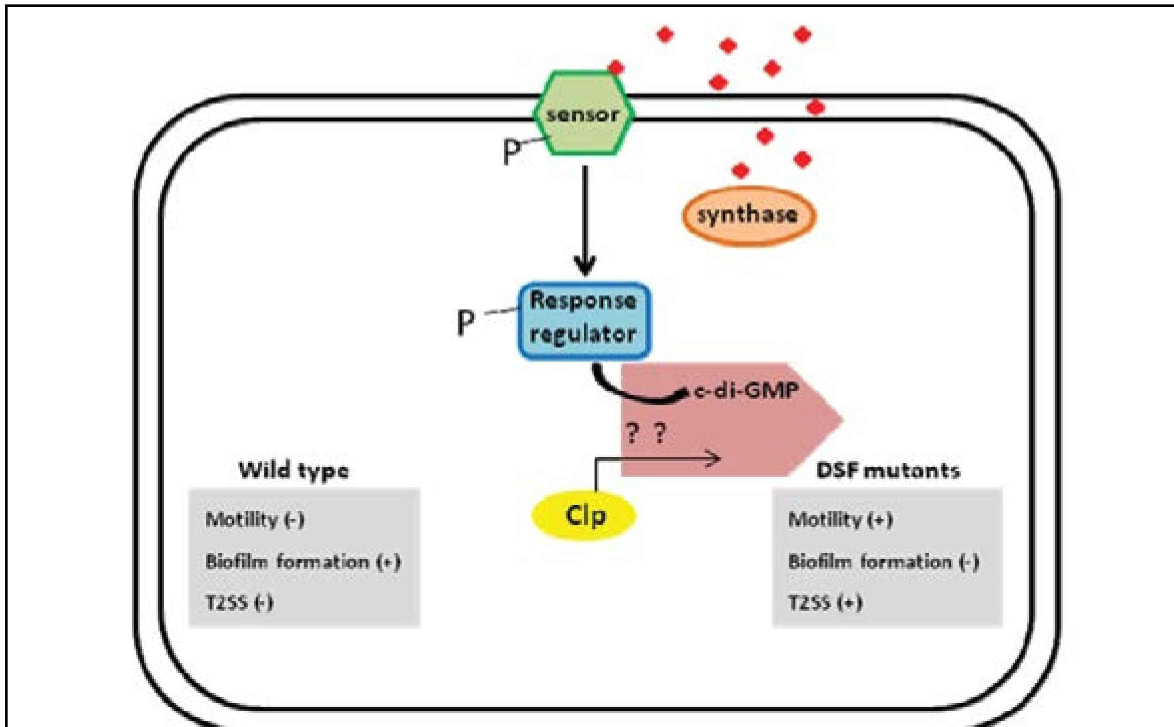

Figure 2. A proposed model for the delineation of the c-di-GMP network in *Xanthomonas oryzae* and its crosstalk with quorum sensing signaling components to co-ordinate production of virulence associated functions. Cell-cell signaling (DSF) sensor RpfC and putative intracellular sensor respond to change in DSF level, which influence the activity of cyclic Di-GMP modulators (GGDEF and EAL domain protein). Cyclic Di-GMP biosynthetic and degradation domain containing protein regulate different virulence associated function such as motility, biofilm formation, epiphytic infection in a contrasting fashion which is influenced by DSF and iron availability.

Project 2: Role of DSF in inducing innate immunity in plants

We have shown that a bacterial fatty acid cell-cell signaling molecule, DSF (diffusible signal factor) elicits innate immunity in plants. The DSF families of signaling molecules are highly conserved among many phytopathogenic bacteria belonging to genus *Xanthomonas* as well as in opportunistic animal pathogens. Using *Arabidopsis*, *Nicotiana benthamiana* and rice as model systems, we show that DSF induces hypersensitivity reaction (HR)-like response, programmed cell death, the accumulation of autofluorescent compounds, hydrogen peroxide production and induced expression of the *PATHOGENESIS-RELATED*1 (*PR-1*) gene. Furthermore, production of the DSF signaling molecule in *Pseudomonas syringae*, a non-DSF producing plant pathogen, induces the innate immune response in *Nicotiana benthamiana* host plant and also affects pathogen growth. By performing pre-and co-inoculation of DSF, we have demonstrated that the DSF induced plant defense reduces disease severity and pathogen growth in the host plant. In this study, we further demonstrate that the wild type *Xanthomonas campestris* suppress the DSF induced innate immunity by secreting xanthan, the main component of extracellular polysaccharide. Our results indicate that plants have evolved to recognize a widely conserved bacterial communication system and may have played a role in the co-evolution of host recognition of the pathogen and the communication machinery. To understand the DSF induction and endogenous DSF level which could affect the plant defense response, we have used DSF based biosensor strains to correlate DSF production level with the induction of defense response. To detect DSF levels produced by the wild type *Xcc* strain in *N. benthamiana* leaves, we infiltrated the wild type *Xcc*8004 (pKLN55) DSF biosensor strain under similar condition at a density of 1 x 10^6 C.F.U/ml. At a low cell density (1 x10^6 C.F.U / ml), the *Xcc* DSF biosensor strain exhibited low GFP fluorescence (uninduced) in PS media (Pradhan and Chatterjee, Mol. Microbiol., 2014). Analysis of *N. benthamiana* leaves by confocal

microscopy indicated that the wild type *Xcc* produced a significant amount of DSF, *in planta*, as indicated by the induced DSF responsive GFP fluorescence after 24 to 48 h post infiltration (Figure 3). These results revealed that the endogenous DSF level fluctuates in planta during *Xanthomonas* –host interaction and the concentration build up inside the plant could sufficiently trigger both early and late defense response.

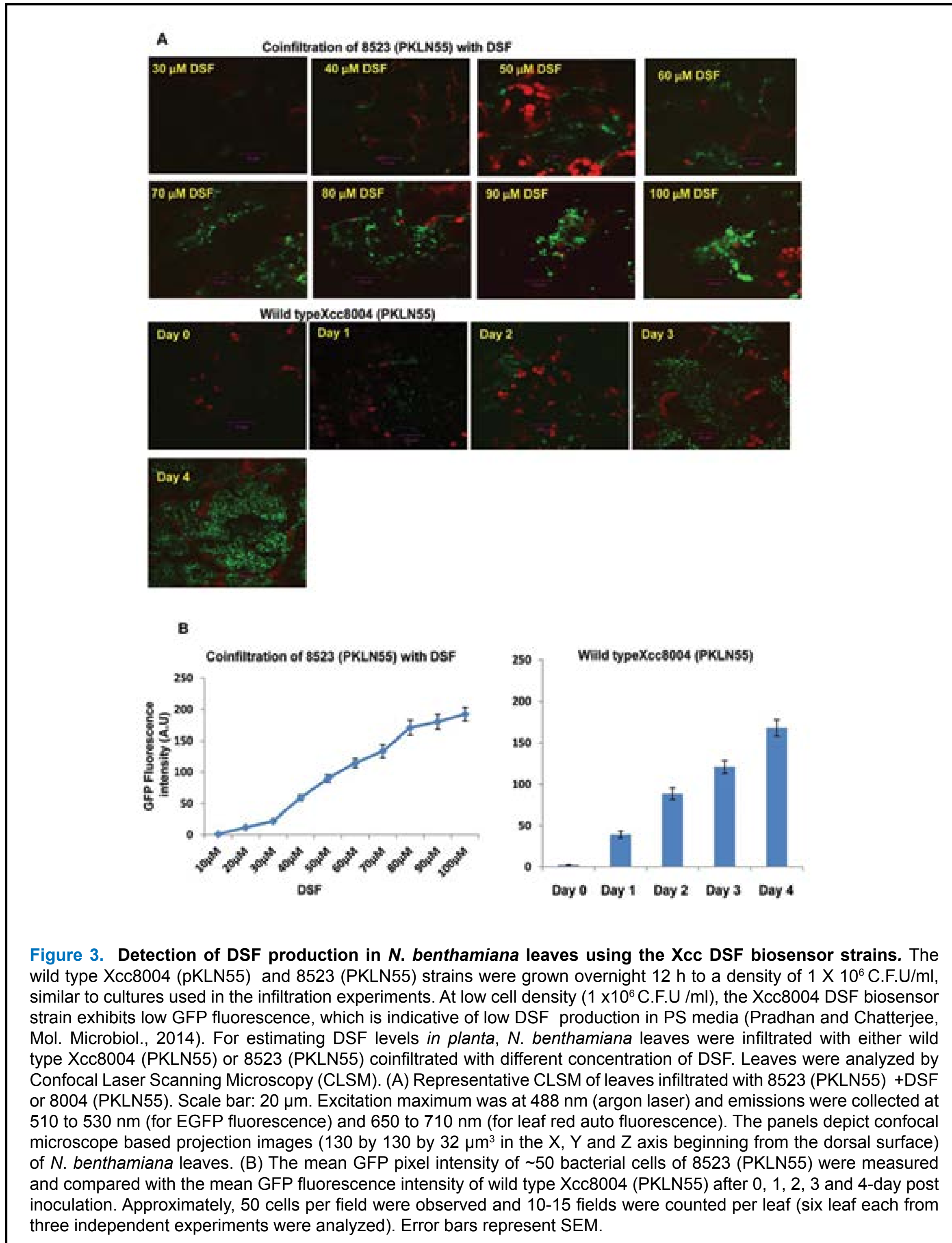

Figure 3. Detection of DSF production in *N. benthamiana* leaves using the Xcc DSF biosensor strains. The wild type Xcc8004 (pKLN55) and 8523 (PKLN55) strains were grown overnight 12 h to a density of 1 X 10^6 C.F.U/ml, similar to cultures used in the infiltration experiments. At low cell density (1 x10^6 C.F.U /ml), the Xcc8004 DSF biosensor strain exhibits low GFP fluorescence, which is indicative of low DSF production in PS media (Pradhan and Chatterjee, Mol. Microbiol., 2014). For estimating DSF levels *in planta*, *N. benthamiana* leaves were infiltrated with either wild type Xcc8004 (PKLN55) or 8523 (PKLN55) coinfiltrated with different concentration of DSF. Leaves were analyzed by Confocal Laser Scanning Microscopy (CLSM). (A) Representative CLSM of leaves infiltrated with 8523 (PKLN55) +DSF or 8004 (PKLN55). Scale bar: 20 µm. Excitation maximum was at 488 nm (argon laser) and emissions were collected at 510 to 530 nm (for EGFP fluorescence) and 650 to 710 nm (for leaf red auto fluorescence). The panels depict confocal microscope based projection images (130 by 130 by 32 μm^3 in the X, Y and Z axis beginning from the dorsal surface) of *N. benthamiana* leaves. (B) The mean GFP pixel intensity of ~50 bacterial cells of 8523 (PKLN55) were measured and compared with the mean GFP fluorescence intensity of wild type Xcc8004 (PKLN55) after 0, 1, 2, 3 and 4-day post inoculation. Approximately, 50 cells per field were observed and 10-15 fields were counted per leaf (six leaf each from three independent experiments were analyzed). Error bars represent SEM.

Publications:

(i) Research papers published in the calendar year 2016:

1. Pandey SS, Patnana PK, Lomada SK, Tomar A, and Chatterjee S (2016) Co-regulation of Iron Metabolism and Virulence Associated Functions by Iron and XibR, a Novel Iron Binding Transcription Factor, in the Plant Pathogen **Xanthomonas**. ***PLoS Pathogens*** 12(11): e1006019. doi:10.1371/journal.ppat.1006019

2. Pandey S.S, Patnana,P.K, Rai S, and Chatterjee S. (2016) Xanthoferrin, the α-hydroxy carboxylate type siderophore of *Xanthomonas campestris* pv. *campestris* is required for optimum virulence and growth inside cabbage. ***Molecular Plant Pathology***. DOI: 10.1111/mpp.12451.

LABORATORY OF TRANSCRIPTION

Mechanism of transcription termination and antitermination in Escherichia coli.

Faculty	Ranjan Sen	Staff Scientist
PhD Students	V. Vishalini	Senior Research fellow (till Feb. 2017)
	Gairika Ghosh	Senior Research fellow
	Richa Gupta	Senior Research fellow (till July 2016)
	Md. Hafeezunnisha	Senior Research fellow
	Passong Immanual	Junior Research Fellow
	Ajay Khatri	Junior Research Fellow
Other Members	Shweta Singh	Post-doctoral Fellow
	Pallavi Maitra	Post-doctoral Fellow
	Sonia Agrawal	Project Assistant (till July 2016)
	Shreyans Jain	Postdoctoral fellow (since May 2016)
	Sushmit Shambhare	Postdoctoral Fellow (since April 2016).
	Sapna Godavarthi	Technical Officer
	Jayvardhan Reddy	Technical Assistant
	Gowresh	Lab Attendant (till Aug. 2016)
Collaborators	Udayaditya Sen	SINP, Kolkata
	V Nagaraja	IISc., Bangalore
	Jayanta Mukhopadhyay	Bose Institute, Kolkata
	Akira Ishihama	Hosei University, Japan

Objectives.

Fundamental questions in the area of mechanism of transcription termination and antitermination processes in bacteria is still not very clear and offers an exciting subject for study. In my laboratory, following studies are underway. 1) Mechanism of action of transcription termination factor, Rho both *in vivo* and *in vitro*. 2) Molecular basis of Rho-NusG interaction. 3) Establishing inhibition of Rho-dependent termination by Rho proteins from different bacteria by the anti-rho factor, Psu.4) In vivo cross-talks between Rho dependent termination and other biological processes. 5) Isolating myco-bacteriocidal factors from the mycobacteriophages using metagenomics approaches.

Summary of the work done until the beginning of this reporting year (April 1, 2015- March 31, 2016).

The bacterial transcription elongation factor NusG stimulates the Rho-dependent transcription termination through a direct interaction with Rho. We showed that NusG imparts conformational changes in the central channel of Rho, yielding faster isomerization of the open to the closed hexameric states of the latter during its RNA-loading step. This acceleration stabilizes the Rho-RNA interactions at many terminators having suboptimal *rut* sites, thus making Rho-NusG interactions so essential in vivo (Vishalini et al., J. Biol.Chem., 2016).

Myco-bacteriophages code numerous protein factors capable of modulating host machineries for their own growth advantages. These are reservoirs of new proteins as well as could be utilized to source novel myco-bacteriocidal factors. In our initial attempts, created a mixed phages genome library using few sequenced phages (Bethlehem, Che9c, Che9d, Che12, D29, L5, I3 and TM4). Colonies those did not grow on in the presence of these factors were screened. Our initial data revealed that gp89 of phage D29, gp79, gp80 of the phage Bethlehem and gp49 and gp50 of the phage Che12 are responsible for lethality. These gene products are unique to mycobacteriophages and their functions are not yet identified.

Details of the progress in the current reporting year (April 1, 2016- March 31, 2017).

A) bacteriophage capsid protein is an inhibitor of a conserved transcription terminator of various bacterial pathogens.

Rho is a homo-hexameric molecular motor protein that functions as a conserved transcription

terminator in majority of the bacterial species. The essentiality of this highly conserved protein makes it a potential target for bactericidal agents. Psu is a unique bacteriophage P4 capsid protein that inhibits *E.coli* Rho by obstructing its ATPase and translocase activity. Here, we explored the anti-Rho activity of Psu for the Rho proteins from different pathogenic bacteria. Multiple sequence alignment and homology modelling of Rho proteins from pathogenic bacteria revealed the conserved nature of the Psu-interacting regions in all the Rho proteins. We chose Rho proteins from various pathogens like, *Mycobacterium smegmatis, Mycobacterium bovis, Mycobacterium tuberculosis, Xanthomonas campestris, Xanthomonas oryzae, Corynebacterium glutamicum, Vibrio cholera, Salmonella enterica and Pseudomonas syringae*, to study the inhibitory prowess of the Psu protein both *in vivo* and *in vitro*. The purified recombinant Rho proteins of these organisms showed variable rate of ATP hydrolysis on the polyC RNA as substrate, but were unable to use *rut site* of *E.coli* Rho. Psu was capable of inhibiting the ATPase activities of all these Rho proteins. Various Rho proteins from pathogens were capable of release RNA from the *E. coli* transcription elongation complexes. Psu could able to inhibit RNA release by these Rho proteins from the stalled elongation complexes. *In vivo* pull down assays revealed direct binding of Psu with these various Rho proteins. *In vivo* expression of *psu* induced growth inhibition of *M. smegmatis*, *M. bovis, X. orizae, and S.entericia,* which is a strong indication of Psu-induced inhibition of Rho proteins of these strains under physiological condition. We propose that the "universal" inhibitory function of the Psu protein for Rho proteins from both the gram negative and gram positive bacteria makes it a potential platform for designing anti-Rho peptides having anti-microbial function. We further speculate that Psu can be a part of synergistic antibiotic treatment by offering bacterial pathogens with compromised Rho functions (Figure 1).

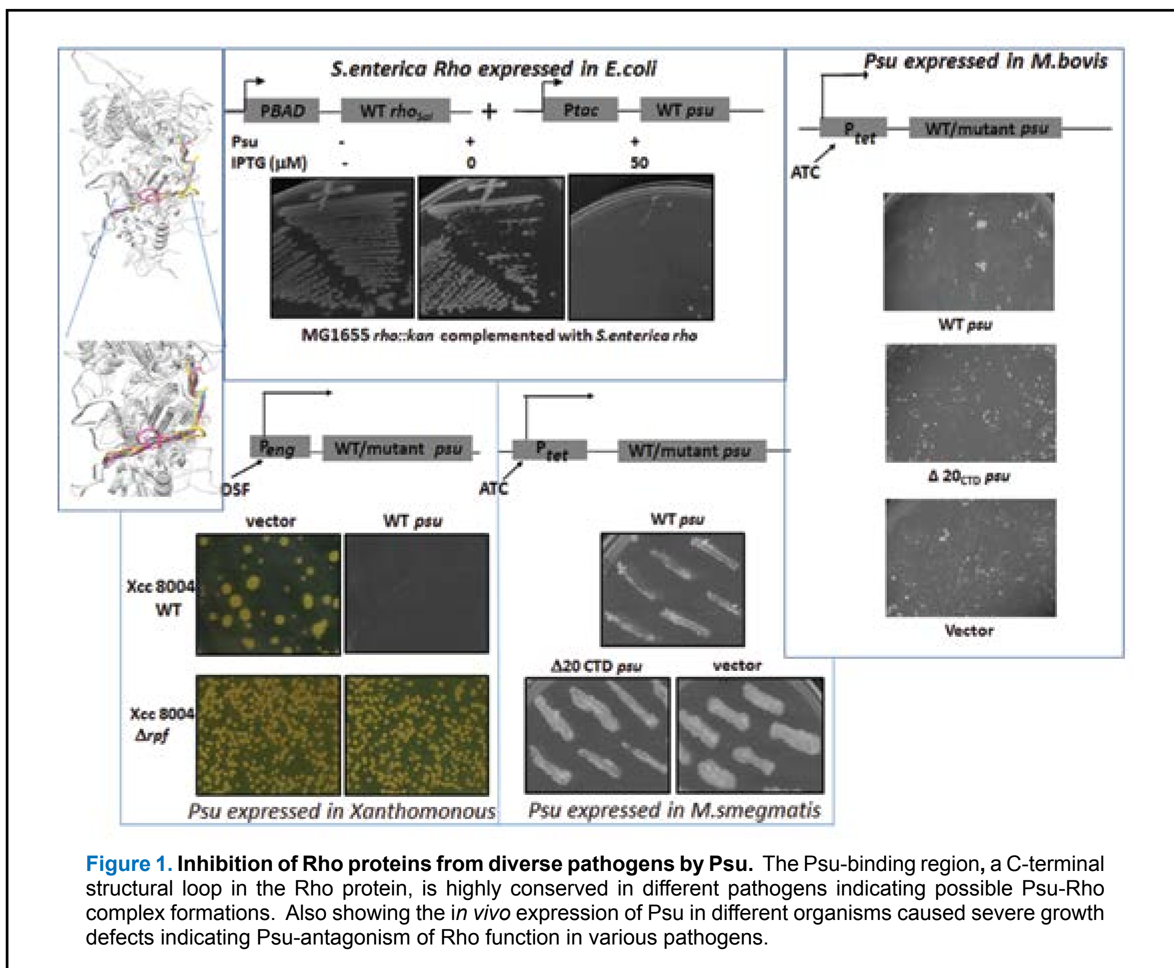

Figure 1. Inhibition of Rho proteins from diverse pathogens by Psu. The Psu-binding region, a C-terminal structural loop in the Rho protein, is highly conserved in different pathogens indicating possible Psu-Rho complex formations. Also showing the i*n vivo* expression of Psu in different organisms caused severe growth defects indicating Psu-antagonism of Rho function in various pathogens.

B) Rho-dependent transcription termination in bacteria is a component of Transcription-coupled DNA repair process.

Stalling of the RNA polymerase (RNAP) at the DNA lesions initiates the transcription-coupled DNA repair (TCR) process. In principle, randomly transcribing RNAPs involved in pervasive transcription could function as a global scanner of different types of DNA lesions. This pervasive transcription is the target of Rho-dependent termination and hence, Rho is likely to be associated with these randomly transcribing elongation complexes. We hypothesized that Rho-induced release of the stalled ECs at the DNA lesion sites could facilitate the TCR repair process by exposing the DNA damage. We have observed that Rho and NusG mutants defective for termination functions caused synthetic lethality in the strains with deletions of uvrA or uvrB or uvrC or mfd that are components of TCR. These mutants exhibited enhanced sensitivity to UV-radiation, mitomycin C and cis-platin treatments that are causative agents for eliciting the TCR process. Deletion of many of the base-excision repair (BER) genes such as, mutM, mutY, mutT etc. was also synthetically lethal with these mutants. These *in vivo* data convincingly connects Rho-dependent termination with the TCR and BER pathways, where the latter may also involve stalling of the EC at the damaged bases on the DNA. In a purified system, like Mfd, Rho was capable of releasing ECs stalled at the T-T dimers with similar efficiency. Similar to Mfd, Rho-dependent termination was also observed to be instrumental in initiating the nicking reactions at the damaged site in the presence of UvrA, UvrB and UvrC. Our data strongly suggest that Rho-dependent termination could be used as an alternative pathway to dislodge stalled RNAPs from the DNA damaged site, and we propose that under non-stressed condition, when level of mfd stays low, bacteria become more dependent on Rho to dislodge the ECs stalled at randomly formed DNA damaged sites (Figure 2).

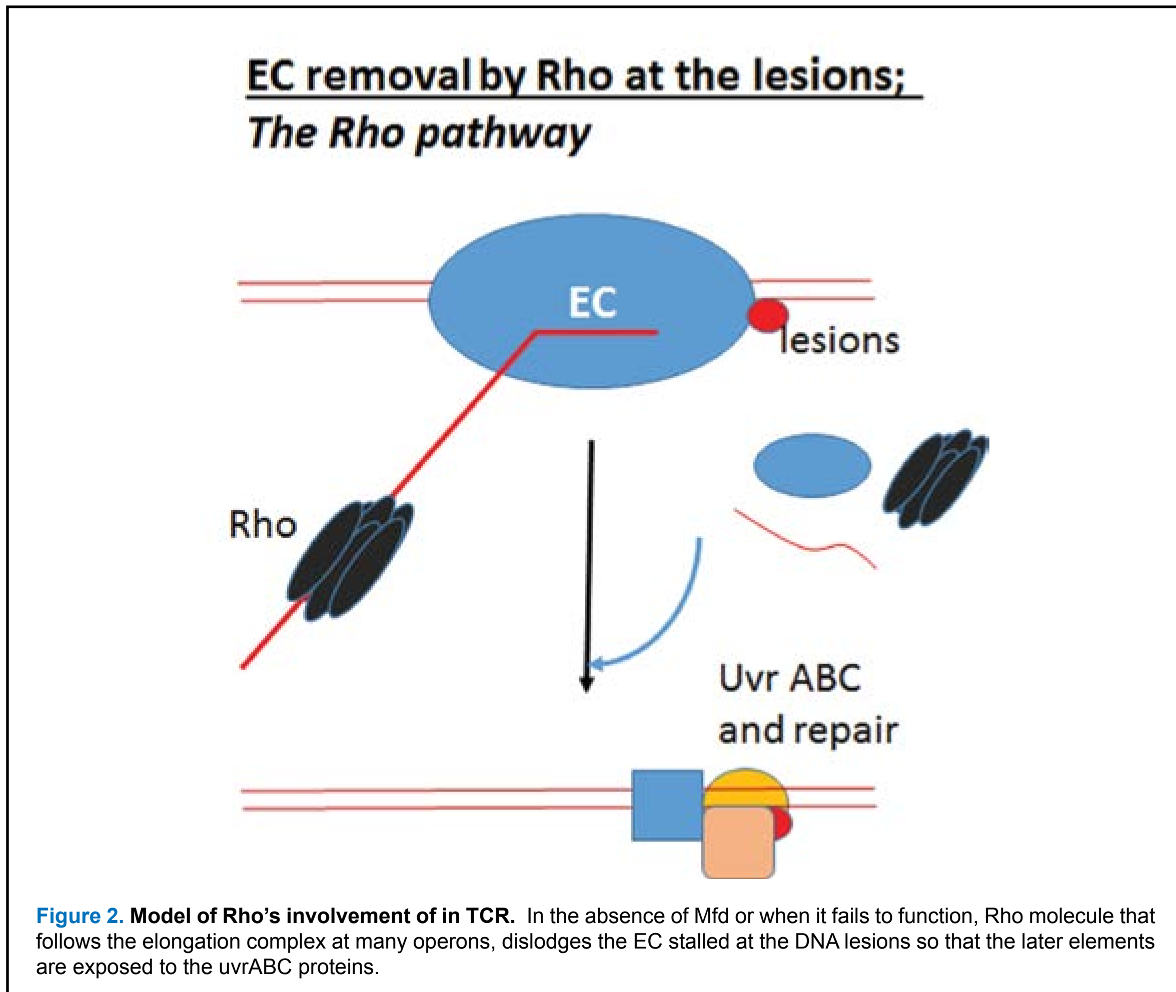

Figure 2. Model of Rho's involvement of in TCR. In the absence of Mfd or when it fails to function, Rho molecule that follows the elongation complex at many operons, dislodges the EC stalled at the DNA lesions so that the later elements are exposed to the uvrABC proteins.

C) Exploring myco-bacteriophages to identify novel myco-bactericidal protein factors

Mycobacteriophages are viruses that infect mycobacterium hosts. To date, thousands of mycobacteriophages have been isolated using a single host strain, *M. smegmatis* mc2155, 1367 of which have been completely sequenced (http://www.phagesdb.org). However, functions of majority of the gene products are not known. Here we investigate mycobacteriophage derived molecules that impair the growth of the mycobacterial host.

In the present study, library of 7 mycobacteriophages (Bethlehem, Che9c, Che9d, Che12, D29, L5, I3 and TM4) from different cluster were made in an inducible shuttle vector. On screening of more than 3000 clones, several cloned fragments from different phages showed either inhibitory or lethal effect, when expressed in mycobacterium. Except for clone 66, bioinformatics analysis of these gene products could not assign any functional domain. Clone 66 (gp49 from phage Che12) was found to carry helix turn helix (HTH) domain implying DNA binding properties.

Using confocal microscopy, we observed that upon expression of these clones in *M. Smegmatis,* vast morphological variations occurred as compared to the control cells. Clones 66, 85, and 1169 showed long filamentous, clones 12N, 122N, and 660 showed bulged structure at one end of the elongated cells and clone 45 showed branch-like outgrowths at different positions along the length of the elongated cell with lots of debris (Figure 1). These phenotypes may indicate impaired cell division as the DAPI staining showed cells were often multinucleoidal as compare to the control cells (Figure 3).

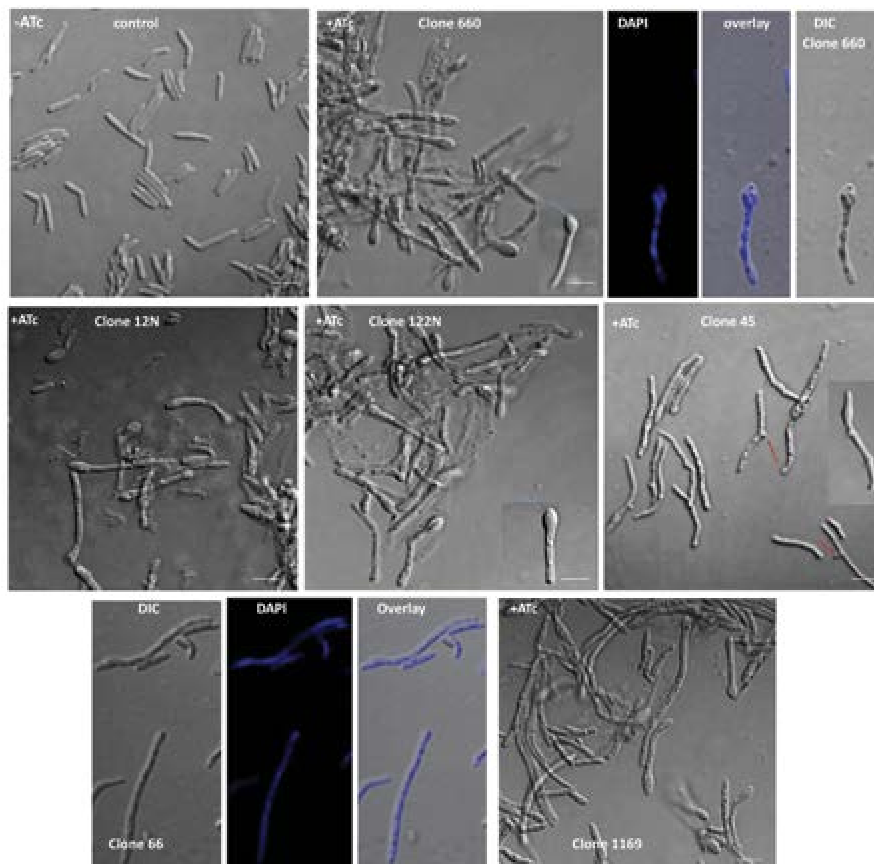

Figure 3. Effect of overexpression of cytotoxic clones on cell morphology of *M. smegmatis*. Expression of some clones induced filamentous morphology (clone 66 and 1169), some showed bulbs/swelling (blue arrow) at one end of the elongated cells (clone 12N, 122N, and 660), and clone 45 showed branch-like outgrowths (red arrow) in different positions along with the length of the elongated cell. These phenotypes indicate impaired cell division. DAPI staining showed cells were often multi-nucleoidal as compare to controls. (Scale bar -2µm).

D) Transcription termination factor Rho regulates antibiotic sensitivity.

Rho-dependent transcription termination is involved in various physiological processes. We observed that Rho mutants exhibit sensitivity to various antibiotics of different classes, indicating that more innate pathways like antibiotic efflux or influx systems and biofilm formation are affected in these mutants. AcrAB-TolC is a major efflux pump in gm- bacteria. WT and mutant Rho strains exhibited synthetic growth defects with tolC, acrA or acrB. This defect was suppressed when strains were grown in minimal media. This indicates Rho mutants are more dependent on TolC and there could be more accumulation of metabolites in the Rho mutant strains. In a separate assays using Biolog plates, we observed that these mutant are capable of utilizing complex nutrients like dipeptides as nitrogen source. Consistent with this observation we also found that *dpp* operon (dipeptide permease) is upregulated in these strains. The ability to assimilate more nutrients by Rho mutants indicate the possibility of existence of high metabolome load in the Rho mutants that keeps the efflux pathways saturated with these metabolites thereby rendering inefficient clearance of antibiotics leading to the broad-spectrum sensitivity. These results strongly suggest that bacterial strains could be made more susceptible to different antibiotics by compromising the Rho-dependent termination pathway (Figure 4).

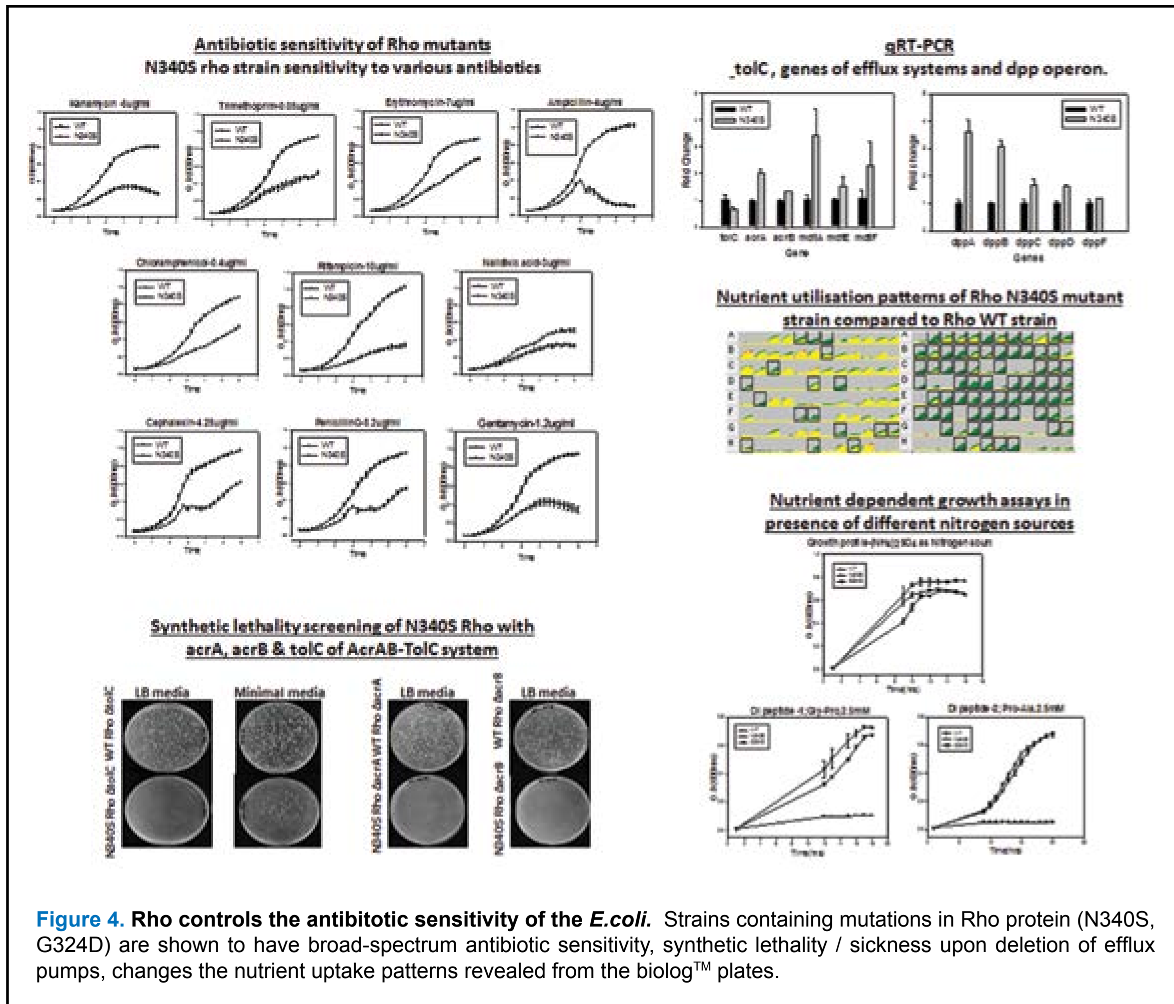

Figure 4. Rho controls the antibitotic sensitivity of the *E.coli*. Strains containing mutations in Rho protein (N340S, G324D) are shown to have broad-spectrum antibiotic sensitivity, synthetic lethality / sickness upon deletion of efflux pumps, changes the nutrient uptake patterns revealed from the biolog™ plates.

Future Plans/directions:

The following projects, being pursued in my lab, are in different stages of completion. 1) Involvement of Rho in transcription coupled repair process, ii) testing efficacy of Psu, as an *E.coli* Rho inhibitor, iv) design of peptide-inhibitors from Psu, iv) characterization of different myco-bacteriocidal factors from mycobacteriophages, v) elucidate the mechanism of control of antibitotic sensitivity by Rho-dependent termination as well as involvement of this process in toxin-antitoxin systems of the E.coli.

Publications.

1. Takada H, Shimada T, Dey D, Quyuum MZ, Nakano N, I Ishiguro A, Yoshida A, Yamamoto K, Sen R and Ishihama A. (2016) Differential regulation of rRNA and tRNA transcription from the rRNA-tRNA composite operon in Escherichia coli. Plos one. Dec 22; 11(12):e0163057.
2. Vishalini V, Agarawal S and Sen R. (2016). Molecular basis of NusG-mediated regulation of Rho-dependent transcription termination in bacteria. Journal of Biological Chemistry. 291, 22386-22403.
3. Qayyum M Z, Dey D and Sen R. (2016). Transcription elongation factor NusA is a negative regulator of Rho-dependent termination. Journal of Biological Chemistry, 291(15), 8090-8108.

In press

Mitra P, Ghosh G, Hafeezunnisa M and Sen R. (2017). Rho protein: mechanism and action. Annual Review of Microbiology, in press.

अन्य वैज्ञानिक सेवाएँ / सुविधाएँ
other scientific services / facilities

LABORATORY ANIMAL FACILITY

Faculty Coordinators	Rashna Bhandari Sanjeev Khosla	Staff Scientist (till June 2016) Staff Scientist (till June 2016)
Research Facility Manager	Raghavendrachar Jois	Staff Scientist (since July 2016)
Other Members	Hole Jayant Pundalikrao	Officer In-Charge (till Aug. 2016) Consultant In-Charge (since Aug. 2016)
	Sridhar Kavela Sravani Edula Navitha Bedarakota	Technical Officer Technical Officer (till Jan. 2017) Laboratory Technician (since Jan. 2017)

Objectives

1. The main objective of the Laboratory Animal Facility (LAF) is to breed, maintain and supply laboratory animals to institutional scientists. Breeding and experimentation of all strains of mice is undertaken in individually ventilated caging systems;
2. To support research programmes that promote the health and well being of people and animals by facilitating high quality and scientifically sound research with animals;
3. To comply with regulatory government body (CPCSEA) requirements for animal experimentation and breeding; and
4. To maintain a stable and contained environment for animals and personnel working in the facility, to ensure consistent animal quality and reduce operational costs.

Summary of work done until the beginning of this reporting year (up to March 31, 2016)

The CDFD LAF started its activities on July 1, 2011, within the premises of M/s Vimta Labs Limited, located at Genome Valley, Shameerpet, Hyderabad. Infrastructure was established to house mice in individually ventilated cages (IVCs), and conduct standard experimental procedures. All procedures conducted on animals housed in this facility are approved by the Institutional Animal Ethics Committee (IAEC) constituted by the Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA), Ministry of Environment, Forest and Climate Change (MoEF & CC), Govt. of India, at M/s Vimta Labs Ltd. Until March 2016, the facility housed approximately 1200 mice of five different strains, and in 2015-16, users were supplied with 891 mice for IAEC approved experimentation.

Details of the progress made in the current reporting year (April 1, 2016 - March 31, 2017)

During this reporting year, the CDFD LAF has housed five inbred mouse strains, including *Ip6k1*, *Nnat*, C57BL/6, *FoxNl^{nu}* and Balb/c. Mice were bred to expand the colonies and meet CDFD users' requirements. Currently this facility has approximately 546 adults and 217 newborn mice housed in 472 IVC cages (Table 1). During the year, 749 mice were supplied to users for IAEC approved experimentation.

Strains	Total (Male+Female)	Under Breeding (Male+Female)	Supplied during 2014-15
Ip6k1	124+96	06+12	35
NnatΔNEO/ΔI^{2}	80+92	06+06	90
Balb/c	46+39	09+18	494
C57BL/6	26+31	06+12	72
Foxn1^{nu}	08+04	08+16	58

Table 1. Strain-wise break up of adult mice housed at LAF as on March 31, 2017, and supplied to users during 2016-17.

Routine IAEC approved procedures conducted on these animals include blood collection for measurement of biochemical parameters, embryo collection for the preparation of embryonic fibroblasts, tail biopsies for genotyping analysis and necropsy for histopathological analysis. Some of the experiments conducted during 2016-17 are highlighted below:

- 151 Balb/c mice were injected with the non-pathogenic mycobacteria, *M. smegmatis,* expressing some candidate *Mtb* proteins, to study the *in vivo* immunomodulatory role of these proteins.
- 150 Balb/c mice were injected intravenously with *Candida glabrata* for studies on the comparative bio-burden of different *Candida* strains.
- 90 *Nnat* mice were used for measurement of biochemical parameters.
- 72 C57BL/6 and 57 Balb/c mice were injected with thioglycolate by intra-peritoneal route for the generation of macrophages.
- 58 *FoxN1*^{nu} athymic mice were injected with oncogenic cell lines to study tumour progression and metastasis.
- 39 Balb/c mice were used to study the effect of *Mycobacterium tuberculosis* protein PPE18 on LPS-induced endotoxaemia.
- 39 Balb/c mice were injected subcutaneously

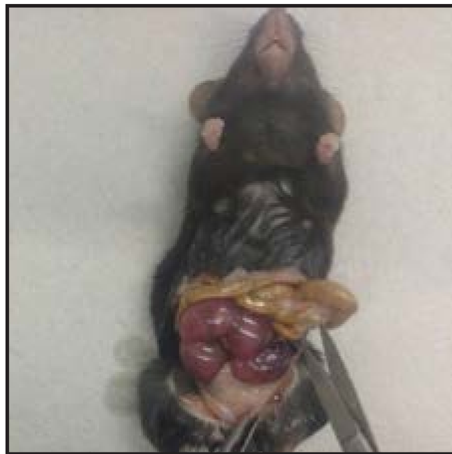

Figure - 1

Figure - 2

Figure - 3

Figure - 4

Figure 1. Collection of 13.5 day old embryos from C57BL/6 mice. **Figure 2**. Surgical procedure for caecal ligation and puncture on a Balb/c mouse. **Figure 3.** Surgical procedure for vasectomy on a Balb/c mouse. **Figure 4**. FoxN1^{nu} athymic nude mice bred successfully at the CDFD Animal Facility.

with protein antigens and polyclonal antibodies were generated successfully.

- 35 *Ip6k1* mice were used for histopathological analysis of testes and the gastrointestinal tract.
- 34 Balb/c mice were used to study the effect of *Mycobacterium tuberculosis* protein PPE18 on caecal ligation and puncture induced sepsis.
- 24 Balb/c mice were used to analyse the vaginal bio-burden of different *Candida glabrata* strains.

The IAEC approved projects in progress during this reporting year are listed in Table 2.

S. No.	Projects in progress
1	Functional analysis of Neuronatin's second intron by knock out strategy
2	Establishment and histopathological characterization of *Ip6k1* knockout mice – version 2
3	Signal transduction pathway in immune cells regulating their innate and effecter functions during oxidative stress
4	Protocol for comparative bio-burden study of fifteen strains of *Candida glabrata* in Balb/c mice
5	Immunization of Balb/c mice for generation of antibodies against few purified recombinant mycobacterial proteins
6	Studying the effect of PPE 18 (Rv1196) on LPS induced endotoxaemia in mice
7	Use of nude mice in the study of tumorigenesis
8	Protocol for generation of mouse / rat polyclonal antibodies - version 2
9	Isolation of macrophages from Balb/c mice
10	Establishment of transgenic mouse model to study the role of *Ip6k1* in tumorigenesis
11	Studying the immunomodulatory role of some candidate recombinantly purified proteins of mycobacteria
12	Studying the *in vivo* immunomodulatory role of some candidate PE/PPE proteins of *Mycobacterium tuberculosis* recombinantly over-expressed in the non pathogenic mycobacterial strain of *M. smegmatis*
13	Studying the *in vivo* epigenetic role of some candidate proteins of *Mycobacterium tuberculosis* recombinantly over expressed in the non pathogenic mycobacterial strain of *M. smegmatis*
14	Protocol for testing tumorogenic and metastatic potential in nude mice
15	Investigating potential of *Mycobacterium tuberculosis* protein *PPE18* coated nanoparticles as therapy for microbial sepsis
16	Protocol for comparative vaginal bio-burden analysis of *Candida glabrata* strains in Balb/c mice
17	Protocol for comparative bio-burden analysis of *Candida glabrata* strains in C57BL/6 mice
18	Protocol for testing tumorogenic and metastatic potential of novel cancer related genes in nude mice

Table 2. IAEC approved projects proposed by various groups at CDFD, in progress during 2016-17.

We are close to the completion of CDFD's own Experimental Animal Facility which is under construction in the upcoming CDFD campus at Uppal, Hyderabad. We have provided our inputs for completion of the state of-the-art facility to maintain the standards of class 10000-100000 as per clean room norms for animal facilities. We are working towards the completion of the facility, to ensure its compliance and registration with CPCSEA, and enable the start of operations.

Future direction

Once the CDFD Experimental Animal Facility is operational, we aim to develop cryopreservation, archiving and retrieval of transgenic mouse strains for future use. Novel methods such as the CRISPR/Cas system will be developed to generate our own transgenic and knockout mice.

BIOINFORMATICS

In-charge	H A Nagarajaram,	Staff Scientist (till June 2016)
	Mr R Chandra Mohan	Technical Officer (from July.2016 to Nov. 2016)
	M Kavita Rao,	Staff Scientist (Since Nov. 2016)
Other Members	R Chandra Mohan	Technical Officer
	Prashanthi Katta	Technical Assistant

Objectives

1. To maintain various servers, workstations, PCs, printers and other peripheral devices;
2. To maintain CDFD website, to provide web based services and e-mail services;
3. To maintain Institute-wide LAN as well as the internet connectivity;
4. To secure CDFD network from security threats;
5. To integrate Institute's network into National and International grid computing networks;
6. To coordinate the procurement process of servers, workstations, PCs, laptops, printers, other peripheral devices and software required.

Summary of work done until the beginning of this reporting year (upto March 31, 2016)

- Activities related to installation, administration and maintenance of servers which provide various services, databases and computational jobs were undertaken.
- Internet, web, email services were provided with enhanced functionalities.
- High-end PCs, workstations, laptops, scanners and printers were procured and installed.
- Existing PC Annual Maintenance Contract with M/s Accel Frontline Limited was renewed.
- Renewed Antivirus licenses -400 Nos. for 3 years.
- Procured Microsoft Office latest verions-2016 -100 Nos. for installing/upgrading the existing versions.
- Initiated the process of procurement of servers, workstations and colour printers.
- Initiated the process of setting up of internet connection and Wi-Fi enabled local network facility at newly constructed student's hostel, Uppal.
- AMC for Dell Servers was awarded to M/s Dell International Services India Pvt. Ltd. for a period of one year.

Details of progress made in the current reporting year (April 1, 2016 - March 31, 2017)

- Activities related to installation, administration and maintenance of servers which provide various services, databases and computational jobs were undertaken.
- Internet, web, email-services have been provided with enhanced functionalities.
- High-end Server was procured and installed.
- Procured Super micro Workstations – 8 Nos. for computational lab purpose and installed.
- High-end PCs, laptops, scanners and printers were procured and installed.
- Existing PC Annual Maintenance Contract with M/s Accel Frontline Limited was renewed.
- Initiated the process of procuring Desktop Computers in bulk.
- Shifted P2 P leased line of 4 Mbps from Guruhakapla building to Uppal Campus.
- Internet facility provided to Hostel Students.
- Initiated the procurement of network equipment for new campus.
- AMC of Mail Server to Dell and AMC of other servers
- Procurement of Schrodinger software

INSTRUMENTATION

Head	Raghavendrachar J	Staff Scientist
Other Members	Mr. R N Mishra	Technical Officer
	Mrs S D Varalaxmi	Technical Officer
	Mr M Laxman	Technical Officer
	Mr Satyanarayana	Technical Officer
	Mr T Ramakrishna Reddy	Tech. Assistant

Objectives

To maintain repair and service all the equipment in laboratory. To provide pre-installation requirements for new instruments and to coordinate with the manufacturers / their agents in Installation and warranty service of the new instruments. Also to provide the reports on the newly arrived instruments and to follow up with the suppliers for short shipped items.

Summary of work done until the beginning of this reporting year

During the year 2015-16, we have installed 59 new equipments like Automatic Vertical Autoclaves, Cytogenetics Workstation (Spectral Karyotyping system) Upright Microscopes, Inverted Fluorescence Microscope, Bio-ruptors, PCR Machines, Refrigerated Centrifuges, Shaking waterbaths, -86^{0}C Deep Freezers, -20^{0}C Freezers, Cold Cabinets, Cooled Incubator, Refrigerators etc. and we have also completed 335 work orders for repair & maintenance of various laboratory equipments.

We are involved in coordinating the operations of sophisticated instruments in CDFD through an Equipment operations outsourcing agency, M/s Sandor Life Sciences. We are also involved in coordinating with M/s Vimta Labs for CDFD animal experimentation facility in their facility at Shameerpet.

Details of progress made in the current reporting year(April 1, 2016 – March 31, 2017)

During the year 2016-17, we have installed 25 new equipments like Shimadzu HPLC Prominence I LC 2030C, AB 3500 Genetic Analyzer HD, Spectromax M5 multimode reader, etc. and we have also completed 269 work orders for repair & maintenance of various laboratory equipments.

We are involved in coordinating the operations of sophisticated instruments in CDFD through an Equipment operations outsourcing agency, M/s Sandor Life Sciences. We are also involved in coordinating with M/s Vimta Labs for CDFD animal experimentation facility in their facility at Shameerpet.

In addition, we were involved in organizing the audio & visual requirements for presentations in various seminars, lectures and workshops, CDFD Foundation day lecture at IICT auditorium. We have maintained most of the equipment with maximum uptime in the Laboratory. Most of the Instruments are maintained by our Instrumentation staff, thereby saving on the expensive AMCs and with very little downtime of the equipment.

प्रकाशन
Publications

RESEARCH PAPERS

A. Publications during the year 2016

1. Abraham PR,Udgata A,Latha GS and Mukhopadhyay S (2016). *The Mycobacterium tuberculosis PPE protein Rv1168c induces stronger B cell response than Rv0256c in active TB patients.* ***Infection, Genetics and Evolution*** 40: 339-345

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12. Chaudhary AK, Sankar VH and Bashyam MD (2016). A novel large deletion that encompasses EDA and the downstream gene AWAT2 causes X-linked hypohidrotic/anhidrotic ectodermal dysplasia. ***Journal of Dermatological Science,*** 84 (1): 105-107

13. Chilukoti N, Kumar CMS and Mande SC (2016). GroEL2 OF M. tuberculosis reveals the importance of structural pliability in chaperonin function. ***Journal of Bacteriology***, 198 (3): 486-497.

14. Dalal A (2016). Dental stem cells: Hope or hype? ***Indian Journal of Dental Research***, 27(2): 113-114

15. Das Bhowmik A, Dalal A, Matta D, Rukmini MK, Kanikannan MA and Aggarwal S (2016). Identification of a novel splice site HSPG2 mutation and prenatal diagnosis in Schwartz Jampel Syndrome type 1 using whole exome sequencing. ***Neuromuscular Disorders,*** 26 (11): 809-814

16. Das Bhowmik A,Dalal A,Matta D,Sundaram C, and Aggarwal S (2016). *Targeted Next Generation Sequencing Identifies a Novel Deletion in LAMA2 Gene in a Merosin Deficient Congenital Muscular Dystrophy Patient.* ***Indian Journal of Pediatrics,*** 83 (4): 354-355.

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Pathogen *Xanthomonas*. ***PLoS Pathogens*** 12(11): e1006019. doi: 10.1371/journal.ppat.1006019

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39. Qayyum MZ, Dey D and Sen R (2016). Transcription Elongation Factor NusA Is a General Antagonist of Rho-dependent Termination in Escherichia coli. ***Journal of Biological Chemistry***, 291 (15): 8090-8108.
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44. Roy A, Reddi RK, Sawhney B, Ghosh DK, Anthony A and Ranjan, A (2016). Expression, Functional Characterization and X-ray Analysis of HosA, A Member of MarR Family of Transcription Regulator from Uropathogenic Escherichia coli. ***The Protein Journal,*** 35 (4): 269-282
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60. Verma N and Manna SK (2016). Advanced Glycation End Products (AGE) Potently Induce Autophagy through Activation of RAF Protein Kinase and Nuclear Factor κB (NF-κB). ***Journal of Biological Chemistry***, 291(3): 1481-1491.

61. Vimala A and Harinarayanan R (2016). Transketolase activity modulates glycerol-3-phosphate levels in Escherichia coli. ***Molecular Microbiology***, 100 (2): 263-277.

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B. Publications in 2017 (Till March 31, 2017)

64. Basu BTS, Dutta D, Duthie A, Guchhait N, Rocha BGM, da Silva, MFCG, Mokhamatam RB, Raviprakash N and Manna SK (2017). New dibutyltin(IV) ladders: Syntheses, structures and, optimization and evaluation of cytotoxic potential employing A375 (melanoma) and HCT116 (colon carcinoma) cell lines in vitro. ***Journal of Inorganic Biochemistry***, 166(1): 34-48

65. Basu BTS, Kehie P, Duthie A, Guchhait N, Raviprakash N, Mokhamatam RB, Manna SK, Armata N, Scopelliti M, Wang R and Englert U (2017). Synthesis, photophysical properties and structures of organotin- Schiff bases utilizing aromatic amino acid from the chiral pool and evaluation of the biological perspective of a triphenyltin compound. ***Journal of Inorganic Biochemistry,*** 168: 76-89

66. Bhat KH, Srivastava S, Kumar KS, Ghosh S and Mukhopadhyay S (2017). The PPE2 protein of Mycobacterium tuberculosis translocates to host nucleus and inhibits nitric oxide production. ***Scientific Reports***, 7: 39706

67. Chaudhary AK, Mohapatra R, Nagarajaram HA, Ranganath P, Dalal A, Dutta A, Danda S, Girisha KM and Bashyam MD (2017). The novel EDAR p.L397H missense mutation causes autosomal dominant hypohidrotic ectodermal dysplasia. ***Journal of the European Academy of Dermatology and Venereology,*** 31(1): e17-e20

68. Guo H, Cheng T, Chen Z, Jiang L, Guo Y, Liu J, Li S, Taniai K, Asaoka K, Kadono-Okuda K, Arunkumar KP, Wu J, Kishino H, Zhang H, Seth RK, Gopinathan KP, Montagné N, Jacquin-Joly E, Goldsmith MR, Xia Q and Mita K (2017). Expression map of a complete set of gustatory receptor genes in chemosensory organs of Bombyx mori. ***Insect Biochemistry and Molecular Biology,*** 82: 74–82

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71. Sarkar A and Nandineni MR (2017). Development of a SNP-based panel for human identification for Indian populations. ***Forensic Science International:*** *Genetics, 27: 58-66*

72. Shah A, Ganguli S, Sen J and Bhandari R (2017). Inositol Pyrophosphates: Energetic, Omnipresent and Versatile Signalling Molecules. **Journal of the Indian Institute of Science,** 97 (1): 23-40

73. Uttarilli A, Pasumarthi D, Ranganath P and Dalal A (2017). Functional characterization of arylsulfatase B mutations in Indian patients with Maroteaux-Lamy syndrome (mucopolysaccharidosis type VI). ***Gene***, 599: 19-27

74. Verma N and Manna SK (2017). Advanced Glycation End Products (AGE) Potentiates Cell Death in p53 Negative Cells via Upregulaion of NF-kappa B and Impairment of Autophagy. ***Journal of Cellular Physiology*** doi: 10.1002/jcp.25828

75. Yerra A, Mysarala DK, Siripurapu P, Jha A, Valluri SV and Mamillapalli A (2017). Effect of polyamines on mechanical and structural properties of Bombyx mori silk. ***Biopolymers,*** 107 (1): 20-27

C. Publications in Press (as on March 31, 2017)

76. Abraham PR, Pathak N, Pradhan G, Sumanlatha G and Mukhopadhyay S (2017). The N-terminal domain of Mycobacterium tuberculosis PPE17 (Rv1168c) protein plays a dominant role in inducing antibody responses in active TB patients. ***PLoS One***.

77. Ali A, Sailaja NV, Chinchole A and Tyagi S (2017). MLL/WDR5 Complex Regulates Kif2A Localization to Ensure Chromosome Congression and Proper Spindle Assembly during Mitosis. ***Developmental Cell.***

78. Das Bhowmik A, Gupta N, Dalal A and Kabra M (2017). Whole exome sequencing identifies a homozygous nonsense variation in ALMS1 gene in a patient with syndromic obesity. ***Obesity Research & Clinical Practice.***

79. Deborah DA, Vemireddy LR, Roja V, Patil S, Choudhary GP, Noor S, Srividhya A, Kaliappan A, Sandhya Rani B, Satyavathi VV, Anuradha G, Radhika K, Yamini KN, Gopalakrishna MK, Ranjith Kumar N, Siddiq EA and Nagaraju J (2017). Molecular dissection of QTL governing grain size traits employing association and linkage mapping in Basmati rice. ***Molecular Breeding***.

80. Dutta U, Bahal A, Vineeth VS, Vasantha S, Ranganath P and Dalal A (2017). A novel mosaic complex supernumerary marker chromosome in a girl with seizures: Systematic characterization of the complex marker. ***Gene Reports***.

81. Dutta U, Vempally S, Saraswat S, and Dalal A (2017). A rare combined balanced translocation t(2;22) and a novel mutation of COL6A2 gene in a girl with myopathy . ***Annals of Rehabilitation Medicine***.

82. Ghosh A, Sengupta A, Pavan Kumar SG, Ali N, Rama Rao EVVS, Bung N, Gopalakrishnan

B, Pal M and Haldar D (2017). A novel SIRT1 inhibitor, 4bb induces apoptosis in HCT116 human colon carcinoma cells partially by activating p53. ***Biochemical and Biophysical Research Communications.***

83. Himabindu P and Anupama K (2017). Decreased expression of stable RNA can alleviate the lethality associated with RNase E deficiency in Escherichia coli. ***Journal of Bacteriology.***
84. Kumar P, Prathyusha M, Chowdary KVS, Shah V, Shinde S, Kolli N, Rachita H, Nagarajaram H and Maddika S (2017). A human tyrosine phosphatase interactome mapped by proteomic profiling. ***Journal of Proteome Research***.
85. Mitra P., Ghosh G., Hafeezunnisa M. and Sen R. (2017). Rho protein: mechanism and action. **Annual Review of Microbiology.**
86. Narmadha Reddy G and Maddika S (2017). Interplay between the phosphatase PHLPP1 and an E3 ligase RNF41 stimulates proper kinetochore assembly via the outer-kinetochore protein SGT1. ***Journal of Biological Chemistry***.
87. Rachana RD, Ganji R, Singh SP, Mahalingam S, Banerjee S and Khosla S (2017). Cytosine methylation by DNMT2 facilitates stability and survival of HIV-1 RNA in the host cell during infection. ***Biochemical Journal***.
88. Saranathan R, Sudhakar P, Sawant AR, Tomar A, Madhangi M, Sah S, Annapurna S, Arunkumar KP and Prashanth K (2017). Disruption of tetR type regulator adeN by mobile genetic element confers elevated virulence in Acinetobacter baumannii. ***Virulence***.
89. Singh M and Nandineni MR (2017). Population genetic analyses and evaluation of 22 autosomal STRs in Indian populations. ***International Journal of Legal Medicine.***
90. Tallapaka KB, Ranganath P, and Dalal A (2017). Variable Expressivity and Response to Bisphosphonate Therapy in a Family with Osteoporosis Pseudoglioma Syndrome. ***Indian Pediatrics.***

D. Other Publications

1. Ali A and Tyagi S(2017).Diverse roles of WDR5-RbBP5-ASH2L-DPY30 (WRAD) complex in the functions of the SET1 histone methyltransferase family. **Journal of Bioscience** 42(1):155-159
2. Bharadwaj K, Jamal MD, Jain N, Dalal A, and Ranganath P (2017). An unexpected cause of microcephaly in a child with leukodystrophy. ***Genetic Clinics*** (Official publication of Society for Indian Academy of Medical Genetics) 10 (1): 7-11.
3. Chakraborty S and Arunkumar KP (2016). Book review of the *Annual Review of Genetics* 2015, Bonnie Bassler et al., (eds) ***Current Science***111: 933-935
4. Chanduri M and Bhandari R (2016). Protein pyrophosphorylation by inositol pyrophosphates. ***Cell Biology Newsletter, published by Indian Society of Cell Biology*** 35: 30-35.
5. Choudhary, R K, Mandal, J K, Auluck, N and Nagarajaram, H A (Eds.) (Springer 2016) Advances in Intelligent Systems and Computing. Advanced Computing and Communication Technologies **Proceedings of the 9th ICACCT, 2015**
6. Kasbekar DP (2016). History and development of genetics research in India: Three case studies. ***Indian Journal of History of Science*** 51.2.2: 423-430.
7. Kasbekar DP (2016). Neurospora deficiencies: The long and short of it. ***Cell Biology Newsletter*** 35: 1-6.
8. Kasbekar DP (2016). Obaid Siddiqi's study of the *PABA1* gene of the fungus *Aspergillus nidulans*. ***Biographical Memoirs of Fellows of the Indian National Science Academy Special*** 42: 16-24.
9. Kasbekar DP (2016). RNA-Seq, and ye shall find: Sexual-stage-specific A-to-I RNA editing in fungi. ***Journal of Bioscience*** 41: 171–172.
10. Kasbekar DP (2017). Sherlock Holmes, David Perkins, and the missing Neurospora inversions. ***Journal of Bioscience*** 42: 5-10.
11. Khosla S, Sharma G and Yaseen I (2016). Learning epigenetic regulation from mycobacteria. ***Microbial Cell*** 3: 92-94.
12. Kumar P, and Maddika S (2017). Cellular dynamics controlled by phosphatases. ***Journal of Indian Institute of Science.*** 97 (1): 129-145.
13. Mukhopadhyay S and Ghosh S. (2017). *Mycobacterium tuberculosis*: what is the role of PPE2 during infection? ***Future Microbiology*** (Invited Editorial Article) (In Press).

14. Rameshwaram NR, Shrivastava R, Pradhan G, Singh P and Mukhopadhyay S. Phagosome-lysosome fusion hijack - An art of intracellular bacteria. ***Proceedings of the Indian National Academy of Sciences*** (In Press).

15. Shinde SR, and Maddika S (2016). A modification switch on a molecular switch: Phosphoregulation of Rab7 during endosome maturation. ***Small GTPases.*** 7(3): 164-7.

16. Shinde SR, and Maddika S (2017). Post-translational modifications of Rab GTPases. ***Small GTPases.*** 1-8.

E. Patent filed/granted : NIL

मानव संसाधन विकास
Human Resource Development

PhD Program

For the PhD program CDFD invites applications from highly motivated candidates willing to take up challenges in modern biology, usually in the month of March. Keeping in view the interdisciplinary nature of modern biology, the Centre especially encourages persons from different scientific disciplines to take up challenges in these areas. Those admitted as Junior Research Fellows (JRFs) are encouraged to take admission in the PhD program of Manipal University or University of Hyderabad.

The eligibility for the program is MBBS or Masters degree in any branch of Science, Technology or Agriculture from a recognized University or Institute. Candidates (other than MBBS graduates) must have cleared National Eligibility Test (NET) with valid CSIR-JRF or UGC-JRF or DBT-JRF or ICMR-JRF or ICAR-JRF or INSPIRE-PhD or JEST or GATE (All India top 50 ranks of all Chemistry, Life Sciences and Biotechnology steams). Those who have appeared for their final semester examination, but are awaiting results, are also eligible to apply. Those with independent Senior Research Fellowships (SRF) from CSIR can also apply. As the number of applicants outnumbers the seats available each year by a ratio of 1:40 or more, eligible candidates are invited for a written examination followed by interviews of short-listed candidates.

As of August 31, 2017 the Centre has 92 research scholars working for their doctorates in different areas of research. In the reporting year15of the Research Scholars have completed PhD and are pursuing careers in science elsewhere in India or abroad.

Postdoctoral Program

In addition to the JRF program, the Centre also carries out training at the post-doctoral level. The post-doctoral fellows are funded through the extramural grants that CDFD receives. Some post-doctoral fellows are also selected competitively by the DST fast track young scientist scheme, or the DBT post-doctoral fellowship program.

Summer Training Program

CDFD provides admissions to summer training program to those students who are supported either by the Indian Academy of Science, Bangalore. In the reporting year21students received summer training at the Centre.

Training for students from BITS, Pilani

CDFD has an agreement with BITS, Pilani to provide project training to their M.Sc. students. Under this programme, the students spend 6 months to 1 year at CDFD and work on active projects being carried out here. The project work helps the students in gaining hands-onexperience in modern biology. In the reporting year, 2 students were given the opportunity to avail training under this programme.

Research Scholars Conferred PhD Degree During the Reporting Period
Students Conferred with Ph.D. Degree During 01.04.2016 - 31.03.2017

Sl. No.	Name of the Scholar	Supervisor from CDFD	Date of *viva voce* examination	Title of thesis
1	Mr. Atul Udgata	Dr. Sangita Mukhopadhyay	13.04.2016	“Role of PE/PPE Proteins in Modulation of Innate Immune Responses”
2	Mr. Vivek Kumar Srivastava	Dr. Rupinder Kaur	15.04.2016	“Mechanisms of iron acquisition and iron homeostasis in candida glabrata”
3	Ms. Anusha Uttarilli	Dr. Ashwin B. Dalal	27.04.2016	“Molecular analysis of Mucopolysaccharidoses in Indian Population”
4	Mr. Sita Rama Raju Adduri	Dr. M D Bashyam	11.05.2016	“Identification and analysis of molecular aberrations in squamous cell carcinoma of the tongue”
5	Mr. Amitava Basu	Dr. Sanjeev Khosla	01.07.2016	“Role of DNA Methyltransferase DNMT3L in Development”
6	Mr. Mohd. Zuhaib Qayyum	Dr. Ranjan Sen	18.07.2016	“Studies on the Mechanistic aspects of Rho-dependent Transcription Termination in Bacteria”
7	Ms. Aditi Sharma	Dr. Shekar C. Mande	08.08.2016	“Structural and functional analysis of Mycobacterium Tuberculosis GroELS”
8	Ms. Aanisa Nazir	Dr. R. Harinarayanan	16.09.2016	“Studies on the Physiological roles of basal (p)ppGpp and DksA in Escherichia Coli”
9	Mr. Bhavik Sawhney	Dr. AkashRanjan	20.09.2016	“Functional genomic studies on Plasmodium falciparum: Identification and Characterization of tRNA - Modifying enzymes and tRNA - drived fragments”
10	Mr. Jadav Rathan Singh	Dr. Rashna Bhandari	04.10.2016	“Investigating the cellular functions of mammalian inositol hexakisphosphate kinase 1 (IP6K1)”
11	Mr. Amit Pathania	Dr. J. Gowrishankar	04.10.2016	“Studies on genes of arginine/lysine transport and its regulation in E.coli”
12	Ms. Neeharika Verma	Dr. Sunil Kumar Manna	12.12.2016	“Understanding the mechanism of autophagy and its regulation”
13	Mr. S. Adeel Hussain Zaidi	Dr. Sunil Kumar Manna	12.12.2016	“Studies on profilin - 1 medicated signal transduction pathways in relevance to its tumour suppressor activity”
14	Ms. V. Vishalini	Dr. Ranjan Sen	10.02.2016	“Studies on bacterial transcription terminator RHO binding factors”
15	Mr. P. Venkata Vivek Reddy	Dr. M V Subba Reddy	08.03.2017	“Investigating the role of HACE1 in distinct cellular processes”

पुरस्कार एवं सम्मान
Awards and Honours

AWARDS & HONOURS

FACULTY & STAFF	
Dr. M Subba Reddy	Awarded the Wellcome Trust/DBT India Alliance Senior Fellowship
Dr. M Subba Reddy	Elected as Member of Guha Research Conference (GRC)
PhD STUDENTS & PROJECT PERSONNEL	
Mr. Sheo Shankar Pandey	Selected for poster for the ASM outstanding student abstract at ASM Microbe 2016.
Mr. Mr Abhishek Kumar (Laboratory of Computational & Functional Genomics) Mr. Raju Kumar (Laboratory of Molecular Oncology) Mr. Amit MahendraKarole (Laboratory of Cell Cycle Regulation) Ms. Mugdha Singh (Laboratory of Genomics and Profiling Applications) Shinde Swapnil Rohidas Anupama (Laboratory of Cell Death & Cell Survival)	Best Poster award in Colloquium held at Manipal University (5 April 2016)
Mr. Tishya Dasgupta Summer student (Laboratory of Computational & Functional Genomics)	Poster award at a Summer Symposium'16 at TFIR, Hyderabad
Ms. AnjanaKar (Diagnostics Divnsion)	2016 Developing Country Travel Grant from American Society of Human Genetics, Vancouver, BC, Canada to attend ASHG2016 conference at Vancouver, Canada (October 18-22, 2016)
CDFD Team (Dr Usha Dutta (Diagnostics), Mr. D S Negi (LDFS) & Ms. NeelimaThota (PDFS)	for poster presentation in DBT Pavilion that was awarded "BEST STALL" at the India International Science Festival 2016, New Delhi (December 7-11, 2016)
Ms Swathi Chodisetty, (Laboratory of Cell Cycle Regulation)	PLOS Genetics – best poster presentation award at the Chromosome Stability Meeting -2016 held at Thiruvananthapuram, Kerala (December 15-18, 2016)
Mr. Swapnil Shinde (Laboratory of Cell Death & Cell Survival)	Awarded Travel Grant from SERB to attend Keystone symposium conference held in British Columbia, Canada (March 5-9, 2017)

व्याख्यान, बैठक, कार्यशाला व अन्य महत्वपूर्ण कार्यक्रम

Lectures, Meetings, Workshops and Important Events

LECTURES

Visitor	Title of Lecture	Date
Dr Kaustuv Sanyal JNCASR, Bangalore	Genome indexing in Candida albicans	15.04.2016
Prof. Sudhir Krishna Senior Scientist NCBS, TIFR, Bangalore	The pathobiology of CD66+ cells in cervical cancers and some musings on the interphase with medicine	24.05.2016
Dr Jayakumar Rajadas Founding Director of Biomaterials and Advanced Drug Delivery Laboratory Stanford University School of Medicine (USA)	Nano patterned lipid soft particles for targeted therapeutic delivery	07.07.2016
Dr Tapas K Kundu Sir J.C. Bose National Fellow Transcription & Disease Laboratory Molecular Biology and Genetics Unit, JNCASR Bangalore	Fine-tuning gene expression in Physiology and Pathophysiology: Implications in therapeutics	18.07.2016
Dr Pankaj Kumar Assistant Professor (Research faculty), Biochemistry and Mol. Genetics, University of Virginia, USA	Transfer RNA Fragments (tRFs): a Novel Class of Non-micro Short RNAs	22.07.2016
Dr Subree Subramanian Assistant Professor Department of Surgery University of Minnesota, USA	Mechanisms of Tumor Progression and Immune Privilege in colon Cancer	17.08.2016
Dr Prem Singh Kaushal Wadsworth Center NYS-Department of Health Albany, NY, USA	Cryo-electron microscopy (cryo-EM) studies of ribonucleoprotein complexes: The group II intron and ribosomes	31.08.2016
Dr Shubhra Dutta Customer Consultant (Core Content) - South Asia A&G Team Research Solution Sales, RELX India Pvt. Ltd	Advantage **Mendeley**: time to change the way we do research	07.09.2016
Dr Parul Mishra University of Massachusetts Medical School Worcester, MA, USA	Investigating Structure-Function Dynamics of Protein Homeostasis Regulators: Applications to Health and Disease	26.09.2016
Dr Ganesh Nagaraju Associate Professor Department of Biochemistry Indian Institute of Science Bangalore	Distinct roles of RAD51 paralogs in DNA damage responses	04.10.2016
Dr. Venkata Chalamcharla National Institutes of Health (NIH/ NCI) USA	Transcription termination primes RNA-mediated epigenetic genome control	18.11.2016

Visitor	Title of Lecture	Date
Dr Srimonta Gayen University of Michigan Michigan, USA	Epigenetic regulation by long non-coding RNAs and histone modifiers through the lens of X chromosome inactivation	06.12.2016
Dr. Deepa Agashe NCBS, Bangalore	Evolution of codon use and tRNA genes in bacteria	09.12.2016
Prof Sreenivas Kurukuti Associate Professor Department of Animal Science University of Hyderabad Hyderabad	Spatio-temporal dynamics of 3-D genome architecture and gene expression during cellular differentiation	28.12.2016
Prof. Aseem Ansari The Genome Center of Wisconsin Department of Biochemistry University of Wisconsin-Madison	Designing Transcription Factors to Target Specific Genomic sites that Control Cell-Fates and Disease States	16.01.2017
Dr. Patrick Western Faculty Hudson Institute of Medical Research, Melbourne, Australia	Epigenetic programming in the germline: setting a foundation for the next generation	18.01.2017
Dr Rajesh S. Gokhale Scientist, National Institute of Immunology, Former Director, CSIR-Institute of Genomics and Integrative Biology New Delhi	Demystifying the Vitiligo Conundrum	27.01.2017
Dr Jose Sebastian Carnegie Institution for Science Stanford University, USA	Dealing with stress: cereal roots enact austerity measures during drought to bank water	07.02.2017
Dr Suresh Ramakrishna Asst prof Hanyang University South Korea	Genome-wide screening for functional deubiquitinating enzymes in human cells by DUB knockout library	20.02.2017
Dr Dipankar Bhandari Department of Biochemistry Max Planck Institute for Developmental Biology Spemannstrasse 35 Tuebingen, Germany	Role of the CCR4-NOT complex in post-transcriptional gene silencing	22.02.2017
Dr Prashanth Kumar Insitute of Bioinformatics Bangalore	Clinical Utility of Biomarkers: A Quest for Noninvasive Detection	24.02.2017
Dr Sharmila Bapat Senior Scientist NCCS, Pune	Expression based networks and functional pathways in molecular classification of ovarian cancer	24.02.2017
Dr Sunil Laxman InStem, Bangalore	Making commitments: how key metabolites determine cell proliferation decisions	02.03.2017
Dr Virander Chauhan Visiting Scientist ICGEB, New Delhi	Challenges in Translational Research: Development of Malaria Vaccine Candidates and functional peptides	03.03.2017

Dr Jerry L Workman Director of Postdoctoral Affairs Stowers Institute for Medical Research Kansas, USA	Protein complexes that modify chromatin for transcription and metabolism	06.03.2017
Dr Somenath Bakshi Harvard Medical School USA	Single-cell Measurement of Microbial Stress-response Dynamics in Complex Growth Conditions	16.03.2017
Dr Prasad Kasturi Department of Cellular Biochemistry Max-Planck Institute for Biochemistry Martinsried, Germany	Proteostasis during stress and aging in C.elegans	17.03.2017

LECTURE UNDER THE PROGRAM OF "LEARN FROM THE MASTER"

Visitor	Title of Lecture	Date
Dr Rajan Sankaranarayanan Chief Scientist CSIR-Centre for Cellular and Molecular Biology Hyderabad	Mechanistic basis of a key chiral checkpoint and functional insights	22.02.2017
Dr Suvendra N Battacharyya Principal Scientist and Head Molecular Genetics Division CSIR-India Institute of Chemical Biology, 4 Raja S.C Mullick Road Kolkata	Mighty regulation of a tiny RNA: miRNA activity and abundance control in mammalian cells	23.03.2017

IMPORTANT EVENTS

Event	Date
Shifting of CDFD building from Gruhakalpa to Residential campus, Uppal, Hyderabad (Inauguration of Two residential buildings of CDFD for Administrative activities.)	29.06.2016
Video-conference talks in partnership with Dr David del Alamo Rodriguez, Programme Manager, regarding EMBO Fellowships	13.07.2016
Video Conference by Hon'ble President of India to address the students and faculty members through Video-Conference using National Knowledge Network(NKN) from Rashtrapati Bhavan	10.08.2016
18thResearch Area Panels &Scientific Advisory Committee (RAP-SAC)	11.08.2016 & 12.08.2016
Independence Day	15.08.2016
Sadbhavana Diwas	19.08.2016
Brainstorming session on “Developing new (“Next gen”) Diagnostics tools”.	02.09.2016
41st Meeting of CDFD Governing Council	20.09.2016
21st Annual General Body meeting of the CDFD Society through Video Conference	22.09.2016
Hindi Day	26.09.2016
Observance of Vigilance Awareness Week from 31.10.2016 to 05.11.2016	31.10.2016 & 02.11.2016
Final meeting of New Indigo project organized by Dr H A Nagarajaram, Laboratory of Computational Biology (3 days meeting)	01.11.2016 to 03.11.2016
CDFD has celebrated IISF-2016 with DBT (Students visit from Tamil Nadu Agricultural University and Arora Degree College, Hyderabad to CDFD under 2nd India International Science Festival (IISF-2016) Celebrations.	30.11.2016
Hon'ble Minister for HRD, Sri Prakash Javadekar addressed all the heads of all higher educational institutions using National Knowledge Network(NKN) towards creating a digital economy	01.12.2016
Foundation Day Lecture by Dr Rajesh S Gokhale, NII, New Delhi	27.01.2017
Foundation Day celebrations at CDFD Uppal Campus	28.01.2017
Meeting on Molecular Microbiology (Mcube)	10.02.2017 to 11.02.2017
CDFD Building Committee meeting	02.03.2017
CDFD Finance Committee meeting	30.03.2017
CDFD Governing Council Committee meeting	30.03.2017
Lecture series under the program “Learn from the master”	22.02.2017, 23.03.2017

सी डी एफ डी कर्मचारियों की विदेशों में प्रतिनियुक्ति

Deputations Abroad of CDFD Personnel

List of Staff Members who had been Abroad on Deputation During the Period from 1st April 2016 to 31st March 2017

Name of the Employee & Designation	Duration of visit	Place & purpose of visit
Dr. Murali Dharan Bashyam Staff Scientist – VI	12.04.2016 to 24.04.2016	**USA:** (i) To visit Dr. Ramana Davuluri and Dr. Deb Chakrabarty at Northwestern University, Chicago, USA during 13-15 April, 2016. (ii) To present his work at the annual meeting of the American Association of Cancer Research, April 16 to 20, 2016, New Orleans, USA.
Dr. Nagarajaram H A Staff Scientist – VI	04.06.2016 to 10.06.2016	**GERMANY:** To attend the first NCDs-CAPomics meeting cum exchange visit under INNO-Indigo project (INDIGO-IPP2-072) held at Rostock, Germany.
Dr. Rupinder Kaur Staff Scientist – VI	17.06.2016 to 26.06.2016	**USA:** To attend the Cellular and Molecular Fungal Biology Gordon Research Conference on “Dynamic Interactions Across Scales from Single Molecules to Fungal Communities” held at Holderness, New Hampshire, USA during 19-24 June, 2016.
Dr. Devyani Halder Staff Scientist – V	31.03.2017 to 12.04.2017	**USA:** (i) To attend the Keystone Symposium on Genetic instability and DNA Repair joint with the meeting on DNA Replication and Recombination held during 02-06 April, 2017 at Santa Fe, New Mexico, USA. (ii) To deliver a lecture at NICHD, NIH, Bethesda held on 10.04.2017.
Dr. N Madhusudan Reddy Staff Scientist – V	02.05.2016 to 10.06.2016	**GERMANY:** To conduct research as Guest Scientist in the laboratory of Prof. Mark Stoneking, Department of Evolutionary Genetics, Max Planck Institute for Evolutionary Anthropology (MPI-EVA), Leipzig, Germany against his sixth visit to Prof. mark Stoneking’s Laboratory as a part of the “Max Planck Partner Group Programme” (MPPGP) between CDFD and MPI-EVA awarded by the Max Planck Society, Germany.

	22.08.2016 to 28.08.2016	**GERMANY:** (i) To attend the "Max Planck Symposium for Alumni and Early Career Researchers" held during 22-24 August, 2016 at Berlin, Germany. (ii) To visit Max Planck Institute for Evolutionary Anthropology (MPI-EVA) at Leipzig, Germany during 25-26 August 2016 to meet Prof. mark Stoneking to discuss about the progress of the project, manuscript preparation and submissions and to plan the future directions in the project.
	16.09.2016 to 23.09.2016	**INDONESIA:** To attend the 12th Indo-Pacific Association of Law, Medicine andScience (INPALMS) Congress 2016 held during 17-23 September, 2016 at the Stones Hotel, Bali, Indonesia.
	26.09.2016 to 30.09.2016	**USA:** To attend and present recent research findings with DNA-based markers in Indian populations in the form of a poster at the 27th International symposium on Human Identification (ISHI) held at Minneapolis, MN, USA.
Dr. Subhadeep Chatterjee Staff Scientist – V	26.06.2016 to 30.06.2016	**UK:** To attend and present a lecture at the scientific workshop on "Minimizing indiscriminate use of antibiotics" held at Ravenhall Hotel, Ravenscar, Scarborough YO13 0ET, UK.
Dr. R Harinarayanan Staff Scientist – III	08.08.2016 to 12.08.2016	**USA:** To attend and present his work at the conference titled "Molecular Genetics of Bacteria and Phages" held at Madison, Wisconsin, USA.

DEPUTATIONS ABROAD - STUDENTS

Name of the Research Scholar	Period of Visit	Name of the Conference
Ms. Nalini Raghunathan	15.05.2016 to 14.06.2016	**Paris**: to attend Carry out RNA-Seq experiments for study of the Mechanisms of Rho-dependant transcription termination
Mr. Anujit Sarkar	21.05.2016 to 24.05.2016	**Spain**: to attend European Human Genetics Conference " European Society of Human Genetics (ESHG)"
Mr. Imtiyaz Yaseen	22.05.2016 to 27.05.2016	**Switzerland**: to attend Gordon Research Conference titled " Chromatin Structure and function"
Mr. Sheo Shankar Pandey	16.06.2016 to 20.06.2016	**USA**: to attend "ASM Microbe 2016"
Mr. P Venkata Vivek Reddy	04.07.2016 to 07.07.2016	**Germany**: to attend Ubiquitin and Autophagy "Quality control in life process"
Ms. Anjana Kar	18.10.2016 to 22.10.2016	**Canada**: to attend Americian Society of Human Genetics
Mr. Swapnil Rohidas Shinde	05.03.2017 to 09.03.2017	**Canada**: to attend Keystone Symposia Conference "Tumor Metabolism: Mechanisms and Targets"

सीडीएफडी के संकाय एवं अधिकारी
Faculty and Officers of CDFD

SCIENTIFIC GROUP LEADERS (FACULTY)

Dr. Ranjan Sen
Dr. Sangita Mukhopadhyay
Dr. Murali Dharan Bashyam
Dr. Sanjeev Khosla
Dr. Sunil Kumar Manna
Dr. Akash Ranjan
Dr. Rupinder Kaur
Dr. Ashwin B Dalal
Dr. Rashna Bhandari
Dr. Devyani Halder
Dr. N Madhusudan Reddy
Dr. Shweta Tyagi
Dr. M V Subba Reddy
Dr. Subhadeep Chatterjee
Dr. Sardesai Abhijit Ajit
Dr. Rohit Joshi
Dr. R Harinarayanan

ADJUNCT FACULTY

Dr. EA Siddiq
Prof. T Ramasarma
Prof. Anuradha Lohia
Dr. Renu Wadhwa
Dr. Prajnya Ranganath
Dr. Shagun Aggarwal

OTHER GROUP LEADERS

Mr. Raghavendrachar J
Ms. Varsha

SENIOR ADMINISTRATIVE STAFF

Mr. J Sanjeev Rao

केन्द्र की समितियाँ
(31.03.2017 तक)

Committees of the Centre
(As on 31.03.2017)

MEMBERS OF CDFD SOCIETY

Dr. Harsh Vardhan Hon'ble Minster for Science & Technology and Earth Sciences	-	President
Prof K Vijay Raghavan Secretary, DBT, New Delhi	-	Member (Ex-officio)
Director General, CSIR, New Delhi	-	Member (Ex-officio)
Director General, Bureau of Police Research and Development (BPR&D) Ministry of Home Affairs, New Delhi	-	Member (Ex-officio)
Ms Sumita Mukherjee Joint Secretary & FA, DBT, New Delhi	-	Member (Ex-officio)
Joint Secretary (PM) Ministry of Home Affairs, New Delhi	-	Member (Ex-officio)
Joint Secretary & Legal Advisor Ministry of Law & Justice, New Delhi	-	Member (Ex-officio)
Prof Partha P Majumder Director, NIBMG, West Bengal Chairman of Scientific Advisory Committee, CDFD	-	Member (Ex-officio)
Dr A K Rawat Director, DBT, New Delhi Member	-	(Ex-officio)
Prof V S Chauhan, ICGEB, New Delhi	-	Member
Prof Dipankar Chatterji Indian Institute of Science (IISc), Bangalore	-	Member
Dr Rakesh K Mishra Director, CCMB, Hyderabad	-	Member
Dr Ranjan Sen In-charge Director, CDFD, Hyderabad	-	Member-Secretary

MEMBERS OF CDFD GOVERNING COUNCIL

Prof K Vijay Raghavan Secretary, DBT, New Delhi	-	Chairperson
Director General, CSIR, New Delhi	-	Member (Ex-officio)
Director General, Bureau of Police Research and Development (BPR&D) Ministry of Home Affairs, New Delhi	-	Member (Ex-officio)
Prof Partha P Majumder Director, NIBMG, West Bengal Chairman of Scientific Advisory Committee, CDFD	-	Member (Ex-officio)
Ms Gargi Kaul Joint Secretary & FA, DBT, New Delhi	-	Member (Ex-officio)
Shri CP Goyal Joint Secretary (Administration), DBT, New Delhi	-	Member (Ex-officio)
Joint Secretary (PM) Ministry of Home Affairs, New Delhi	-	Member (Ex-officio)
Joint Secretary & Legal Advisor, Ministry of Law & Justice, New Delhi	-	Member (Ex-officio)
Dr A K Rawat Director, DBT, New Delhi	-	Member (Ex-officio)
Prof V S Chauhan ICGEB, New Delhi	-	Member
Prof Dipankar Chatterji Indian Institute of Science (IISc), Bangalore	-	Member
Dr Rakesh K Mishra, Director, CCMB, Hyderabad	-	Member
Dr Ranjan Sen In-charge Director, CDFD, Hyderabad	-	Member-Secretary

MEMBERS OF CDFD RESEARCH AREA PANELS – SCIENTIFIC ADVISORY COMMITTEE

Name		Role
Dr Partha P Majumder NIBG, West Bengal	-	Chairman
Dr Arun Kumar Rawat DBT, New Delhi (DBT representative)	-	Member
Dr I Haque CFSL, Guwahati (MHA representative)	-	Member
Dr Manisha Madkaikar Natl. Instt. of Immunohaematology, Mumbai (ICMR representative)	-	Member
Dr K V Bhat NBPGR, New Delhi (ICAR representative)	-	Member
Dr Jyotsna Dhawan CCMB representative, Hyderabad	-	Member
Prof Sriram Ramaswamy TIFR Centre for Interdisciplinary Sciences Hyderabad	-	Member
Prof. B.K. Thelma University of Delhi (South Campus), New Delhi	-	Member
Prof Dr Seyed E Hasnain IIT, New Delhi	-	Member
Dr Saman Habib CDRI, Lucknow	-	Member
Dr Krishanu Ray TIFR, Mumbai	-	Member
Prof Tapas Kundu JNCASR, Bangalore	-	Member
Dr Anurag Agrawal IGIB, New Delhi	-	Member
Dr Debasisa Mohanty NII, New Delhi	-	Member
Dr R Sankaranarayanan CCMB, Hyderabad	-	Member
Prof Umesh Varshney IISc., Bangalore	-	Member
Dr Jaya Sivaswami Tyagi AIIMS, New Delhi	-	Member
Dr Usha Vijayraghavan IISc., Bangalore	-	Member
Dr Ranjan Sen Incharge-Director, CDFD, Hyderabad	-	Member Secretary

COMPOSITION OF FINANCE COMMITTEE

Prof. V S Chauhan, Visiting Scientist, International Centre for Genetic Engineering & Biotechnology (ICGEB), ICGEB Campus, Aruna Asaf Ali Marg,New Delhi-67	-	Chairman
Dr. Dipankar Chatterji, Chairman, Molecular Biophysics Unit, Indian Institute of science, Banglore-12	-	Member
Ms. Gargi Kaul, JS & FA, Dept. of Biotechnology, Ministry of Science & Technology, Block-2, 7th Floor, CGO Complex, Lodi Road, New Delhi-03	-	Member
Dr. A K Rawat, Director, Dept. of Biotechnology, Ministry of Science & Technology, Block-2, 6th Floor, CGO Complex, Lodi Road, New Delhi-03	-	Member
Shri A P Rao, FAO, CCMB, Hyderabad	-	Member
Dr. Ranjan Sen, Incharge Director, CDFD, Hyderabad	-	Member
T Abhishek Accounts Officer, CDFD, Hyderabad	-	Convener

MEMBERS OF THE INSTITUTIONAL BIO-SAFETY COMMITTEE (IBSC)

Dr. D P Kasbekar Haldane Chair, CDFD	-	Chairman
Dr. Arvind Kumar Principal Scientist, CCMB	-	DBT Nominee
Dr. Rashna Bhandari Staff Scientist – V, CDFD	-	Member Secretary
Dr. Krishnaveni Mishra Asso. Professor, Department of Biochemistry, SLS, University of Hyderabad, Hyderabad	-	Outside Expert
Dr. Ashwin B Dalal Staff Scientist – VI, CDFD	-	Biosafety Officer
Dr. M D Bashyam Staff Scientist – VI, CDFD	-	Internal Expert
Dr. Sanjeev Khosla Staff Scientist – VI, CDFD	-	Internal Expert
Dr. Rupinder Kaur Staff Scientist – VI, CDFD	-	Internal Expert

MEMBERS OF SEXUAL HARASSMENT COMPLAINTS COMMITTEE

Dr. Sangita Mukhopadhyay Staff Scientist – VI	-	Chairperson
Mr. J Sanjeev Rao Head – Administration	-	Member
Ms. V Naga Sailaja Technical Officer – II	-	Member
Ms. M V Sukanya Technical Officer – II	-	Member
Mr. MSA Zaman Khan Section Officer	-	Member
Ms. P Jamuna Gramya Resource Centre for Women (representing an NGO)	-	Member

MEMBERS OF INSTITUTIONAL BIO-ETHICS COMMITTEE

Prof. G B Reddy University College of Law, OU, Hyderabad	-	Chairperson
Prof. Sheela Prasad Associate Professor, Centre for Regional Studies, School of Social Sciences, University of Hyderabad	-	Member
Dr. Mahtab S Bamji Emeritus Scientist Dangoria Charitable Trust, Hyderabad	-	Member
Dr. Amita Kasbekar VP, Deloitte Consulting India Pvt. Ltd., RMZ, Hitech City, Hyderabad	-	Member
Dr. M D Bashyam Staff Scientist – VI, CDFD	-	Member
Dr. Sanjeev Khosla Staff Scientist – VI, CDFD	-	Member
Dr. Ashwin B Dalal Staff Scientist – VI, CDFD	-	Member Secretary

MEMBERS OF CDFD BUILDING COMMITTEE

Prof VS Chauhan JC Bose Fellow (DST), Distinguished Biotechnology, Research Professor, New Delhi	-	Chairman
Joint Secretary (Admin.) DBT, New Delhi	-	Member
Dr. Ranjan Sen In-charge Director, CDFD, Hyderabad	-	Member
Shri J Sanjeev Rao Head-Administration, CDFD, Hyderabad	-	Member
Shri T. Abhishake Account Officer, CDFD, Hyderabad	-	Member
Shri Raghavendrachar Jois In-charge Engineering, CDFD, Hyderabad	-	Member-Convenor

OFFICIAL LANGUAGE IMPLEMENTATION COMMITTEE-OLIC

STATUTORY MEMBERS

Dr Ranjan Sen, Incharge-Director CDFD, Hyderabad	-	Chairman
Mr J Sanjeev Rao Head-Administration	-	Member
Mr Abhishek Accounts Officer	-	Member
Mr Ravinder I/c Stores &Purchase	-	Member
Mrs Varsha Staff Scientist	-	Member Secretary

OTHER MEMBERS

Dr Hari Narayan
Staff Scientist

Mr R Jois
Staff Scientist

Mr V. Punnaiah
Executive Engineer

Mrs Mutthulakshmi
Technical Officer

Mr M S Rao
Management Assistant

MEMBERS OF CDFD MANAGEMENT COMMITTEE

Director CDFD, Hyderabad	-	Chairman
Dr. D P Kasbekar Haldane Chair	-	Member
Dr. Sunil Kumar Manna SS – VI	-	Member (for a 2 year period)
Dr. Shweta Tyagi SS – V	-	Member (for a 2 year period)
Accounts Officer	-	Member
Head – Administration	-	Member – Convenor

सूचना अधिकार अधिनियम, 2005 का परिपालन
Implementation of RTI Act, 2005

IMPLEMENTATION OF RIGHT TO INFORMATION (RTI) ACT, 2005

Appellate Authority : Dr D P Kasbekar

Central Public Information Officer : Ms M Kavita Rao (Till 30.06.2016) & Ms Varsha (from 01.07.2016)

Details about the RTI applications and appeals received in CDFD

As received under RTI Act 2005	**Opening Balance as on 1.4.2016**	**Received during the year 2016-17**			**Disposed of during the year 2016-17**				**Closing Balance as on 31.3.2017**
		Received directly	Received as transfer from other Public Authorities [u/s 6(3) of Act]	Total	Decisions where applications accepted/ appeals upheld	Decisions where applications/ appeals rejected	Transferred to other Public Authorities [u/s 6(3) of Act]	Total	
Applications	1	34	28	63	55	2	0	57	6
Appeals	01	04	Not applicable	04	05	Nil	Not applicable	5	Nil

बजट एवं वित्त
Budget and Finance

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS HYDERABAD

Budget & Finance 2016-17

Sources of Funds

The Financial resources of the Centre are the Core Plan Grant-in Aid provided by the Department of Biotechnology, Government of India as against Annual Budgetary projections made by the Institute. Other resources are in the form of Research Grants provided by various National and International agencies and also from Services rendered by CDFD. The components of the core grants are Plan (Recurring) essentially for meeting expenditures on salaries, Operating expenses etc., and Plan (Non-Recurring) for meeting expenses on account of Equipments, Infrastructure and Furnishing etc.,

Receipts during the year 2016-17

Particulars	Amount in Lakhs	Percentage - %
Plan Grant in Aid	6000.00	73.74
Sponsored Projects	901.96	11.08
CDFD Services	70.71	0.87
Misc Receipts	1164.49	14.31
Total	**8137.16**	**100.00**

I. Application of Funds during 2016-17 (Plan Grant in Aid)

S No	Particulars	Amount in Lakhs	Percentage- %
1	**Recurring**		
	GIA- Salaries	1203.99	27.80
	GIA-General	1865.93	43.08
	Total	**3069.92**	**70.88**
2	**Non-Recurring**		
	GIA- Capital	1261.18	29.12
	Total	**1261.18**	**29.12**
	Grand Total	**4331.10**	**100.00**

II. Application of Funds during 2016-17 (Extra Mural Projects)

S No	Particulars	Amount in Lakhs	Percentage- %
1	**Recurring**		
	Salaries	298.48	45.05
	General	289.32	43.67
	Total	**587.80**	**88.72**
2	**Non-Recurring**		
	Capital	74.74	11.28
	Total	**74.74**	**11.28**
	Grand Total	**662.54**	**100.00**

लेखा परिक्षक की रिपोर्ट
Auditor's Report

B Purushottam & Co

Chartered Accountants

AUDITOR'S REPORT

Date: 20-09-2017

The Director,
Centre for DNA Fingerprinting and Diagnostics,
Uppal, Hyderabad – 500 039

We have audited the attached Balance Sheet of CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, Hyderabad, as at 31st March 2017 and also the Income & Expenditure Account for the year ended on that date annexed there to. These Financial Statements are the responsibility of the organization management. Our responsibility is to express an opinion on these Financial Statements based on our audit.

We report that:

1. We have obtained all the information and explanations, which to the best of our knowledge and belief, were necessary for the purpose of our audit.
2. In our opinion, the organization has kept proper books of account as required by law so far, as appears from our examination of these books.
3. The Balance Sheet and Income & Expenditure account dealt with by this report is in agreement with the books of account.
4. (a) The centre has maintained accounts on accrual basis.
   (b) The Centre receives extra mural grants from various National & International agencies for specific research activities. The Centre has a policy of allocating the overheads and transfer of expenditure of CDFD to different projects at the end of the financial year after taking into account the amount of maximum permissible limit of overheads and also based on the approved budget estimates and expenditure of the respective projects during the financial year.
5. In our opinion and to the best of our information and according to the explanations given to us, the said Balance Sheet and the Income & Expenditure account read together with the notes thereon gives the required information in the manner so required and gives a true and fair view.
   a) In so far it relates to the Balance Sheet as at 31st March 2017 and
   b) In so far as it relates to the Income & Expenditure account excess of expenditure over income for the year ended on 31st March 2017.

for **B Purushottam & Co.,**
Chartered Accountants
Reg. No.002808S

[CH SATYANARAYANA]
M.No.19092

Place: Hyderabad
Date: 20/09/2017

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS,HYDERABAD

BALANCE SHEET AS ON 31st MARCH 2017

(Amount - Rs.)

	Schedule	Current Year	Previous Year
Corpus / Capital Fund	1	1942028103	1686691192
Reserves and Surplus	2	25990202	16484058
Earmarked / Endowment funds	3	5912597	0
Secured Loans & Borrowings	4	0	0
Unsecured Loans & Borrowings	5	0	0
Deffered Credit Liabilities	6	0	0
Current Liabilities and Provisions	7	81773812	85746032
Current Liabilities and Provisions	7	70028009	70814398
TOTAL		**2055704714**	**1788921282**
ASSETS			
Fixed Assets	8	1586265401	1537816689
Investments- From Earmarked / Endowment Funds	9	291098273	71098273
Investments - Others	10	31870241	30065721
Current Assets, Loans, Advances etc.	11	146470799	149940599
Miscellaneous Expenditure			
TOTAL		**2055704714**	**1788921282**
Significant Accounting Policies	24		
Contingent Liabilities and Notes on Accounts	25		

DIRECTOR
CDFD

For B.PURUSHOTTAM & CO.
CHARTERED ACCOUNTANTS
(B.PURUSHOTTAM)

ACCOUNTS OFFICER
CDFD

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS,HYDERABAD

INCOME & EXPENDITURE FOR THE YEAR ENDED 31st MARCH 2017

(Amount - Rs.)

INCOME	Schedule		Current Year		Previous Year
Income from Sales/Services	12		7071528		8641034
Grants/Subsides	13		300000000		345000000
Fees/Subscriptions	14		0		0
Income from Investments	15		5685649		18375260
Income from Royality, Publications etc.	16		0		0
Interest Earned	17		1785882		1390306
Other Income	18		4788491		7236505
Increase/(decrease) in stock of Finished goods and works-in-progress	19		0		0
TOTAL (A)			**319331550**		**380643105**
EXPENDITURE					
Establishment Expenses	20		122420108		119831151
Administrative Expenses	21		164271394		212729759
Expenditure on Grants, Subsides etc.	22		0		0
Interest	23		0		0
Depreciation (Net Total at the year-end -corresponding to Schedule 8)		67006639		70461166	
Less:Transferred to Grants-in-Aid		67006639	0	70461166	0
Provision For Salaries			**8264377**		**9780756**
TOTAL (B)			**294955879**		**342341666**

DIRECTOR
CDFD

For B.PURUSHOTTAM & CO.
CHARTERED ACCOUNTANTS
(B.PURUSHOTTAM)

ACCOUNTS OFFICER
CDFD

Balance being excess of Income over Expenditure (A-B)			24375671		38301439
Transfer to Special Reserve (Specify each)					
Transfer to/from General Reserve			9506144		8641034
BALANCE BEING SURPLUS/(DEFLICT) CARRIED TO CORPUS/CAPITAL FUND			14869527		29660405
SIGNIFICANT ACOUNTING POLICIES	24				
CONTINGENT LIABILITIES AND NOTES ON ACCOUNTS	25				

DIRECTOR
CDFD

For B.PURUSHOTTAM & CO.
CHARTERED ACCOUNTANTS
(B.PURUSHOTTAM)

ACCOUNTS OFFICER
CDFD

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS,HYDERABAD

RECEIPTS AND PAYMENTS ACCOUNT FOR THE YEAR ENDED 31st MARCH 2017

(Amount - Rs.)

RECEIPTS	Current Year	Previous Year	PAYMENTS	Current Year	Previous Year
1.Opening Balances			**1. Expenses**		
a) Cash in hand	0	0	a) Establishment Expenses	140596026	119831151
b) Bank Balances			b) Administrative Expenses	164271394	212729759
i) In current accounts	27660890	13313617	c) Schedule 22	0	0
ii) In deposit accounts	0	0			
iii) Savings accounts	11145109	9433617			
2. Grants Received			**2. Payments made against funds for various projects**		
a) From Government of India	600000000	845000000	(Name of the fund or project should be shown along with the particulars of payments made for each project)		
b) From State government					
c) From other sources (details)			Projects (Annexure F)	66254246	102743689
(Grants for capital & revenue			CSIR(Stipend)	7591957	11956274
exp. To be shown seperately)			DBT(Stipend)	7088271	9595329
Research Associates - CSIR(Stipend)	6712089	8453559	DST(Stipend)	1867781	2238533
Research Associates - DBT(Stipend)	9882757	5344314	ICMR(Stipend)	2622586	3338763
Research Associates - ICMR(Stipend)	1922748	1754439	UGC(Stipend)	9594702	11836172
Research Associates - IISC(Stipend)	106012	36400	IISC(Stipend)	0	265938
Research Associates - UGC(Stipend)	4127705	2064806	**3. Investments and deposits made**		
Research Associates - DST(Stipend)	0	1362000	a) Out of Earmarked/Endowement funds	750000000	420000000
Projects (Annexure - C)	90196329	98445682	b) Out of Own Funds (Investments-Others)	0	0
3. Income on Investments from			**4. Expenditure on Fixed Assets & Capital Work-in-Progress**		
a) Earmarked/Endow. Funds	5504248	3168348	a) Purchases of Fixed Assets:		

DIRECTOR
CDFD

For B.PURUSHOTTAM & CO.
CHARTERED ACCOUNTANTS
(B.PURUSHOTTAM)

ACCOUNTS OFFICER
CDFD

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS,HYDERABAD

RECEIPTS AND PAYMENTS ACCOUNT FOR THE YEAR ENDED 31st MARCH 2017

(Amount - Rs.)

RECEIPTS	Current Year	Previous Year	PAYMENTS	Current Year	Previous Year
b) Own Funds (Oth. Investment)			Books & Journals	572775	560767
Investments EnCashed	530000000	384000000	Equipment -Lab/Office/Furniture	9889057	23244176
			b) Expenditure on Capital Work-in-Progress:	97519496	479498388
4. Interest Received					
a) On Bank deposits	507346	106041	**5. Refund of surplus money/Loans**		
b) Loans, Advances etc	0	18012496	a) To the Government of India	0	0
Interest on LC	1278536	1284265	b) To the State Government	0	0
Interest on Computer Advance, Conveyance Advance and HBA	6171	19018	c) To other providers of funds	0	0
5. Other Income(Specify)			**6. Finance Charges (Interest)**	0	0
a) Analysis Charges	7071528	8641034			
	0	7843024	**7. Other Payments (Specify)**		
6. Any Other Receipts(Give Details)			Advances (Annexure-D)	91775754	158544851
I-Remittances (Annexure-A)	26977196	29358677	I-Remittances (Annexure-E)	25538602	28161879
			CPF A/c	16872120	7756535
CPF-SUB,Arrears and adv.Refund	13734820	15265679	New Pension Scheme	3284824	3424598
Sundry Receipts	4312588	7090257	NIMS	1481353	3376101
Application Fee	15125	17500	**8. Closing Balances**		
Provident Fund Salwage	0	0	a) Cash in hand		
Free Gifts - Donations	0	0	b) Bank Balances		
Sale OF Tender Forms	90500	10500	i) In current accounts	17665452	27660890
Leave Salary-Pension Contribution	52836	44030	ii) In deposit accounts	0	0
License Fee	54880	55200	iii) Savings accounts	9699923	11145109

DIRECTOR
CDFD

For B.PURUSHOTTAM & CO.
CHARTERED ACCOUNTANTS
(B.PURUSHOTTAM)

ACCOUNTS OFFICER
CDFD

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS,HYDERABAD

RECEIPTS AND PAYMENTS ACCOUNT FOR THE YEAR ENDED 31st MARCH 2017

(Amount - Rs.)

RECEIPTS	Current Year	Previous Year	PAYMENTS	Current Year	Previous Year
Welfare Fund	0	0			
NPS	3284180	3453474			
Advance/Refunds/Recovery/Adj(Annexure-B)	73435613	170319917			
NIMS	6107113	4011009			
TOTAL	**1424186319**	**1637908903**	**TOTAL**	**1424186319**	**1637908903**

DIRECTOR
CDFD

For B.PURUSHOTTAM & CO.
CHARTERED ACCOUNTANTS
(B.PURUSHOTTAM)

ACCOUNTS OFFICER
CDFD

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

BALANCE SHEET AS ON 31st MARCH 2017

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 1 - CORPUS/CAPITAL FUND :				
Balance as at the begining of the year		1686691192.00		1212702539.00
Add : Contribution towards Corpus/Capital Fund				
CDFD Core - Plan (Non-Recurring)	300000000.00		500000000.00	
Capitalised portion of Capital Expenditure of projects	7474023.00	307474023.00	14789414.00	514789414.00
Less : Depreciation For the Year 2016-2017	67006639.00	67006639.00	70461166.00	70461166.00
Add : Excess of Expenditure over Income	14869527.00	14869527.00	29660405.00	29660405.00
BALANCE AS AT THE YEAR - END		**1942028103.00**		**1686691192.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 2 -RESERVES AND SURPLUS :				
1.Capital Reserve :				
As per last Account	0.00		0.00	
Addition during the year	0.00		0.00	
Less : Deductions during the year	0.00	0.00	0.00	0.00
2.Revolution Reserve :				
As per last Account	0.00		0.00	
Addition during the year	0.00		0.00	
Less : Deductions during the year	0.00	0.00	0.00	0.00
3.Special Reserves :				
As per last Account	0.00		0.00	
Addition during the year	0.00		0.00	
Less : Deductions during the year	0.00	0.00	0.00	0.00
4.General Reserve :				
As per last Account	16484058.00		0.00	
Addition during the year	9506144.00		16484058.00	16484058.00
Less : Deductions during the year	0.00	25990202.00	0.00	0.00
Total		**25990202.00**		**16484058.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 3 - EARMARKED/ENDOWMENT FUNDS :				
(Refer Annexures)				
(a) Opening balance of the Funds		**-18029485.84**		**-13731478.00**
(b) Additions to the Funds :				
i. Donations /grants	90196329.00		98445681.00	
ii. Income from investments made on account of funds	0.00		0.00	
iii. Other additions	0.00	90196329.00	0.00	98445681.00
TOTAL (a+b)		**72166843.16**		**84714203.00**
(c) Utilisation/Expenditure towards objective of funds				
(i) Capital Expenditure (Refer Annexures I & II)				
- Fixed Assets	7474023.00		14354226.00	
- Others	0.00	7474023.00	435188.00	14789414.00
- Total				
(ii) Revenue Expenditure (Refer Annexures I & II)				
- Salaries, Wages and allowances etc.	29848272.00		31698402.00	
- Rent	0.00		0.00	
- Other Expenses	28931951.13	58780223.13	56255873.00	87954275.00
Total				
TOTAL (c)		66254246.13		102743689.00
NET BALANCE AS AT THE YEAR-END [(a + b)-c]		**5912597.03**		**-18029486.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 4 - SCHEDULE LOANS AND BORROWINGS :				
1. Central Government		0		0
2. State Government (Specify)		0		0
3. Financial Institutions				
a) Term Loans	0		0	
b) Interest accured and due	0	0	0	0
4. Banks :				
a) Terms Loans	0	0	0	0
- Interest accured and due	0		0	
b) Other Loans	0		0	
- Interest accured and due	0	0	0	0
5. Other Institutions and Agencies		0		0
6. Debentures and Bonds		0		0
7. Others (Specify)				
TOTAL		**0**		**0**
Note: Amount due within one year				

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2015

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 5 - UNSECURED LOANS AND BORROWINGS :				
1. Central Government		0		0
2. State Government (Specify)		0		0
3. Financial Institutions		0		0
4. Banks :				
a) Terms Loans	0		0	
b) Other Loans	0	0	0	0
5. Other Institutions and Agencies		0		0
6. Debentures and Bonds		0		0
7. Fixed Deposits		0		0
8. Others (Specify)		0		0
TOTAL		**0**		**0**
Note: Amount due within one year				

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 6 - DEFFERED CREDIT LIABILITIES :				
a) Acceptances secured by hypothecation of capital equipment and other assets		0		0
b) Others		0		0
TOTAL		**0**		**0**
Note: Amount due within one year				

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 7 - CURRENT LIABILITIES AND PROVISIONS :				
A. CURRENT LIABILITIES				
1. Acceptances				
2. Sundry Creditors				
3. Advances Received				
4. Interest accured but not due on:				
5. Statutory Liabilities:				
6. Other current Liabilities				
CDFD.CP Fund A/C(Annexure-G)	43287242.00		44620022.00	
Diagnostics Collabration With NIMS	5260668.00		634908.00	
ECCS		163285.00		0.00
EMD		2303652.00		1858034.00
GSLI		24616.00		33339.00
House Building Advance	129831.00		129831.00	
Income Tax	910797.00		97507.00	
Lab Security Deposit & Hostel Security Deposit	1294396.00		1272716.00	
LIC		2550.00		2550.00
Others (I-Remittances)	487642.00		296555.00	
Out Standing Liabilities	11845456.00		20240618.00	
PPF EMPLOYER SHARE	622172.00		562436.00	
Professional Tax	96592.00		98642.00	
Public Provident Fund	391158.00		406240.00	
Royalty & Consultancy	1531642.00		1531642.00	
Security Deposit	2547185.00		1643475.00	
Service Tax	502477.00		0.00	
STAFF BENEVOLENT FUND	12569.00		0.00	
TA Abroad [Advance]	109576.00		0.00	
TA-DA-Hon within India [Advance]	65909.00		0.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 7 - CURRENT LIABILITIES AND PROVISIONS :				
TDS		1559790.00		1920764.00
Works Tax	360230.00		255858.00	
Workshop & Conference	0.00	73509435.00	360139.00	75965276.00
TOTAL (A)		**73509435.00**		**75965276.00**
B.PROVISIONS				
1. For Taxation	0.00		0.00	
2. Gratuity	0.00		0.00	
3. Superannuation/Pension	0.00		0.00	
4. Accumulated Leave Encashment	0.00		0.00	
5. Trade Warranties/Claims	0.00		0.00	
6. Others (Specify)	8264377.00	8264377.00	9780756	9780756
TOTAL (B)		**8264377.00**		**9780756.00**
TOTAL (A+B)		**81773812.00**		**85746032.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

SCHEDULE 8 - FIXED ASSTES :	GROSS BLOCK				DEPRECIATION				NET BLOCK	
	Cost/valuation As at begining of the the year	Addition during during the year	Deductions during the year	Cost/valuation at at the year end	As at the begining the year	On additions during the year	On Deductions during the year	Total up to the uo to the year end	As at the Current current year end	As at the prevous year end
A. FIXED ASSETS:										
1. LAND:										
a) Freehold	3900000.00	0.00	0.00	3900000.00	0.00	0.00	0.00	0.00	3900000.00	3900000.00
b) Leasehold	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
2. BUILDINGS										
a) On Freehold Land	220052369.00	0.00	0.00	220052369.00	87694995.00	13235737.00	0.00	100930732.00	119121637.00	132357374.00
b) On Leasehold Land	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
c) Ownership Flats/Premises	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
d) Superstructures on Land not belongs to the entity	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
3. PLANT MACHINERY & EQUIPMENT	711815162.05	17356080.00	0.00	729171242.05	390541754.00	52019927.00	0.00	442561681.00	286609561.05	321273408.05
4. VEHICLES	4153026.00	0.00	0.00	4153026.00	3673085.00	71991.00	0.00	3745076.00	407950.00	479941.00
5. FURNITURE, FIXTURES	16037396.00	7000.00	0.00	16044396.00	11370161.00	445116.00	0.00	11815277.00	4229119.00	4667235.00
6. OFFICE EQUIPMENT	12149882.00	0.00	0.00	12149882.00	9576455.00	428386.00	0.00	10004841.00	2145041.00	2573427.00
7. COMPUTER/PERIPHERALS	132023.00	0.00	0.00	132023.00	0.00	0.00	0.00	0.00	132023.00	132023.00
8. ELECTRIC INSTALLATIONS	0.00				0.00	0.00	0.00	0.00		
9. LIBRARY BOOKS	19013189.00	572775.00	0.00	19585964.00	18526977.00	728181.00	0.00	19255158.00	330806.00	486212.00
10. TUBEWELLS & WATER SUPPLY	0.00				0.00	0.00	0.00	0.00		
11. OTHER FIXED ASSETS	8857898.00	0.00	0.00	8857898.00	8084889.00	77301.00	0.00	8162190.00	695708.00	773009.00
Airconditioning works		0.00	0.00		0.00	0.00	0.00	0.00		
Aluminium partition work		0.00	0.00		0.00	0.00	0.00	0.00		
DG Set		0.00	0.00		0.00	0.00	0.00	0.00		
Paintings		0.00	0.00		0.00	0.00	0.00	0.00		
Typewriters		0.00	0.00		0.00	0.00	0.00	0.00		
Miscellaneous non consumables		0.00	0.00		0.00	0.00	0.00	0.00		
Other Assets		0.00	0.00		0.00	0.00	0.00	0.00		
EMB Net		0.00	0.00		0.00	0.00	0.00	0.00		
TOTAL	996110945.05	17935855.00	0.00	1014046800.05	529468316.00	67006639.00	0.00	596474955.00	417571845.05	466642629.05
B. CAPITAL WORK-IN-PROGRESS	1071174059.70	97519496.00	0.00	1168693555.70	0.00	0.00	0.00	0.00	1168693555.70	1071174059.70
TOTAL	**2067285004.75**	**115455351.00**	**0.00**	**2182740355.75**	**529468316.00**	**67006639.00**	**0.00**	**596474955.00**	**1586265400.75**	**1537816688.75**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 9 - INVESTMENTS FROM EARMARKED/ENDOWMENT FUNDS :		
1. In Government Securities	0.00	0.00
2. Other approved securities	0.00	0.00
3. Shares	0.00	0.00
4. Debentures and Bonds	0.00	0.00
5. Subsidiaries and Joint Ventures	0.00	0.00
6. Others (to be specified) - STDRs (Annexure-J)	291098273.00	71098273.00
TOTAL	**291098273.00**	**71098273.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 10 - INVESTMENTS - OTHERS :		
(Annexure-K)		
1. In Government Securities	0.00	0.00
2. Other approved securities	0.00	0.00
3. Shares	0.00	0.00
4. Debentures and Bonds : UTI Bonds		
5. Subsidiaries and Joint Ventures	0.00	0.00
6. Others (to be specified) - STDRs,(CPF),CDFD CP FUND A/C	31870241.00	30065721.00
TOTAL	**31870241.00**	**30065721.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 11 - INVESTMENTS - OTHERS :	**Current Year**		**Previous Year**	
A. CURRENT ASSETS				
1. Inventors				
a) Stores and Spares	0.00		0.00	
b) Loose Tools	0.00		0.00	
c) Stock-in-trade				
Finished Goods	0.00		0.00	
Work-in-progress	0.00		0.00	
Raw Materials	0.00	0.00	0.00	0.00
2. Sundry Debtors:				
a) Debts Outstanding for a period exceeding six months	0.00		0.00	
b) Others-Life Membership Fees	169236.00	169236.00	169236.00	169236.00
3. Cash balances in hand (including cheques/drafts and imprest)				
4. Bank Balances:				
a) With Scheduled Banks:				
-On Current Accounts	17665451.85		27660889.85	
-On Deposit Accounts (includes margin money)	0.00		0.00	
-On Savings Accounts	9699922.91	27365374.76	11145109.42	38805999.27
b) With non-Schedules Banks:				
-On Current Accounts	0.00		0.00	
-On Deposit Accounts	0.00		0.00	
-On Savings Accounts	0.00	0.00	0.00	0.00
5. Post Office-Savings Accounts				
TOTAL (A)		**27534610.76**		**38975235.27**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 11 - INVESTMENTS - OTHERS :				
B. LOANS, ADVANCES AND OTHER ASSETS				
1. Loans:				
a) Staff	0.00		0.00	
b) Other Entities engaged in activities/objectives similar to that of the Entity	0.00	0.00	0.00	0.00
2. Advances and other amounts recoverable in cash or in kind or for value to be received				
a) On Capital Account (Annexure-H)	86818537.00		61240068.00	
b) Prepayments - Deposits (Annexure-I)	16472947.00		16488897.00	
c) Others (TDS Receivable)	437792.00	103729276.00	0.00	77728965.00
3. Income Accured:				
a) On Investments from Earmarked/Endowments Funds	0.00		0.00	
b) On Investments - Others	15206912.00		15206912.00	
c) On Loans and Advances	0.00		0.00	
d) Others	0.00	15206912.00	0.00	15206912.00
4. Claims Receivable			18029485.84	18029485.84
TOTAL (B)		118936188.00		110965362.84
TOTAL (A+B)		**146470798.76**		**149940598.11**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 12 - INCOME FROM SALES/SERVICES :		
1) Income from sales		
a) Sale of Finished Goods	0.00	0.00
b) Sale of Raw Material	0.00	0.00
c) Sale of Scraps	0.00	0.00
2) Income from Services		
a) Labour and Processing Charges	0.00	0.00
b) Professional/Consultancy Services (Analysis Charges)	7071528.00	8641034.00
c) Agency Commission and Brokerage	0.00	0.00
d) Maintenance Services (Equipment/Property)	0.00	0.00
e) Others (Specify)	0.00	0.00
TOTAL	**7071528.00**	**8641034.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 13 - GRANTS/SUBSIDES :		
(Irrevocable Grants & Subsides Received)		
1) Central Government (DBT Plan Grant-in-Aid)	300000000.00	345000000.00
2) State Government(s)	0.00	0.00
3) Government Agencies	0.00	0.00
4) Institutions/Welfare Bodies	0.00	0.00
5) International Organisations	0.00	0.00
6) Others (Specify)	0.00	0.00
TOTAL	**300000000.00**	**345000000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 14 - FEES/SUBSCRIPTIONS :		
1) Entrance Fees	0	0
2) Annual Fees/Subscriptions	0	0
3) Seminar/Program Fees	0	0
4) Consultancy Fees	0	0
5) Others (Specify)	0	0
TOTAL	**0**	**0**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 15 - INCOME FROM INVESTMENTS :		
(Income on Invest from Earmarked/Endowment Funds transferred to Funds)		
1) Interest:		
a) On Govt. Securities	0.00	0.00
b) Other Bonds/Debentures	0.00	0.00
2) Dividends:		
a) On Shares	0.00	0.00
b) On Mutual Fund Securities	0.00	0.00
3) Rents	0.00	0.00
4) Others (Specify) STDRs	5685649.00	18375260.00
TOTAL	**27138910.00**	**23220086.00**
TRANSFERRED TO EARMARKED/ENDOWMENT FUNDS		

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 16 - INCOME FROM ROYALITY, PUBLICATION ETC. :		
1) Income from Royality	0	0
2) Income from Publications	0	0
3) Others (Specify)	0	0
TOTAL	**0**	**0**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 17 - INTEREST EARNED :		
1) On Term Deposits		
a) With Schedule Banks	1278536.00	1284265.00
b) With Non-Scheduled Banks	0.00	0.00
c) With Institutions	0.00	0.00
d) Others	0.00	0.00
2) On Saving Accounts		
a) With Schedule Banks	507346.00	106041.00
b) With Non-Scheduled Banks	0.00	0.00
c) post Office Savings Accounts	0.00	0.00
d) Others	0.00	0.00
3) On Loans		
a) Employees/Staff		
b) Others	0.00	0.00
4) Interest on Debtors and Other Receivables	0.00	0.00
TOTAL	**1785882.00**	**1390306.00**
Note :- Tax deducted at source to be indicated		

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

SCHEDULE 18 - OTHER INCOME	Current Year	Previous Year
1) Profit on Sale/disposal of Assets:	0.00	0.00
a) Owned assets	0.00	0.00
b) Assets acquired out of grants, or received free of cost	0.00	0.00
2) Export Incentives realized	0.00	0.00
3) Fees for Miscellaneous Services	0.00	0.00
4) Miscellaneous Receipts		
5) Other Receipts		
Sundry Receipts	4568979.00	7090257.00
Application Fee	15125.00	17500.00
Sales Of Tender Forms	90500.00	10500.00
Licence Fee	54880.00	55200.00
Interest On Computer Advance,Conveyance Advance And HBA	6171.00	19018.00
Leave Salary-Pension Contribution	52836.00	44030.00
Provident Fund Salwage	0.00	0.00
Free.Gifts-Donations	0.00	0.00
TOTAL	**4788491.00**	**7236505.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

SCHEDULE 19 - INCREASE/(DECREASE) IN STOCK OF FINISHED GOODS & WORK IN PROGRESS :	Current Year	Previous Year
a) Closing stock		
-Finished Goods	0	0
-Work-in-progress	0	0
Total (a)	0	0
b) Less: Opening stock		
-Finished Goods	0	0
-Work-in-progress	0	0
Total (b)	**0**	**0**
NET INCREASE/(DECREASE) [a-b]	0	0

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 20 - ESTABLISHMENT EXPENSES :		
a) Salaries and Wages	45425480.00	53877441.00
b) Allowances and Bonus	62477804.00	58836726.00
c) Contribution to Provident Fund	4407988.00	2247900.00
d) Contribution to Other Fund (NPS)	3162884.00	2767432.00
e) Staff Welfare Expenses - Medical charges	2219993.00	2101652.00
f) Expenses on Employees Retirement and Terminal Benefits	4725959.00	0.00
g) Others (specify) - Staff leased House	0.00	0.00
TOTAL	**122420108.00**	**119831151.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 21 - OTHER ADMINISTRATIVE EXPENSES :		
a) Purchases	33249114.00	55705243.00
b) Electricity and power	22793626.00	21498750.00
c) Water charges	1662990.00	903057.00
d) Insurance	97432.00	106035.00
e) Repairs and maintenance	16694133.00	11702293.00
f) Rent, Rates and Taxes	21280489.00	30557063.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 21 - OTHER ADMINISTRATIVE EXPENSES :		
g) Vehicles Running and Maintenance	1386497.00	1176998.00
h) Postage, Telephone and Communication Charges	2229539.00	4578419.00
i) Printing and Stationary	1344515.00	1748631.00
j) Travelling and Conveyance Expenses	5982640.38	9363448.00
k) Expenses on Seminar/Workshops	78900.00	219573.00
l) Subscription Expenses	54500.00	50894.00
m) Expenses on Fees	94777.00	34246.00
n) Auditors Remuneration	39500.00	62126.00
o) Hospitality Expenses	813197.00	952328.00
p) Professional Charges	1389456.00	3686097.00
q) Advertisement and Publicity	1779225.00	472477.00
r) Bank Charges	5297.00	26600.00
s) Security & Cleaning Contract Charges	24811357.00	21601902.00
t) Training Course /Symposia	9600.00	20600.00
u) Other Contingencies	5202138.00	9373811.00
v) Liveries & Blankets	0.00	127754.00
w) Other Research Expenses	23260806.00	38760374.00
x)Office Books	11666.00	1040.00
y)Over Heads	0.00	0.00
TOTAL	**164271394.38**	**212729759.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 22 - EXPENDITURE ON GRANTS, SUBSIDES, ETC.		
a) Grants given to Institutions/Organisations	0.00	0.00
b) Subsidies given to Institutions/Organisations	0.00	0.00
TOTAL	**0.00**	**0.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2017

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 23 - INTEREST		
a) On Fixed Loans	0.00	0.00
b) On Other Loans (including Bank Charges)	0.00	0.00
c) Others	0.00	0.00
TOTAL	**0.00**	**0.00**

Schedule 24: Significant Accounting Policies & Schedule 25: Contingent Liabilities & Notes on Account for the period ended 31/03/2017

1. **Method of Accounting:**

   a. The accounting system adopted by the organization is on "accrual basis".

   b. The organization has been getting plan Grant-In-Aid under the "Non-recurring" & "Recurring" heads.

2. **Revenue recognition:**

   Income comprises of Grant-in-Aid, Internal Resources through services and interest from short term deposits. Income accounted on the basis of the Cash/DD/Cheques/Cr notes/ on line transfers received.

3. **Fixed Assets:**

   (a) Fixed assets are stated at cost. Cost includes freight, duties, and taxes etc.,

   (b) Depreciation: Depreciation Account on Fixed Assets has since been prepared at the rate prevailing to the concerned Fixed Assets as specified in the Income Tax Act, 1961 on Written Down Value Method of Depreciation.

   (c) Capital work in progress has been entered to the extent of the last running account bills paid.

   (d) Realization on sale of obsolete/surplus fixed assets which is not required for the purpose of research activities are adjusted against capital cost.

4. **Inventories:**

   All purchases of chemicals, glassware and other consumables have been charged to consumption at the time of purchase.

5. **Foreign Currency transactions:**

   Foreign Currency transactions are recognized in the books at the exchange rates prevailing on the date of transaction.

6. **Interest earned on Deposits:**

   Interest Accrued on Deposits with RITES for financial year 2015-16 has not been received till 31st March 2017.

7. **Investments:**

   Investments in STDR's are stated at book values.

8. **Advances**

   It is observed from the objection book register that advances to suppliers for consumables & Equipments are to be reconciled and adjustment entries are to be passed in the books of accounts.

9. The previous year balances have been regrouped / rearranged, wherever necessary.

Director CDFD	Accounts Officer CDFD	for B Purushottam & Co Chartered Accountants Reg.No.002808S

[CH SATYANARAYANA]
M.No.019092

Place: Hyderabad

Date: 20/09/2017

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

CLARIFICATION ON NOTES ON ACCOUNTS: 2016-17

- **Notes on Accounts 1 to 2 & 4 to 6: Method of Accounting/ Revenue recognition/Fixed Asset/Inventories/ Foreign Currency transactions/Investments**:

  These are all only informatory items.

- **Notes on Accounts 3**: Fixed Assets:

  Depreciation has been calculated on Written down Value method and at the rates prevailing to the concerned Fixed Asset as specified on the Income Tax Act, 1961 and set off against the Grant-in-Aid (non-recurring). The details of the Depreciation on Fixed Assets are at Schedule -8 is an integral part of the financial statements

- **Notes on Accounts 6: Interest earned on Deposits**:

  This issue has been pursued with concerned authorities (M/s RITES) and the same will be accounted during the financial year 2017-18.

- **Notes on Accounts 7: Advances**:

  The observation of the audit has been noted. The action has already been initiated to reconcile the objection book register.

T ABHISHEK
Account Officer
CDFD

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-I

Details of Closing balances of various Earmarked / Endowement Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2017

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
-13755933	COE1	COE1	-9650327
-25772516	COE2	COE2	-23954089
0	others	Others	2028298
-630047	P-03	“Transgenesis and Genetic basis of Pathogen Resistance in the Silkworm, Bombyx Mori	-630047
244305	P-09	“NMITLI Project on – Latent M.Tuberculosis: New targets, Drug delivery systems, Bio enhancers & Therapeutics”	244305
-28332	P-10	“Role of upstream sequence elements in Hyper activation of transcription from Baculovirus polyhedrin gene promoter”	-28332
-576590	P-100	Effect of raective oxygen species on T-Cell immune response: An approach to understand the molecular mechanism of immunosuppression during tuberculosis - National Bioscience Award	-576590
-27922	P-102	Understanding the role of Mycobacterium tuberculosis heat shockprotein 60 as Th1/Th2 immuno modular	-27922
-300000	P-103	National Bioscience Award - Regulation of mast cell signaling, apoptosis and surface receptors	-300000
-1289897	P-104	Virtual Centre of Excellence on Epigenetics	-1289897
-862685	P-105	Cloning, Characterization and analysis of chromosomal rearrangements in human genetic disorders	-862685
366575	P-107	IYBA Project - Mechanism and role of bacterial cell-cell signaling molecules in plant defense response	327575
-454643	P-108	Establishment of EBV transformed cell lines from families with rare genetic disorders	-454643
767943	P-109	Molecular dissection of PI3-Kinase/Akt pathway by suing proteomics based approach: A study to identify novel potential oncogenes and tumor suppressors	-362393
-191391	P-110	India-Japan research project title"Identification and analysis of sex determining genes in silkmoths"	-19391
-450859	P-114	Evaluating the Calcineurin-NFAT Pathway and its regulators superoxide dismutase (SOD) AND RCAN1 (regular of Calcineurin) Down Syndrome	-450859
-1251366	P-116	DBT-India and AIST - Japan : Understanding molecular mechanisms controlling dual role of Ras, Sirtuins and CARF in relation to cellular proliferation and senescence: Novel Strategy for developing cancer therapeutics	-1251366
-2892	P-119	Analysis of DNA copy number alterations in esophaeal cancer	-2892
-769484	P-120	Effect of reactive oxygen species on macrophage signalosome: impact on antigen presentation functions and T Cell priming responses	-769484
-1130866	P-121	Identification and characterization of PTEN regulators	-1130866
2951109	P-122	Understanding the role of Hox genes in anterior-posterior axis determination of the central nervous system	21124
771699	P-123	Establish a Max Planck Partner Group for Genetic Diversity Studies at CDFD	1440687
-748411	P-124	Preparation and characterization of peroxometal compounds and studies and their biological significance in cellular signalling	-748411
209670	P-126	Rho-dependent transcription termination machinery: mechanism of action	160270
1895283	P-127	Systematic studies on the functional network of phosphatases in cell life and death	0
-158488	P-128	Mechanism of iron acquisition and iron homeostasis in an opportunistic human pathogen Candida glabrata	-158488
3947	P-13	“Programme to delineate gene functions in the post – genomics era by a systematic two gene knockout method”	3947
869	P-130	Comparative genetic analysis of sex chromosomes and sex determining genes in silkmoths	-142258
398632	P-131	Structural and functional studies of Acyl CoA Binding proteins from plasmodium falciparum	398632
-12199	P-132	Characterization of tumor suppressor function of ARIDIB, a component of the human SWI/ SNF chromatin remodelling complex	-12199
-702990	P-133	Investigating the role of Hox gene deformed in central nervous system patterning in Drosophila melanogaster	-1324223
-77061	P-134	Exploration of wild silk moth biodiversity in Manipur and their genetic characterization using molecular markers	-77061

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-I

Details of Closing balances of various Earmarked / Endowement Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2017

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
-336135	P-135	Sys TB: A Network Program for Resolving the Intracellular Dynamics of Host Phthogen Interaction in TB Infection	-1118756
-196001	P-136	Raf Kinase - a key target for modem-day theraphy against tumors	-196001
-1500300	P-138	Co-evaluation of Dnmt3l and Genomic imprinting	-1451500
20000	P-139	Evaluating the role of Sirtuins and epigenetic changes during cellular senescense in context of p53 status	20000
-608652	P-140	Development of baculovirus resistant silkworms strains through synthetic miRNA based knockdown of essential viral genes	-608652
-125000	P-141	Evaluating the functional role of PTEN interacting proteins in cell survival signaling and tumor suppression	-125000
-81861	P-142	Identification of H3K4 TRI Demethylase involved in erasing H3K4 trimethylation marks at E2F Responsive promoters	-81861
-1381684	P-143	Microarray based characterisation of squamous cell carcinoma of the tongue occuring in non smokers	-719139
122130	P-144	Tri-National Training Program for Psychiatric Genetics	122130
3222	P-145	"H3K4 HMT family regulatescell cycle progression ”	3222
59533	P-146	"Role of MLL in ribosomal RNA transcription”	59533
-272874	P-147	"The Effect of Parental Education, Ethics of Research Participation and Array Comparative Genomic Hybridization in Subjects with Mental Retardation (MR) and /or Autism ”	-272874
-59917	P-149	"Role of SUMOylation in the pathobiology of Candida Glabrata ”	-73001
375851	P-151	"Human Exome Sequencing to Identify Novel Genes for Medelian Disorders ”	199137
-30814	P-152	"Global transcriptomics of sex specific spilicing ”	-1123979
-64305	P-153	"An attractive and promising stragegy for early cancer diagnosis through the assembly of the human cancer volatome"	1161773
13510	P-154	"Rational design, synthetic strategies for developing organometallic anticancer compounds based on organotin and organoiron”	-434393
335194	P-155	"Studies on thecellular roles of calcium signalling proteins in Neurospora crassa”	335194
239949	P-156	"Targeting microbial quorum sensing to demonstrate potential application of cell-cell signaling molecules from Xanthomonas group of plant pathogen in diesease control”	-605123
-1361799	P-157	"Identification of novel antifungal drug and delineation of drug resistance mechanisms in an opportunistic human fungal pathogen Candida glabrata”	124009
-2575346	P-158	"Modulation of host immune responses by a PPE Protein of Mycobacterium tuberculosis: Understanding its role in host - pathogen cross-talk”	-168374
-300000	P-159	"Gene Targeting of microbial isolates to demonstrate potential plant growth promoting (PGP) traits by third generation sequencing”	-300000
-41667	P-160	"Understanding the role of novel adhesins of Xanthomonas oryzae PV orzae in Virulence and colonization in Rice”	-147180
-1021767	P-162	”Characterization and design of inhibitors of Mycobacterium tuberculosis transcription”	-464167
678659	P-163	Unravelling new functions for the H-NS family of proteins in Gram-negative bacterial pathogens	1530338.17
-29200	P-164	"A Yeast based screen for discovery of novel sirtuin inhibitors as anticancer agents	-29200
1567830	P-165	"Identification and functional characterization of immune response genes in silkmoths ”	862906
35696	P-166	"Sequencing analysis of transcriptome variants in early-onset sporadic rectal cancer ”	-368609
569787	P-167	"To elucidate the role of MLL complex in epigenetic specification of centromeres ”	780652
0	P-168	"A Search for nucleus -limited genes in Neurospora ”	-161318
16915	P-169	"Implementation of 3 year DNB Program in Medical Genetics by Department of Biotechnology in colloboration with National Board of Examination ag SGHR, NIBMG&CDFD	-332017
-687887	P-17	“Studies on inosital-phosphate synthesis – a novel enzyme from Mycobacterium tuberculosis H37RV” – Transfer from IMTECH, Chandigarh	-687887
-659867	P-170	"Women Scientist Scheme ""Identification and character of deregulated micro RNAs in defined sub-set of early onset sporadic rectal cancer patients using transcriptome sequencing"	-383863

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-I

Details of Closing balances of various Earmarked / Endowement Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2017

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
211423	P-171	"Role of vesicle-mediated transport and chromatin remodelling in the virulence of Candida glabrata"	-1237535
111850	P-172	"Molecular Characterization of early onset sporadic rectal cancer"	40020
487953	P-173	"Development and application of a next generation sequencing approach for molecular genetic analysis of lysosomal storage disorders"	1672130
520542	P-174	"Is non-canonical Wnt signalling a major player in early-onset sporadic rectal cancer"	209406
-1432672	P-175	"Multi Centri Collaborative study of the Clinical, Biochemical and Molecular Characterization of Lysosomal storage disorders in India - The initiative for research in Lysosomal Storage Disorders"	-121669
200103	P-176	International Atomic Energy Agency "	208017
-197394	P-177	"Morphological and molecular taxonomy of the Phlebotomus argendtipes species complex in relation to transmission of Kala-azar in India""	-119970
0	P-178	"Understanding differential signaling via toll like receptor-2: A proteomics approach	184199
-50000	P-179	"Quality Assurance Programme for Molecular and Prenatal Diagnosis of Hemoglobin Opathies"	50000
-274286	P-18	"Mapping of receptor binding site on the Eythrocyte binding of malaria parasyte"	-274286
117886	P-180	"Collaborative studies on genomic diversity among bombycoid silkmoths in Asia	63384
1744000	P-181	"To conduct multilocational field trails on transgenic BmNPV resistant silkworm strains to establish their efficacy and generate data for their regulatory approval"	1223096
-277500	P-182	"Ramalingaswami Fellowship "	533274
0	P-183	""Prevalence and predictors of vitamin B12 deficiency: genetic associations for low vitamin B12 levels-multi-center a pan India study",	-1091800
957742	P-184	"Computational Approaches to Understanding Peptide- Protein Interactions involved in the Regulatory Events in the Cell "	123065
1632207	P-185	"Investigating potential of mycobacterium tuberculosis protein PPE18 encapsulated nanoparticle as therapy for microbial sepsis"	1271410
2410000	P-186	"In vivo corss-talks between Rho-dependent transcription termination and other biological processes"	449029
1368000	P-187	"Understanding the mechanism of induction of innate immunity in plants by the Xanthomonas Diffusible signal factor (DSF)"	1282677
1450000	P-188	"Identification of Novel Genes for Intellectual Disability"	832894
16858467	P-189	"Characterization of glycosylphosphatidylinositol-linked aspartyl proteases in Candida glabrata: role in pathosgenicity"	17423746
1100000	P-190	"Exploring mycobacteriophages to source novel factors / regulators of bacterial transcription machinery	245026
0	P-191	"Human Frontier Science Program Reseearch Grant - A comprehensive approach towards the chemistry & biology of polyphosphate: the forgotten biopolymer"	5718535
0	P-192	"Design of peptide inhibitor(s) for the bacterial trancription terminator Rho, a potent drug target"	458917
0	P-193	"Screening for male infertility markers in the human Yq12 heterochromatic block"	1001347
0	P-194	"Mechanisms and regulation of iron transportin the pathogenic yeast Candida glabrata"	210034
0	P-195	"Molecular and biophysical characterization of the ESAT-6: 2M complex and its effect on intracellular iron concentration and macrophage anti-mycobacterial effector responses"	872204
0	p-196	"Exploring the volatome of noncommunicable diseases as a promising, innovative and integrating approach for its rapid diagnostics"	1164020.7
0	P-197	"National Post Doctoral Fellowship "	583730
0	P-198	"Whole Genome Sequencing for characterization of novel genes and de novo balanced chromosomal rearrangements in human genectic disorders"	2493600
0	P-199	"Investigating cellular processes and pathways controlled by phosphatases"	4013536
-1888111	P-20	"Genomic Micro array R&D Programmes on infectious diseases and Neurological Disorders"	-1888111
0	P-200	"Characterization of divergent functions of ARID1A and ARID1B: the two alternative DNA binding constituents of the human SWI/SNF chromatic remodelling complex	1806199

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-I

Details of Closing balances of various Earmarked / Endowement Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2017

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
0	P-201	"Defining the functions of MLL in mitosis ”	1241000
0	P-202	"To decipher the role of MLL Complex in the process of cytokinesis”	603000
0	P-203	"Investigation of a potential novel function of fission yeast sirtuin family histone deacetylase Hst4 in regulation of DNA replication”	1186706
-34495	P-23	“Development of PCR base assays for detection of GMO S”	-34495
-529111	P-25	“Functional studies of Human Immuno - deficiency Virus Type– 2 (HIV-2) Viral protien X (VPX)”	-529111
-79533	P-26	Occurrence of Mutations in Non dividing cells of Escherichia Coli”	-79533
-37624	P-28	Baculovirus resistance in transgenic silkworms	-37624
-310302	P-29	“Development of Hospital Surveillance system by advanced diagnostics method & Molecular DNA fingerprinting techniques”	-310302
-234000	P-33	“Molecular and Epidemiological characterisation of cryptosporidium – An enteric protozoon parasite”	-234000
26334	P-34	“Molecular analysis of lepidopteran – specific immune protiens from silkmoths”	26334
-283883	P-35	“Identification, Characterization and Physical mapping of Z-Chromosome linked genes of the silk worm, Bombyxmori”	-283883
2073896	P-36	“Development of Artificial retina using Bacterio rhodospin and genetically engineered analogues ”	2073896
-4058	P-40	“Antioxidants as a potential immuno adjuvant in anti tuberculosis immunotherapy”	-4058
1873605	P-41	“Construction, characterization and analysis of expressed sequences from silkworm ”	1873605
-457538	P-44	“Understanding of role of Ras and NO / iNOS signalling in promotion of hepatocellular carcinomas with persistent HBV infection”	-457538
-1586965	P-47	Research cum Training for DRDO Programme	-1586965
151826	P-48	‘Molecular characterization of human liver stem cells for use in the treatment of hepatic diseases’.	151826
1041952	P-49A	International Atomic Energy Agency (IAEA)	1041952
-284065	P-51	“Understanding the mechanism of doxorubicin resistance in breast cancer celline MCF-7”	-284065
-1231118	P-52	“Nucleo Cytoplasmic transport of HIV – 1 Vpr”	-1231118
-37877	P-54	“Study of viability of Mycobacterium leprae in clinical samples and possibility of its presence in the environment using nucleic acid amplification techniques.”	-37877
224	P-55	“Identification of DNA Markers for baculovirus resistance in silkworm, Bombyx mori”	224
-1231164	P-56	“Genetics of transcription-replication interplay and of stress adaptation in bacteria”	-1231164
-2215024	P-59	“An integrated Approach towards understanding the biology of Mycobacterium tuberculosis: Genetic, biochemical, immunological and structural analyses.”	-2215024
482124	P-60	“National Database of Prevalent Genetic Disorders in India: Development, Curation and Services”	482124
-280000	P-61	“Dissection of a novel phenotype of lethal accumulation of potassium in Escherichia coli mutants defective in thioredoxin/thioredoxin reductase and nucleoied protein H-NS”	-280000
-278928	P-62	“HIV – 1 Pathogenesis: Role of Integrase in Reverse Transciption and Nuclear Transport of Viral Genome”	-278928
-773874	P-63	“Upgradation of the existing computing infrastructure at the Bioinformatics facility at CDFD”	-773874
-158	P-64	Biotechnology for Leather: Towards cleaner processing phase-II	-158
-582647	P-65	“Molecular, genetic and functional analysis of the chromosomal plasticity region of the gastric pathogen Helicobater pylori”	-582647
22811205	P-65A	APEDA-CDFD Centre for Basmati DNA Analysis	23733305
-681246	P-66	Human Epigenome Variation: Analysis of CpG island methylation in chromosomes 18 and Y, and in some Hox, insulin signaling and chromatin reprogramming genes	-681246
-113545	P-67	Identification of novel Esophageal Squamous cell carcinoma (ESCC) genes by using a combination of array-based CGH and gene expression micro arrays	-113545
-59874	P-68	Identification of High risk individual with pre-cancerous states of esophageal cancer.	-59874

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-I

Details of Closing balances of various Earmarked / Endowement Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2017

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
-21336	P-70	Identification of disease causing mutations in familial hypertrophic cardiomyopathy (FHC) patients from Andhra Pradesh	-21336
-1421653	P-72	Nuances of non coding DNA near insulin-responsive genes.	-1421653
-857136	P-73	Identification and characterization of pancreatic cancer genes located within novel localized cpy number alterations	-857136
-10840	P-75	Preparing blueprint for the macromolecular crystallography beamline at Indus-II synchrotron source	-10840
-50234	P-76	A study of molecular markers in childhood autism with special references to nuclear factors - α APPA B	-50234
124277	P-77	Functional characterization of Mycobacterium tuberculosis PE/PPE proteins having SH3 binding domain : Understanding their role in modulating macrophage functions	124277
1304	P-78	Task force- IMD Newborn screening for Congenital Hypothyroidism & Congenital Adrenal Hyperplasia: A multicentric study	1304
-105086	P-79	Understanding the role of AGE proteins in inducing inflammatory responses and its regulation	-105086
-608222	P-80	Referral centre for detection of genetically modified foods employing DNA-based markets	-608222
143470	P-81	Reconstructing Cellular Networks: Two-component regulatory systems	143470
2620	P-81A	Financial assistance for award of J C Bose Fellowship to Dr J Gowrishankar	850453
-369021	P-82	Functional genomic analysis of Candida Glabrata-macrophage	-369021
-1155594	P-83	Prokaryotic Transcription termination factor, Rho: Mechanism of Action and Biology	-1155594
-1150	P-84	Preparing for vaccine efficacy trials: Baseline epidemiology, improved diagnosis, markers of protection and phase I/II trials	-1150
-106479	P-84A	Human epigenetic to the rescue of human identification process: Enriching human DNA from DNA mixture employing antibodies directed against 5-methylcytosine followed by whole genome amplification	-106479
-1118755	P-85	IdeR associated gene regulatory network in mycobacteria	-1118755
-65698	P-87	Comparative genomics of wild silkmoths	-65698
-636286	P-90	Role of Yapsins in the Pathobiology of Candida Glabrata	-636286
-1098900	P-91	DMMT3L: epigenetic correlation with cancer	-1098900
268823	P-92	Swarnajayanti fellowship proj on "Designing transcription anti-terminators: a novel approach for making new inhibitors of gene expression"	268823
-611833	P-93/A1	Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against tuberculosis	-611833
-3038491	P-93/A2	Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against Mycobacterium tuberculosis	-3228626
483835	P-93B2 (II)	Evaluation of peptides / small molecules targeting ESAT-6:B2M interaction and PPE18-TLR2 interaction as potent anti tuberculosis therapautics	837745
-276552	P-97	Proteome-wide Analysis of Serine pyrophosphorylation by inositol pyrophosphates	-276552
-236042	P-98	Role of cell - cell signaling mediated by Diffusible signaling factor (DSF) in Xanthomonas virulence	-236042
-567516	P-99	Role of inositol Pyrophosphates in eukaryotic cell growth, proliferation and ribosomae biogenesis	-567516
-18029486.64			**5912596.23**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-II

Details of Closing balances of various Earmarked / Endowement Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2017

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
11713327	COE-I	COE for Genetics and Genomics of silkmoths	11713327
12450437	COE-II	DBT Centre of Excellence for Microbial Biology	12465940
600000	P-03	“Transgenesis and Genetic basis of Pathogen Resistance in the Silkworm, Bombyx Mori	600000
329289	P-07	“Collection of well characterised clinical samples and strains of Mycobacterium tuberculosis and development of molecular techniques for detection of drug resistant strains – Multi Centric Project”	329289
588400	P-09	“NMITLI Project on – Latent M.Tuberculosis: New targets, Drug delivery systems, Bio enhancers & Therapeutics”	588400
47400	P-10	“Role of upstream sequence elements in Hyper activation of transcription from Baculovirus polyhedrin gene promoter”	47400
17784	P-100	Effect of raective oxygen species on T-Cell immune response: An approach to understand the molecular mechanism of immunosuppression during tuberculosis - National Bioscience Award	17784
14378004	P-101	Role of inositol pyrophosphates in cell physiology: Investigating the biochemical significance of protein pyrophosphorylation - Senior Fellowship	14378004
698550	P-102	Understanding the role of Mycobacterium tuberculosis heat shockprotein 60 as Th1/Th2 immuno modular	698550
1000000	P-107	IYBA Project - Mechanism and role of bacterial cell-cell signaling molecules in plant defense response	1000000
3711105	P-109	Molecular dissection of PI3-Kinase/Akt pathway by suing proteomics based approach: A study to identify novel potential oncogenes and tumor suppressors	3911516
206800	P-111	Ramalingaswami Fellowship - Refractoriness mechanism in Mosquito: cracking molecular codes at genomic scale	206800
670095	P-113	Clinical and molecular genetic analysis of squamous cell carcinoma of the tongue	670095
475900	P-114	Evaluating the Calcineurin-NFAT Pathway and its regulators superoxide dismutase (SOD) AND RCAN1 (regular of Calcineurin) Down Syndrome	475900
4580214	P-115	Setting up of the National Institute of Animal Biotechnology	4580214
800000	P-116	DBT-India and AIST - Japan : Understanding molecular mechanisms controlling dual role of Ras, Sirtuins and CARF in relation to cellular proliferation and senescence: Novel Strategy for developing cancer therapeutics	800000
183443	P-118	Construction of regulatory networks in Mycobacterium tuberculosis through analysis of gene expression data and transcription regulation predictions. (MOU with Russian Foundation)	183443
529750	P-12	Molecular genetics and Functional genomics of M.Tuberculosis patient isolates in India	529750
12079632	P-122	Understanding the role of Hox genes in anterior-posterior axis determination of the central nervous system	13632420
1509561	P-123	Establish a Max Planck Partner Group for Genetic Diversity Studies at CDFD	1674539
758900	P-126	Rho-dependent transcription termination machinery: mechanism of action	758900
6776327	P-127	Systematic studies on the functional network of phosphatases in cell life and death	6776327
1770000	P-128	Mechanism of iron acquisition and iron homeostasis in an opportunistic human pathogen Candida glabrata	1770000
1334600	P-13	“Programme to delineate gene functions in the post – genomics era by a systematic two gene knockout method”	1334600
1008000	P-130	Comparative genetic analysis of sex chromosomes and sex determining genes in silkmoths	1008000
1054297	P-133	Investigating the role of Hox gene deformed in central nervous system patterning in Drosophila melanogaster	1054297
5500000	P-135	Sys TB: A Network Program for Resolving the Intracellular Dynamics of Host Phthogen Interaction in TB Infection	5500000
900000	P-137	Signaling pathways involved in down regulation of proinflammatory responses by PPE18 protein of Mycobacterium tuberculosis: Implication of PPE18 as therapeutics	900000
700000	P-138	Co-evaluation of Dnmt3l and Genomic imprinting	700000

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-II

Details of Closing balances of various Earmarked / Endowement Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2017

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
500000	P-139	Evaluating the role of Sirtuins and epigenetic changes during cellular senescense in context of p53 status	500000
5163243	P-14	“Comparitive and functional genomics approaches for the identification and characterization of genes responsible for multi drug resistance of mycobacterium tuberculosis”	5163243
500000	P-140	Development of baculovirus resistant silkworms strains through synthetic miRNA based knockdown of essential viral genes	500000
650000	P-142	Identification of H3K4 TRI Demethylase involved in erasing H3K4 trimethylation marks at E2F Responsive promoters	650000
1868000	P-145	"H3K4 HMT family regulatescell cycle progression	1868000
1000000	P-146	"Role of MLL in ribosomal RNA transcription	1000000
469000	P-149	"Role of SUMOylation in the pathobiology of Candida Glabrata	469000
6000000	P-15	“The Helicobacter Pylori genome programme – Genome sequencing, functional analysis and comparitive genomics of the strains obtained from Indian patients”	6000000
0	P-152	Global transcriptomics of sex specific spilicing	17421
3000000	P-153	"An attractive and promising stragegy for early cancer diagnosis through the assembly of the human cancer volatome""	3000000
132495	P-154	"Rational design, synthetic strategies for developing organometallic anticancer compounds based on organotin and organoiron	132495
-4634	P-156	"Targeting microbial quorum sensing to demonstrate potential application of cell-cell signaling molecules from Xanthomonas group of plant pathogen in diesease control”	-4634
992265	P-157	"Identification of novel antifungal drug and delineation of drug resistance mechanisms in an opportunistic human fungal pathogen Candida glabrata”	992265
343121	P-158	"Modulation of host immune responses by a PPE Protein of Mycobacterium tuberculosis: Understanding its role in host - pathogen cross-talk”	343121
1814901	P-16	NMITLI Project on – Latent M.Tuberculosis: New targets, Drug delivery systems, Bio enhancers & Therapeutics	1814901
160082	P-165	Identification and functional characterization of immune response genes in silkmoths	160082
2000000	P-166	Sequencing analysis of transcriptome variants in early-onset sporadic rectal cancer	2000000
560757	P-167	"To elucidate the role of MLL complex in epigenetic specification of centromeres	560757
396000	P-168	"A Search for nucleus -limited genes in Neurospora ”	396000
295560	P-171	Role of vesicle-mediated transport and chromatin remodelling in the virulence of Candida glabrata	295560
1388150	P-172	Molecular Characterization of early onset sporadic rectal cancer	1500000
0	P-184	Computational Approaches to Understanding Peptide- Protein Interactions involved in the Regulatory Events in the Cell"	166729
0	P-185	Investigating potential of mycobacterium tuberculosis protein PPE18 encapsulated nanoparticle as therapy for microbial sepsis	84421
0	P-186	In vivo corss-talks between Rho-dependent transcription termination and other biological processes	2180896
0	P-189	Characterization of glycosylphosphatidylinositol-linked aspartyl proteases in Candida glabrata: role in pathosgenicity	600000
0	P-190	Exploring mycobacteriophages to source novel factors / regulators of bacterial transcription machinery	50000
0	P-191	"Human Frontier Science Program Reseearch Grant - A comprehensive approach towards the chemistry & biology of polyphosphate: the forgotten biopolymer	39060
0	P-192	Design of peptide inhibitor(s) for the bacterial trancription terminator Rho, a potent drug target	2000000
0	P-194	Mechanisms and regulation of iron transportin the pathogenic yeast Candida glabrata	289966
244400	P-17	“Studies on inosital-phosphate synthesis – a novel enzyme from Mycobacterium tuberculosis H37RV” – Transfer from IMTECH, Chandigarh	244400

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-II

Details of Closing balances of various Earmarked / Endowement Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2017

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
344020	P-18	"Mapping of receptor binding site on the Eythrocyte binding of malaria parasyte"	344020
7246511	P-19	"Construction of Integrated RAPD, RFLP and Microsatellite linkage map of the Silkworm, Bombyx mori and its corelation with the Phenotypic linkage Map"	7246511
27331134	P-20	"Genomic Micro array R&D Programmes on infectious diseases and Neurological Disorders"	27331134
5300000	P-21	Development of Versatile, portable software for Bio-informatics	5300000
603747	P-22	"Biotechnology for leather – towards cleaner processing"	603747
375999	P-23	"Development of PCR base assays for detection of GMO S"	375999
0	P-24	Establishing a central facility on "Aerosol challenge in a containment facility"	0
600000	P-25	"Functional studies of Human Immuno - deficiency Virus Type– 2 (HIV-2) Viral protien X (VPX)"	600000
500000	P-26	Occurrence of Mutations in Non dividing cells of Escherichia Coli"	500000
260367	P-29	"Development of Hospital Surveillance system by advanced diagnostics method & Molecular DNA fingerprinting techniques"	260367
3746538	P-30	Transcription termination and anti termination in E-coli	3746538
3131006	P-31	Role of K-ras in Lung type II epithelial cells	3131006
4857938	P-36	"Development of Artificial retina using Bacterio rhodospin and genetically engineered analogues "	4857938
358470	P-39	"Computational analysis and functional characterization of mycobacterial protien(s) interacting with macrophase effector – APC functions – an approach to understand the molecular basis of pathogenesis of M. tuberculosis"	358470
49738	P-40	"Antioxidants as a potential immuno adjuvant in anti tuberculosis immunotherapy"	49738
3894086	P-41	"Construction, characterization and analysis of expressed sequences from silkworm "	3894086
9500000	P-42	"Structural and functional studies on Mycobacterium tuberculosis heat shock proteins".	9500000
11970000	P-43	"A generalized mechanism of transcription termination in prokaryotes: a quest for mechanism based transcription inhibitors for microbial pathogens".	11970000
3331377	P-45	Specialized chromatin structures as epigenetic imprints to distinguish parental alleles".	3331377
416137	P-46	"Effect of reactive oxygen species (ROS) on immune response : Relevance in immunosuppression and Pathogenesis"	416137
377567	P-47	Research cum Training for DRDO Programme	377567
1413292	P-48	'Molecular characterization of human liver stem cells for use in the treatment of hepatic diseases'.	1413292
198095	P-50	"Cervical cancer prevention by multiple strategies in rural community in Andhra pradesh"	198095
401738	P-51	"Understanding the mechanism of doxorubicin resistance in breast cancer celline MCF-7"	401738
1359129	P-52	"Nucleo Cytoplasmic transport of HIV – 1 Vpr"	1359129
1114495	P-53	Collaborative research project on molecular ecology and systematics	1114495
1163764	P-56	"Genetics of transcription-replication interplay and of stress adaptation in bacteria"	1163764
2131403	P-57	Improved genome annotation through a combination of machine learning and experimental methods: Plasmodium falciparum as a case study.	2131403
63000	P-58	"Indo-Malaysian collaboration in Bioinformatics: Development and maintenance of a web-portal hosting databases and tools of common interest"	63000
32974662	P-59	"An integrated Approach towards understanding the biology of Mycobacterium tuberculosis: Genetic, biochemical, immunological and structural analyses."	32974662
5720800	P-60	"National Database of Prevalent Genetic Disorders in India: Development, Curation and Services"	5720800
4308314	P-62	"HIV – 1 Pathogenesis: Role of Integrase in Reverse Transciption and Nuclear Transport of Viral Genome"	4308314
9637574	P-63	"Upgradation of the existing computing infrastructure at the Bioinformatics facility at CDFD"	9637574
600585	P-64	Biotechnology for Leather: Towards cleaner processing phase-II	600585

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Details of Closing balances of various Earmarked / Endowement Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2017

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
260000	P-65	“Molecular, genetic and functional analysis of the chromosomal plasticity region of the gastric pathogen Helicobater pylori”	260000
16924622	P-65A	APEDA-CDFD Centre for Basmati DNA Analysis	16924622
264430	P-66	Human Epigenome Variation: Analysis of CpG island methylation in chromosomes 18 and Y, and in some Hox, insulin signaling and chromatin reprogramming genes	264430
622747	P-67	Identification of novel Esophageal Squamous cell carcinoma (ESCC) genes by using a combination of array-based CGH and gene expression micro arrays	622747
235593	P-69	ICMR adhoc New Scheme Understanding the role of PE/PPE family of M tuberculosis in the activation of HIV virus type I long terminal repeat (HIV-ILTP)	235593
1012807	P-70	Identification of disease causing mutations in familial hypertrophic cardiomyopathy (FHC) patients from Andhra Pradesh	1012807
1573795	P-71	Referral Centre for Genetic fidelity testing of tissue culture raised plants	1573795
45653	P-72	Nuances of non coding DNA near insulin-responsive genes.	45653
1000000	P-74	Molecular basic of insect plant interactions in rice under the national fund for basic and strategic research in agriculture	1000000
33672	P-75	Preparing blueprint for the macromolecular crystallography beamline at Indus-II synchrotron source	33672
245266	P-76	A study of molecular markers in childhood autism with special references to nuclear factors - α APPA B	245266
1543605	P-77	Functional characterization of Mycobacterium tuberculosis PE/PPE proteins having SH3 binding domain : Understanding their role in modulating macrophage functions	1543605
0	P-78	Task force- IMD Newborn screening for Congenital Hypothyroidism & Congenital Adrenal Hyperplasia: A multicentric study	0
496826	P-79	Understanding the role of AGE proteins in inducing inflammatory responses and its regulation	496826
4192480	P-80	Referral centre for detection of genetically modified foods employing DNA-based markets	4192480
205073	P-81A	Financial assistance for award of J C Bose Fellowship to Dr J Gowrishankar	205073
1480220	P-82	Functional genomic analysis of Candida Glabrata-macrophage	1480220
912255	P-83	Prokaryotic Transcription termination factor, Rho: Mechanism of Action and Biology	912255
388583	P-83A	Understanding the mechanism of Azadirachtin-mediated cell signaling: role in anti-inflammation and anti-tumorigenesis	388583
44854	P-84	Preparing for vaccine efficacy trials: Baseline epidemiology, improved diagnosis, markers of protection and phase I/II trials	44854
1430573	P-84A	Human epigenetic to the rescue of human identification process: Enriching human DNA from DNA mixture employing antibodies directed against 5-methylcytosine followed by whole genome amplification	1430573
374630	P-89	Characterization of Mycobacterium tuberculosis transcription machinery and Bacteriophage metagenomics	374630
1376869	P-90	Role of Yapsins in the Pathobiology of Candida Glabrata	1376869
932151	P-91	DMMT3L: epigenetic correlation with cancer	932151
8500000	P-92	Swarnajayanti fellowship proj on "Designing transcription anti-terminators: a novel approach for making new inhibitors of gene expression"	8500000
2212534	P-93/ A1	Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against tuberculosis	2212534
913430	P-93/ A2	Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against Mycobacterium tuberculosis	913430

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

Annexure-II

Details of Closing balances of various Earmarked / Endowement Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2017

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
246320	P-95	Construction of regulatory networks in prokaryotes through protein: Protein interaction predictions and transcription regulation predictions. (MOU with Russian Foundation)	246320
1000000	P-97	Proteome-wide Analysis of Serine pyrophosphorylation by inositol pyrophosphates	1000000
2816418	P-98	Role of cell - cell signaling mediated by Diffusible signaling factor (DSF) in Xanthomonas virulence	2816418
2963482	P-99	Role of inositol Pyrophosphates in eukaryotic cell growth, proliferation and ribosomae biogenesis	2963482
313375529			**320849552**

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FOR THE YEAR ENDED 31st MARCH 2017

Annexure: A Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	I-Remittances	
6628892.00	TDS	4910125.00
9360877.00	Income Tax	8974333.00
2509.00	Works Tax	278372.00
1824286.00	LIC	1865076.00
208037.00	GSLI	251264.00
2806680.00	Public Provident Fund	1143660.00
584200.00	Professional Tax	506200.00
4374299.00	Service Tax	4987454.00
769380.00	Others (I-Remittances)	899765.00
533695.00	Health Insurance	462714.00
1462386.00	ECCS	2304183.00
803436.00	PPF EMPLOYER SHARE	381481.00
0.00	STAFF BENEVOLENT FUND	12569.00
29358677.00		**26977196.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2017

Annexure: B Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	Advance refunds/recovery/Adjst.	
531359.00	Advance for Expenses- purchases by Staff	734321.00
12309522.00	Chemicals [Advance]	6067820.00
97626.00	Computer Advance [Research Fellows]	70328.00
121892.00	Computer Advance [Staff]	168592.00
10273920.00	Consumables, glassware and Spares [Advance]	29685.00
0.00	Conveyance [Advance]	1800.00
64360.00	Conveyance Advance	78324.00
0.00	DA [Advance]	6638.00
38500.00	EMD	909438.00
15673247.00	Equipment [Advance]	5613268.00
171225.00	Festival Advance	138600.00
2450.00	GDA [Others]	0.00

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2017

Annexure: B Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
3357295.00	General Deposits And Advances	15950.00
121500000.00	Inter Bank Transfer	55200000.00
159000.00	Lab Security Deposit & Hostel Security Deposit	157200.00
824965.00	LTC [Advance]	690500.00
0.00	Miscellaneous Salary [Advance]	30843.00
36264.00	Others [Advances]	260129.00
0.00	Pay of Establishment [Advance]	53387.00
343759.00	Revolving Advance	456821.00
0.00	Security Deposit	952850.00
206595.00	TA Abroad [Advance]	199732.00
2481663.00	TA-DA-Hon within India [Advance]	1363959.00
12000.00	Trainee Security Deposit	11500.00
0.00	Water [Advance]	45000.00
2114275.00	Workshop & Conference	178928.00
170319917.00		**73435613.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2017

Annexure: C Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	Projects - Receipts	
8335000.00	COE1/CORE	8768000.00
638000.00	COE1/P-I	775000.00
491000.00	COE1/P-II	643000.00
1086000.00	COE1/P-III	1090000.00
650000.00	COE2-II/P-1	2100000.00
0.00	COE2-II/P-A	1061000.00
0.00	COE2-II/P-B	488000.00
0.00	COE2-II/P-C	1061000.00
0.00	COE2-II/P-D	496000.00
0.00	COE2-II/P-E	866000.00
0.00	COE2-II-Core	3447000.00
331000.00	COE-I/P-IV	442000.00
0.00	others	2028298.00

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS

FOR THE YEAR ENDED 31st MARCH 2017

Annexure: C Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
3868930.00	P-101	0.00
2479000.00	P-109	0.00
0.00	P-110	172000.00
8005983.00	P-122	2722184.00
1413360.00	P-123	1648000.00
0.00	P-125	0.00
0.00	P-126	0.00
6736571.00	P-127	663747.00
0.00	P-128	0.00
4024000.00	P-130	0.00
0.00	P-133	500000.00
2430700.00	P-135	0.00
-464025.00	P-137	0.00
196800.00	P-142	0.00
0.00	P-143	662545.00
1200000.00	P-145	0.00
500000.00	P-147	0.00
1420800.00	P-149	0.00
1756400.00	P-151	0.00
1931400.00	P-152	0.00
0.00	P-153	1787000.00
930000.00	P-154	0.00
1706000.00	P-156	0.00
0.00	P-157	1638000.00
0.00	P-158	2790992.00
687200.00	P-160	0.00
0.00	P-162	699600.00
1062777.00	P-163	1483389.00
2858334.00	P-165	0.00
574700.00	P-166	0.00
1500000.00	P-167	900000.00
1000000.00	P-168	0.00
0.00	P-169	2535600.00
0.00	P-170	1100000.00
1200000.00	P-172	1000000.00

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2017

Annexure: C Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
699782.00	P-173	2107380.00
500000.00	P-174	0.00
0.00	P-175	2214648.00
0.00	P-176	207044.00
225000.00	P-177	225000.00
1000000.00	P-178	1000000.00
50000.00	P-179	100000.00
200000.00	P-180	0.00
1744000.00	P-181	0.00
0.00	P-182	2110000.00
1060000.00	P-184	0.00
1648000.00	P-185	0.00
2410000.00	P-186	1841600.00
1368000.00	P-187	0.00
1450000.00	P-188	0.00
16858467.00	P-189	5629854.00
1100000.00	P-190	0.00
0.00	P-191	7765092.00
0.00	P-192	3819000.00
0.00	P-193	1050000.00
0.00	P-194	500000.00
0.00	P-195	1285000.00
0.00	p-196	1281744.00
0.00	P-197	960000.00
0.00	P-198	2556000.00
0.00	P-199	4013536.00
0.00	P-200	1830000.00
0.00	P-201	1241000.00
0.00	P-202	603000.00
0.00	P-203	1186706.00
6869464.00	P-42	0.00
75039.00	P-43	0.00
1338000.00	P-65A	1004370.00
1300000.00	P-81A	1360000.00
0.00	P-93B2 (II)	737000.00
98445682.00		**90196329.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS

FOR THE YEAR ENDED 31st MARCH 2017

Annexure: D Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	Advances	
596022.00	Advance for Expenses- purchases by Staff	653985.00
4716258.00	Chemicals [Advance]	6024000.00
140000.00	Computer Advance [Research Fellows]	48400.00
120000.00	Computer Advance [Staff]	60000.00
4743564.00	Consumables, glassware and Spares [Advance]	1613098.00
1800.00	Conveyance [Advance]	0.00
120000.00	Conveyance Advance	60113.00
559000.00	EMD	463820.00
17952399.00	Equipment [Advance]	23750711.00
166500.00	Festival Advance	81000.00
105900.00	GDA [Others]	0.00
2541000.00	General Deposits And Advances	0.00
121500000.00	Inter Bank Transfer	55200000.00
129000.00	Lab Security Deposit & Hostel Security Deposit	135520.00
0.00	Liveries & Blankets [Advance]	27849.00
698550.00	LTC [Advance]	522400.00
0.00	Magzines [Advance]	854.00
3301.00	Membership Fee [Advance]	0.00
209077.00	Others [Advances]	407759.00
358000.00	Revolving Advance	442756.00
122500.00	Royalty & Consultancy	0.00
0.00	Scientific Workshops - Symposiums - Seminars [Advance]	8000.00
47800.00	Security Deposit	49140.00
362000.00	TA Abroad [Advance]	0.00
2215217.00	TA-DA-Hon within India [Advance]	1293660.00
0.00	Telephone [Advance]	50000.00
10500.00	Trainee Security Deposit	11000.00
11510.00	Transport maintenance [Advance]	0.00
0.00	Water [Advance]	45000.00
1114953.00	Workshop & Conference	826689.00
158544851.00		**91775754.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2017

Annexure: E Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	I-Remittances	
1462386.00	ECCS	2140898.00
205483.00	GSLI	259987.00
672784.00	Health Insurance	835000.00
9360458.00	Income Tax	8161043.00
1824286.00	LIC	1865076.00
769380.00	Others (I-Remittances)	708678.00
275566.00	PPF EMPLOYER SHARE	321745.00
585300.00	Professional Tax	508250.00
2525070.00	Public Provident Fund	1158742.00
4972523.00	Service Tax	4134084.00
0.00	STAFF BENEVOLENT FUND	0.00
5508643.00	TDS	5271099.00
0.00	Works Tax	174000.00
28161879.00		**25538602.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2017

Annexure: F Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	Projects - Expenditure	
8636177.00	COE1/CORE	6942349.00
693390.00	COE1/P-I	143520.00
664953.00	COE1/P-II	193527.00
1059200.00	COE1/P-III	225358.00
2216484.00	COE2-II/P-1	1655776.00
829368.00	COE2-II/P-A	1258535.00
810077.00	COE2-II/P-B	953955.00
225665.00	COE2-II/P-C	330000.00
200000.00	COE2-II/P-D	357000.00
362287.00	COE2-II/P-E	597400.00
7786755.00	COE2-II-Core	2547907.00
340400.00	COE-I/P-IV	107640.00
10728730.00	P-101	1.00
129389.00	P-104	0.00

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2017

Annexure: F Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
670116.00	P-107	39000.00
5062393.00	P-109	1130336.00
1169677.00	P-111	0.00
5443566.00	P-122	5652169.00
2043796.00	P-123	979012.00
232854.00	P-126	49400.00
4546772.00	P-127	2559030.00
81380.00	P-128	0.00
1473081.00	P-130	143127.00
-627804.00	P-132	0.00
1163107.00	P-133	1121233.00
2409567.00	P-135	782621.00
-96333.00	P-136	0.00
295449.00	P-137	0.00
147062.00	P-138	(48800.00)
205316.00	P-140	0.00
-1935.00	P-142	0.00
847180.00	P-143	0.00
302000.00	P-144	0.00
84535.00	P-145	0.00
374325.00	P-146	0.00
95035.00	P-147	0.00
464382.00	P-149	13084.00
779183.00	P-151	176714.00
1991314.00	P-152	1093165.00
705857.00	P-153	560922.00
947322.00	P-154	447903.00
1290886.00	P-156	845072.00
1566171.00	P-157	152192.00
1195688.00	P-158	384020.00
300000.00	P-159	0.00
937200.00	P-160	105513.00
84656.00	P-161	0.00
705303.00	P-162	142000.00
1436589.00	P-163	631709.83
4529.00	P-164	0.00
1620639.00	P-165	704924.00
2704642.00	P-166	404305.00

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2017

Annexure: F Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
1563993.00	P-167	689135.00
1788623.00	P-168	161318.00
1741193.00	P-169	2884532.00
937316.00	P-170	823996.00
1543024.00	P-171	1448958.00
2549897.00	P-172	1071830.00
796711.00	P-173	923203.00
479458.00	P-174	311136.00
922958.00	P-175	903645.00
0.00	P-176	199130.00
422394.00	P-177	147576.00
1000000.00	P-178	815801.00
100000.00	P-179	0.00
82114.00	P-180	54502.00
0.00	P-181	520904.00
277500.00	P-182	1299226.00
0.00	P-183	1091800.00
102258.00	P-184	834677.00
15793.00	P-185	360797.00
0.00	P-186	3802571.00
0.00	P-187	85323.00
0.00	P-188	617106.00
0.00	P-189	5064575.00
0.00	P-190	854974.00
0.00	P-191	2046557.00
0.00	P-192	3360083.00
0.00	P-193	48653.00
0.00	P-194	289966.00
0.00	P-195	412796.00
0.00	p-196	117723.30
0.00	P-197	376270.00
0.00	P-198	62400.00
0.00	P-200	23801.00
2045696.00	P-30	0.00
746453.00	P-31	0.00
4632179.00	P-42	0.00
760945.00	P-43	0.00
605714.00	P-45	0.00

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2017

Annexure: F Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
-63700.00	P-63	0.00
355200.00	P-65A	82270.00
0.00	P-71	0.00
1360000.00	P-81A	512167.00
13430.00	P-93/A2	190135.00
626165.00	P-93B2 (II)	383090.00
102743689.00		**66254246.13**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2017

Annexure: G Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	CDFD C.P.F ACCOUNT	
40638533.00	Opening Balance	44620022.00
Add:		
5518714.00	Employee subscription/ refunds	5192511.00
466203.00	Transfer from other departments	6986.00
0.00	Institute contribution (inc. Projects staff)	0.00
86454.00	Interest received	277728.00
2089882.00	Less Advances/withdrawals/Transfer/Adjst	6810005.00
44620022.00		**43287242.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2017

Annexure: H Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	LOANS AND ADVANCES	
270904.00	Advance for Expenses- purchases by Staff	190569.00
4310.00	Advances [Previous Years]	4310.00
2960132.00	Chemicals [Advance]	2916312.00
157373.00	Computer Advance [Research Fellows]	135445.00
325378.00	Computer Advance [Staff]	216786.00
12104705.00	Consumables, glassware and Spares [Advance]	13688118.00
1800.00	Conveyance [Advance]	0.00

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2017

Annexure: H Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
183288.00	Conveyance Advance	165077.00
6638.00	DA [Advance]	0.00
2550016.00	Equipment [Advance]	20687459.00
99450.00	Festival Advance	41850.00
421261.00	Health Insurance	793547.00
130351.00	Liveries & Blankets [Advance]	158200.00
2559549.00	LTC [Advance]	2391449.00
0.00	Magzines [Advance]	854.00
0.00	Miscellaneous Salary	95678.00
30843.00	Miscellaneous Salary [Advance]	0.00
66681.00	NPS Subscription	67325.00
22700.00	Office Equipment [Advance]	22700.00
5825681.00	Others [Advances]	5973311.00
0.00	Pay of Establishment	40821.00
53387.00	Pay of Establishment [Advance]	0.00
304569.00	Rent [Advance]	304569.00
32559396.00	Research Fellows-Associates	38436883.00
119707.00	Revolving Advance	105642.00
0.00	Scientific Workshops - Symposiums - Seminars [Advance]	8000.00
350893.00	Service Tax	0.00
90156.00	TA Abroad [Advance]	0.00
4390.00	TA-DA-Hon within India [Advance]	0.00
0.00	Telephone [Advance]	50000.00
25000.00	Trainee Security Deposit	24500.00
11510.00	Transport maintenance [Advance]	11510.00
0.00	Workshop & Conference	287622.00
61240068.00		**86818537.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2017

Annexure: I Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	DEPOSITS	
15649470.00	General Deposits And Advances	15633520.00
839427.00	GDA[Others]	839427.00
16488897.00		**16472947.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2017

Annexure: J Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	INVESTMENT A/C	
71098273.00	Investments	291098273.00
0.00	Other Investments	0.00
71098273.00		**291098273.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2017

Annexure: K Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	CDFD C.P.F INVESTMENT A/C	
33593376.00	Deposit with Banks	33741214.00
5666653.00	Employee subscription	5062115.00
9194308.00	Less Transfer To Bank A/C	6933088.00
30065721.00		**31870241.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-03: "Transgenesis and Genetic basis of Pathogen Resistance in the Silkworm, Bombyx Mori
P.I:
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	630047.00	Opening Balance	630047.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.0		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**630047.00**		**630047.00**
630047.00	Excess of Expenditure over Income	630047.00	0.00	Closing Balance	0.00
630047.00		**630047.00**	**630047.00**	**630047.00**	**630047.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-09: "NMITLI Project on – Latent M.Tuberculosis: New targets, Drug delivery systems, Bio enhancers & Therapeutics"
P.I: Dr Seyed E Hasnain
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
244305.00	Opening Balance	244305.00		Opening Balance	
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
244305.00		**244305.00**	**0.00**		**0.00**
0.00	Excess of Expenditure over Income	0.00	244305.00	Closing Balance	244305.00
2443050.00		**244305.00**	**244305.00**		**244305.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-10: "Role of upstream sequence elements in Hyper activation of transcription from Baculovirus polyhedrin gene promoter"

P.I: Dr M D Bashyam

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	28332.00	Opening Balance	28332.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	28332.00		0.00
28332.00	Excess of Expenditure over Income	28332.00	0.00	Closing Balance	**28332.00**
28332.00		**28332.00**	**28332.00**		**28332.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-13: "Programme to delineate gene functions in the post – genomics era by a systematic two gene knockout method"

P.I: Dr J Gowrishankar

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
3947.00	Opening Balance	3947.00	0.00	Opening Balance	
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
3947.00		**3947.00**	**0.00**		**0.00**
0.00	Excess of Expenditure over Income	0.00	3947.00	Closing Balance	3947.00
3947.00		**3947.00**	**3947.00**		**3947.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-17: "Studies on inosital-phosphate synthesis – a novel enzyme from Mycobacterium tuberculosis H37RV" – Transfer from IMTECH, Chandigarh
P.I: Dr Sekhar C Mande
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	687887.00	Opening Balance	687887.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**687887.00**		**687887.00**
687887.00	Excess of Expenditure over Income	687887.00	0.00	Closing Balance	0.00
687887.00		**687887.00**	**687887.00**		**687887.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-18: "Mapping of receptor binding site on the Eythrocyte binding of malaria parasyte"
P.I: Dr Akash Ranjan
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	274286.00	Opening Balance	274286.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**274286.00**		**274286.00**
274286.00	Excess of Expenditure over Income	274286.00	0.00	Closing Balance	0.00
274286.00		**274286.00**	**274286.00**		**274286.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-20: "Genomic Micro array R&D Programmes on infectious diseases and Neurological Disorders"
P.I: Dr Hasnain & Dr Bashyam
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	1888111.00	Opening Balance	1888111.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**1888111.00**		**1888111.00**
1888111.00	Excess of Expenditure over Income	1888111.00	0.00	Closing Balance	0.00
1888111.00		**1888111.00**	**1888111.00**		**1888111.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-23: "Development of PCR base assays for detection of GMO S"
P.I: Dr Nagaraju & Dr Niyaz Ahmed
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	34495.00	Opening Balance	34495.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**34495.00**		**34495.00**
34495.00	Excess of Expenditure over Income	34495.00	0.00	Closing Balance	0.00
34495.00		**34495.00**	**34495.00**		**34495.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-25: "Functional studies of Human Immuno - deficiency Virus Type– 2 (HIV-2) Viral protien X (VPX)"
P.I: Dr Mahalingam & Dr Mande
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	529111.00	Opening Balance	529111.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**529111.00**		**529111.00**
529111.00	Excess of Expenditure over Income	529111.00	0.00	Closing Balance	0.00
529111.00		**529111.00**	**529111.00**		**529111.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-26: Occurrence of Mutations in Non dividing cells of Escherichia Coli"
P.I:
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	79533.00	Opening Balance	79533.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**79533.00**		**79533.00**
79533.00	Excess of Expenditure over Income	79533.00	0.00	Closing Balance	0.00
79533.00		**79533.00**	**79533.00**		**79533.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-28: Baculovirus resistance in transgenic silkworms
P.I:
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	37624.00	Opening Balance	37624.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**37624.00**		**37624.00**
37624.00	Excess of Expenditure over Income	37624.00	0.00	Closing Balance	0.00
37624.00		**37624.00**	**37624.00**		**37624.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-29: "Development of Hospital Surveillance system by advanced diagnostics method & Molecular DNA fingerprinting techniques"
P.I: Dr K Prashanth
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	310302.00	Opening Balance	310302.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**310302.00**		**310302.00**
310302.00	Excess of Expenditure over Income	310302.00	0.00	Closing Balance	0.00
310302.00		**310302.00**	**310302.00**		**310302.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-33: "Molecular and Epidemiological characterisation of cryptosporidium – An enteric protozoon parasite"
P.I: Dr Radha Rama Devi
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	234000.00	Opening Balance	234000.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**234000.00**		**234000.00**
234000.00	Excess of Expenditure over Income	234000.00	0.00	Closing Balance	0.00
234000.00		**234000.00**	**234000.00**		**234000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-34: "Molecular analysis of lepidopteran – specific immune protiens from silkmoths"
P.I: Dr J Nagaraju
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
26334.00	Opening Balance	26334.00			
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
26334.00		**26334.00**	**0.00**		**0.00**
0.00	Excess of Expenditure over Income	0.00	26334.00	Closing Balance	26334.00
26334.00		**26334.00**	**26334.00**		**26334.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-35: "Identification, Characterization and Physical mapping of Z-Chromosome linked genes of the silk worm, Bombyxmori"
P.I: Dr J Nagaraju
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	283883.00	Opening Balance	283883.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**283883.00**		**283883.00**
283883.00	Excess of Expenditure over Income	283883.00	0.00	Closing Balance	0.00
283883.00		**283883.00**	**283883.00**		**283883.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-36: "Development of Artificial retina using Bacterio rhodospin and genetically engineered analogues "
P.I: Dr Sekhar C Mande
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
2073896.00	Opening Balance	2073896.00		Opening Balance	
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2073896.00		**2073896.00**	**0.00**		**0.00**
0.00	Excess of Expenditure over Income	0.00	2073896.00	Closing Balance	2073896.00
2073896.00		**2073896.00**	**2073896.00**		**2073896.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-40: "Antioxidants as a potential immuno adjuvant in anti tuberculosis immunotherapy"
P.I: Dr Sangita Mukhopadhyay
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	4058.00	Opening Balance	4058.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**4058.00**		**4058.00**
4058.00	Excess of Expenditure over Income	4058.00	0.00	Closing Balance	0.00
4058.00		**4058.00**	**4058.00**		**4058.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-41: "Construction, characterization and analysis of expressed sequences from silkworm "
P.I: Dr J Nagaraju
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
1873605.00	Opening Balance	1873605.00		Opening Balance	
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1873605.00		**1873605.00**	**0.00**		**0.00**
0.00	Excess of Expenditure over Income	0.00	1873605.00	Closing Balance	1873605.00
1873605.00		**1873605.00**	**1873605.00**		**1873605.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-44: "Understanding of role of Ras and NO / iNOS signalling in promotion of hepatocellular carcinomas with persistent HBV infection"
P.I: Dr Gayatri Ramakrishna
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	457538.00	Opening Balance	457538.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**457538.00**		**457538.00**
457538.00	Excess of Expenditure over Income	457538.00	0.00	Closing Balance	0.00
457538.00		**457538.00**	**457538.00**		**457538.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-47: Research cum Training for DRDO Programme
P.I: Dr Gowrishankar, Dr Mahalingam, Dr Mande, Dr Nagaraju, Dr Ni
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	1586965.00	Opening Balance	1586965.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**1586965.00**		**1586965.00**
1586965.00	Excess of Expenditure over Income	1586965.00	0.00	Closing Balance	0.00
1586965.00		**1586965.00**	**1586965.00**		**1586965.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-48: 'Molecular characterization of human liver stem cells for use in the treatment of hepatic diseases'.

P.I: Dr Sanjeev Khosla

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
151826.00	Opening Balance	151826.00			
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
151826.00		**151826.00**	**0.00**		**0.00**
0.00	Excess of Expenditure over Income	0.00	151826.00	Closing Balance	151826.00
151826.00		**151826.00**	**151826.00**		**151826.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-49A: International Atomic Energy Agency (IAEA)

P.I: J Nagaraju

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
1041952.00	Opening Balance	1041952.00			0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1041952.00		**1041952.00**	**0.00**		**0.00**
0.00	Excess of Expenditure Over Income	0.00	1041952.00	Closing Balance	1041952.00
1041952.00		**1041952.00**	**1041952.00**		**1041952.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-51: "Understanding the mechanism of doxorubicin resistance in breast cancer celline MCF-7"
P.I: Dr Sunil Kumar Manna
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	284065.00	Opening Balance	284065.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**284065.00**		**284065.00**
284065.00	Excess of Expenditure over Income	284065.00	0.00	Closing Balance	0.00
284065.00		**284065.00**	**284065.00**		**284065.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-52: "Nucleo Cytoplasmic transport of HIV – 1 Vpr"
P.I: Dr Mahalingam & Dr Manna
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	1231118.00	Opening Balance	1231118.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**1231118.00**		**1231118.00**
1231118.00	Excess of Expenditure over Income	1231118.00	0.00	Closing Balance	0.00
1231118.00		**1231118.00**	**1231118.00**		**1231118.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-54: "Study of viability of Mycobacterium leprae in clinical samples and possibility of its presence in the environment using nucleic acid amplification techniques."

P.I: Dr Niyaz Ahmed

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	37877.00	Opening Balance	37877.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**37877.00**		**37877.00**
37877.00	Excess of Expenditure over Income	37877.00	0.00	Closing Balance	0.00
37877.00		**37877.00**	**37877.00**		**37877.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-55: "Identification of DNA Markers for baculovirus resistance in silkworm, Bombyx mori"

P.I: Dr J Nagaraju

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
224.00	Opening Balance	224.00			
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
224.00		**224.00**	**0.00**		**0.00**
0.00	Excess of Expenditure over Income	0.00	224.00	Closing Balance	224.00
224.00		**224.00**	**224.00**		**224.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-56: “Genetics of transcription-replication interplay and of stress adaptation in bacteria”

P.I: Dr Gowrishankar & Dr K Anupama

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	1231164.00	Opening Balance	1231164.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**1231164.00**		**1231164.00**
1231164.00	Excess of Expenditure over Income	1231164.00	0.00	Closing Balance	0.00
1231164.00		**1231164.00**	**1231164.00**		**1231164.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-59: “An integrated Approach towards understanding the biology of Mycobacterium tuberculosis: Genetic, biochemical, immunological and structural analyses.”

P.I: Dr Hasnain, Dr Gowrishankar, Dr Mande, Dr Ranjan Sen

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	2215024.00	Opening Balance	2215024.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**2215024.00**		**2215024.00**
2215024.00	Excess of Expenditure over Income	2215024.00	0.00	Closing Balance	0.00
2215024.00		**2215024.00**	**2215024.00**		**2215024.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-60: "National Database of Prevalent Genetic Disorders in India: Development, Curation and Services"

P.I: Dr H A Nagarajaram

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
482124.00	Opening Balance	482124.00			
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
482124.00		**482124.00**	**0.00**		**0.00**
0.00	Excess of Expenditure over Income	0.00	482124.00	Closing Balance	482124.00
482124.00		**482124.00**	**482124.00**		**482124.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-61: "Dissection of a novel phenotype of lethal accumulation of potassium in Escherichia coli mutants defective in thioredoxin/thioredoxin reductase and nucleoied protein H-NS"

P.I: Dr Abhijit A Sardesai

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	280000.00	Opening Balance	280000.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**280000.00**		**280000.00**
280000.00	Excess of Expenditure over Income	280000.00	0.00	Closing Balance	0.00
280000.00		**280000.00**	**280000.00**		**280000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-62: "HIV – 1 Pathogenesis: Role of Integrase in Reverse Transciption and Nuclear Transport of Viral Genome"
P.I: Dr S Mahalingam
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	278928.00	Opening Balance	278928.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**278928.00**		**278928.00**
278928.00	Excess of Expenditure over Income	278928.00	0.00	Closing Balance	0.00
278928.00		**278928.00**	**278928.00**		**278928.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-63: "Upgradation of the existing computing infrastructure at the Bioinformatics facility at CDFD"
P.I: Dr Seyed E Hasnain
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	837574.00	Opening Balance	77387400
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	(63700).00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**773874.00**		**773874.00**
773874.00	Excess of Expenditure over Income	773874.00	0.00	Closing Balance	0.00
773874.00		**773874.00**	**773874.00**		**773874.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-64: Biotechnology for Leather: Towards cleaner processing phase-II
P.I: Dr J Gowrishankar
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	158.00	Opening Balance	158.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**158.00**		**158.00**
158.00	Excess of Expenditure over Income	158.00	0.00	Closing Balance	0.00
158.00		**158.00**	**158.00**		**158.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-65: “Molecular, genetic and functional analysis of the chromosomal plasticity region of the gastric pathogen Helicobater pylori”
P.I: Dr Ayesha Alvi
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	582647.00	Opening Balance	582647.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**582647.00**		**582647.00**
582647.00	Excess of Expenditure over Income	582647.00	0.00	Closing Balance	0.00
582647.00		**582647.00**	**582647.00**		**582647.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-65A: APEDA-CDFD Centre for Basmati DNA Analysis
P.I: Dr J Nagaraju
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
21828405.00	Opening Balance	22811205.00		Opening Balance	0.00
1338000.00	Grant In Aid	1004370.00	355200.00	Salaries - Manpower	69445.00
0.00		0.00	0.00	Consumables	12825.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
23166405.00		**23815575.00**	**355200.00**		**82270.00**
0.00	Excess of Expenditure Over Income	0.00	22811205.00	Closing Balance	23733305.00
23166405.00		**23815575.00**	**23166405.00**		**23815575.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-66: Human Epigenome Variation: Analysis of CpG island methylation in chromosomes 18 and Y, and in some Hox, insulin signaling and chromatin reprogramming genes
P.I: Dr Sanjeev Khosla
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	681246.00	Opening Balance	681246.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**681246.00**		**681246.00**
681246.00	Excess of Expenditure over Income	681246.00	0.00	Closing Balance	0.00
681246.00		**681246.00**	**681246.00**		**681246.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-67: Identification of novel Esophageal Squamous cell carcinoma (ESCC) genes by using a combination of array-based CGH and gene expression micro arrays

P.I: Dr M D Bashyam

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	113545.00	Opening Balance	113545.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**113545.00**		**113545.00**
113545.00	Excess of Expenditure over Income	113545.00	0.00	Closing Balance	0.00
113545.00		**113545.00**	**113545.00**		**113545.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-68: Identification of High risk individual with pre-cancerous states of esophageal cancer.

P.I: Dr Gayatri Ramakrishna

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	59874.00	Opening Balance	59874.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**59874.00**		**59874.00**
59874.00	Excess of Expenditure over Income	59874.00	0.00	Closing Balance	0.00
59874.00		**59874.00**	**59874.00**		**59874.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-70: Identification of disease causing mutations in familial hypertrophic cardiomyopathy (FHC) patients from Andhra Pradesh
P.I: Dr M D Bashyam
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	21336.00	Opening Balance	21336.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**21336.00**		**21336.00**
21336.00	Excess of Expenditure over Income	21336.00	0.00	Closing Balance	0.00
21336.00		**21336.00**	**21336.00**		**21336.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-72: Nuances of non coding DNA near insulin-responsive genes.
P.I: Dr Nirmala Yabaluri
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	1421653.00	Opening Balance	1421653.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**1421653.00**		**1421653.00**
1421653.00	Excess of Expenditure over Income	1421653.00	0.00	Closing Balance	0.00
1421653.00		**1421653.00**	**1421653.00**		**1421653.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-73: Identification and characterization of pancreatic cancer genes located within novel localized cpy number alterations

P.I: Dr M D Bashyam

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	857136.00	Opening Balance	857136.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**857136.00**		**857136.00**
857136.00	Excess of Expenditure over Income	857136.00	0.00	Closing Balance	0.00
857136.00		**857136.00**	**857136.00**		**857136.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-75: Preparing blueprint for the macromolecular crystallography beamline at Indus-II synchrotron source

P.I: Dr Sekhar C Mande

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	10840.00	Opening Balance	10840.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**10840.00**		**10840.00**
10840.00	Excess of Expenditure over Income	10840.00	0.00	Closing Balance	0.00
10840.00		**10840.00**	**10840.00**		**10840.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-76: A study of molecular markers in childhood autism with special references to nuclear factors - ± APPA B

P.I: Dr S K Manna

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	50234.00	Opening Balance	50234.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**50234.00**		**50234.00**
50234.00	Excess of Expenditure over Income	50234.00	0.00	Closing Balance	0.00
50234.00		**50234.00**	**50234.00**		**50234.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-77: Functional characterization of Mycobacterium tuberculosis PE/PPE proteins having SH3 binding domain : Understanding their role in modulating macrophage functions

P.I: Dr Sangita Mukhopadhyay

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
124277.00	Opening Balance	124277.00			
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
124277.00		**124277.00**	**0.00**		**0.00**
0.00	Excess of Expenditure over Income	0.00	124277.00	Closing Balance	124277.00
124277.00		**124277.00**	**124277.00**		**124277.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-78: Task force- IMD Newborn screening for Congenital Hypothyroidism & Congenital Adrenal Hyperplasia: A multicentric study
P.I: Dr A Radha Rama Devi
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
1304.00	Opening Balance	1304.00		Opening Balance	
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1304.00		**1304.00**	**0.00**		**0.00**
0.00	Excess of Expenditure over Income	0.00	1304.00	Closing Balance	1304.00
1304.00		**1304.00**	**1304.00**		**1304.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-79: Understanding the role of AGE proteins in inducing inflammatory responses and its regulation
P.I: Dr S K Manna
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	105086.00	Opening Balance	105086.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**105086.00**		**105086.00**
105086.00	Excess of Expenditure Over Income	105086.00	0.00	Closing Balance	0.00
105086.00		**105086.00**	**105086.00**		**105086.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-80: Referral centre for detection of genetically modified foods employing DNA-based markets
P.I: Dr Madhusudan Reddy
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	608222.00	Opening Balance	608222.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	608222.00	Transfer of Funds	0.00
0.00		**0.00**	**608222.00**		**608222.00**
608222.00	Excess of Expenditure over Income	608222.00	0.00	Closing Balance	0.00
608222.00		**608222.00**	**608222.00**		**608222.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-81: Reconstructing Cellular Networks: Two-component regulatory systems
P.I: Dr Shekhar Mande
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
143470.00	Opening Balance	143470.00			
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
143470.00		**143470.00**	**0.00**		**0.00**
0.00	Excess of Expenditure over Income	0.00	143470.00	Closing Balance	143470.00
143470.00		**143470.00**	143470**.00**		**143470.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-81A: Financial assistance for award of J C Bose Fellowship to Dr J Gowrishankar
P.I: Dr J Gowrishankar
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
62620.00	Opening Balance	2620.00			0.00
1300000.00	Grant In Aid	1360000.00	300000.00	Salaries - Manpower	275000.00
0.00		0.00	526318.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	37435.00
0.00		0.00	473682.00	Travel	199732.50
0.00		0.00	60000.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1362620.00		**1362620.00**	**136000.00**		**512167.00**
0.00	Excess of Expenditure Over Income	0.00	2620.00	Closing Balance	850453.00
1362620.00		**1362620.00**	**1362620.00**		**1362620.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-82: Functional genomic analysis of Candida Glabrata-macrophage
P.I: Dr Rupinder Kaur
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	369021.00	Opening Balance	369021.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**369021.00**		**369021.00**
369021.00	Excess of Expenditure Over Income	369021.00	0.00	Closing Balance	0.00
369021.00		**369021.00**	**369021.00**		**369021.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-83: Prokaryotic Transcription termination factor, Rho: Mechanism of Action and Biology
P.I: Dr Ranjan Sen
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	1155594.00	Opening Balance	1155594.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**1155594.00**		**1155594.00**
1155594.00	Excess of Expenditure over Income	1155594.00	0.00	Closing Balance	0.00
1155594.00		**1155594.00**	**1155594.00**		**1155594.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-84: Preparing for vaccine efficacy trials: Baseline epidemiology, improved diagnosis, markers of protection and phase I/II trials
P.I: Dr Niyaz Ahmed
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	1150.00	Opening Balance	1150.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**1150.00**		**1150.00**
1150.00	Excess of Expenditure over Income	1150.00	0.00	Closing Balance	0.00
1150.00		**1150.00**	**1150.00**		**1150.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-84A: Human epigenetic to the rescue of human identification process: Enriching human DNA from DNA mixture employing antibodies directed against 5-methylcytosine followed by whole genome amplification
P.I: Dr Madhusudan Reddy
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	106479.00	Opening Balance	106479.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**106479.00**		**106479.00**
106479.00	Excess of Expenditure over Income	106479.00	0.00	Closing Balance	0.00
106479.00		**106479.00**	**106479.00**		**106479.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-85: IdeR associated gene regulatory network in mycobacteria
P.I: Dr Akash Ranjan
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	1118755.00	Opening Balance	1118755.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00 1**	**118755.00**		**1118755.00**
1118755.00	Excess of Expenditure over Income	1118755.00	0.00	Closing Balance	0.00
1118755.00		**1118755.00**	**1118755.00**		**1118755.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-87: Comparative genomics of wild silkmoths
P.I: Dr J Nagaraju
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	65698.00	Opening Balance	65698.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**65698.00**		**65698.00**
65698.00	Excess of Expenditure over Income	65698.00	0.00	Closing Balance	0.00
65698.00		**65698.00**	**65698.00**		**65698.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-90: Role of Yapsins in the Pathobiology of Candida Glabrata
P.I: Dr Rupinder Kaur
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	636286.00	Opening Balance	636286.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**636286.00**		**636286.00**
636286.00	Excess of Expenditure over Income	636286.00	0.00	Closing Balance	0.00
636286.00		**636286.00**	**636286.00**		**636286.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-91: DMMT3L: epigenetic correlation with cancer
P.I: Dr Sanjeev Khosla
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	1098900.00	Opening Balance	1098900.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**1098900.00**		**1098900.00**
1098900.00	Excess of Expenditure over Income	1098900.00	0.00	Closing Balance	0.00
1098900.00		**1098900.00**	**1098900.00**		**1098900.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-92: Swarnajayanti fellowship proj on "Designing transcription anti-terminators: a novel approach for making new inhibitors of gene expression"
P.I: Dr Ranjan Sen
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
268823.00	Opening Balance	268823.00	0.00	Opening Balance	0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
268823.00		**268823.00**	**0.00**		**0.00**
0.00	Excess of Expenditure Over Income	0.00	268823.00	Closing Balance	268823.00
268823.00		**268823.00**	**268823.00**		**268823.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-93/A1 : Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against tuberculosis
P.I.: Dr Shekar
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	611833.00	Opening Balance	611833.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**611833.00**		**611833.00**
611833.00	Excess of Expenditure Over Income	611833.00	0.00	Closing Balance	0.00
611833.00		**611833.00**	**611833.00**		**611833.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-93/A2 : Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against Mycobacterium tuberculosis
P.I.: Dr. Sangita Mukhopadhyay
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	3025061.00	Opening Balance	3038491.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	190135.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	13430.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**3038491.00**		**3228626.00**
3038491.00	Excess of Expenditure Over Income	3228626.00	0.00	Closing Balance	0.00
3038491.00		**3228626.00**	**3038491.00**		**3228626.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-93B2 (II) : Evaluation of peptides / small molecules targeting ESAT-6:B2M interaction and PPE18-TLR2 interaction as potent anti tuberculosis therapautics
P.I.: Dr Sangita Mukhopadhyay
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
1110000.00	Opening Balance	483835.00		Opening Balance	261800.00
0.00	Grant In Aid	737000.00	301209.00	Salaries - Manpower	67467.00
0.00		0.00	305725.00	Consumables	30000.00
0.00		0.00	11581.00	Contingencies	23823.00
0.00		0.00	7623.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1110000.**00**		**1220835.00**	**626165.00**		**383090.00**
0.00	Excess of Expenditure Over Income	0.00	423835.00	Closing Balance	837745.00
1110000.**00**		**1220835.00**	**1110000.00**		**1220835.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-97: Proteome-wide Analysis of Serine pyrophosphorylation by inositol pyrophosphates
P.I: Dr Rashna Bhandari
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	276552.00	Opening Balance	276552.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**276552.00**		**276552.00**
276552.00	Excess of Expenditure Over Income	276552.00	0.00	Closing Balance	0.00
276552.00		**276552.00**	**276552.00**		**276552.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-98: Role of cell - cell signaling mediated by Diffusible signaling factor (DSF) in Xanthomonas virulence

P.I: Dr Subhadeep Chatterjee

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	236042.00	Opening Balance	236042.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**236042.00**		**236042.00**
236042.00	Excess of Expenditure Over Income	236042.00	0.00	Closing Balance	0.00
236042.00		**236042.00**	**236042.00**		**236042.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-99: Role of inositol Pyrophosphates in eukaryotic cell growth, proliferation and ribosomae biogenesis

P.I: Dr Rashna Bhandari

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	567516.00	Opening Balance	567516.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**567516.00**		**567516.00**
567516.00	Excess of Expenditure Over Income	567516.00	0.00	Closing Balance	0.00
567516.00		**567516.00**	**567516.00**		**567516.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-100: Effect of raective oxygen species on T-Cell immune response: An approach to understand the molecular mechanism of immunosuppression during tuberculosis - National Bioscience Award

P.I: Dr Sangita Mukhopadhyay

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	576590.00	Opening Balance	576590.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**576590.00**		**576590.00**
576590.00	Excess of Expenditure Over Income	576590.00	0.00	Closing Balance	0.00
576590.00		**576590.00**	**576590.00**		**576590.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-102: "Understanding the role of Mycobacterium tuberculosis heat shockprotein 60 as Th1/Th2 immuno modular"

P.I: Dr Sangita Mukhopadhyay

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	27922.00	Opening Balance	27922.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**27922.00**		**27922.00**
27922.00	Excess of Expenditure Over Income	27922.00	0.00	Closing Balance	0.00
27922.00		**27922.00**	**27922.00**		**27922.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-103: National Bioscience Award - Regulation of mast cell signaling, apoptosis and surface receptors
P.I: Dr Sunil Kumar Manna
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	300000.00	Opening Balance	300000.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	300000.**00**		**300000.00**
300000.00	Excess of Expenditure Over Income	300000.00	0.00	Closing Balance	0.00
300000.**00**		**300000.00**	300000.**00**		**300000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-104: Virtual Centre of Excellence on Epigenetics
P.I: Dr Sanjeev Khosla
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	1160508.00	Opening Balance	1289897.00
0.00	Grant In Aid	0.00	125806.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	3583.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**1289897.00**		**1289897.00**
1289897.00	Excess of Expenditure Over Income	1289897.00	0.00	Closing Balance	0.00
1289897.00		**1289897.00**	**1289897.00**		**1289897.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-105: Cloning, Characterization and analysis of chromosomal rearrangements in human genetic disorders

P.I: Dr Ashwin B Dalal

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	862685.00	Opening Balance	862685.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**862685.00**		**862685.00**
862685.00	Excess of Expenditure Over Income	862685.00	0.00	Closing Balance	0.00
862685.00		**862685.00**	**862685.00**		**862685.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-107: DBT IYBA Project on "Mechanism and role of bacterial cell-cell signaling molecules in plant defense response"

P.I: Dr Subhadeep Chatterjee

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
1036691.00	Opening Balance	366575.00		Opening Balance	0.00
0.00	Grant In Aid	0.00	70166.00	Salaries - Manpower	39000.00
0.00		0.00	589798.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	10202.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1036691.00		**366575.00**	**670116.00**		**39000.00**
0.00	Excess of Expenditure Over Income	0.00	366575.00	Closing Balance	327575.00
1036691.00		**366575.00**	1036691.00		**366575.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-108: Establishment of EBV transformed cell lines from families with rare genetic disorders
P.I: Dr Ashwin B Dalal
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	454643.00	Opening Balance	454643.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**454643.00**		**454643.00**
454643.00	Excess of Expenditure Over Income	454643.00	0.00	Closing Balance	0.00
454643.00		**454643.00**	**454643.00**		**454643.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-109: Molecular dissection of PI3-Kinase/Akt pathway by suing proteomics based approach: A study to identify novel potential oncogenes and tumor suppressors
P.I: Dr M Subba Reddy
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
3351336.00	Opening Balance	767943.00			0.00
2479000.00	Grant In Aid	0.00	739256.00	Salaries - Manpower	689891.00
0.00		0.00	1517891.00	Consumables	224855.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	10109.00	Travel	15179.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	2795137.00	Equipment	200411.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
5830336.00		**767943.00**	**5062393.00**		**1130336.00**
0.00	Excess of Expenditure Over Income	362393.00	767943.00	Closing Balance	0.00
5830336.00		**1130336.00**	**5830336.00**		**1130336.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-110: India-Japan research project title"Identification and analysis of sex determining genes in silkmoths"
P.I: Dr J Nagaraju
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	191391.00	Opening Balance	191391.00
0.00	Grant In Aid	172000.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**191391.00**		**191391.00**
191391.00	Excess of Expenditure Over Income	191391.00	0.00	Closing Balance	0.00
191391.00		**191391.00**	**191391.00**		**191391.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-114: Evaluating the Calcineurin-NFAT Pathway and its regulators superoxide dismutase (SOD) AND RCAN1 (regular of Calcineurin) Down Syndrome
P.I: Dr Gayatri Ramakrishna, Dr Ashwin Dalal
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	450859.00	Opening Balance	450859.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**450859.00**		**450859.00**
450859.00	Excess of Expenditure Over Income	450859.00	0.00	Closing Balance	0.00
450859.00		**450859.00**	**450859.00**		**450859.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-116: DBT-India and AIST - Japan : Understanding molecular mechanisms controlling dual role of Ras, Sirtuins and CARF in relation to cellular proliferation and senescence: Novel Strategy for developing cancer therapeutics

P.I: Dr Gayatri Ramakrishna

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	1251366.00	Opening Balance	1251366.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**1251366.00**		**1251366.00**
1251366.00	Excess of Expenditure Over Income	1251366.00	0.00	Closing Balance	0.00
1251366.00		**1251366.00**	**1251366.00**		**1251366.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-119: Analysis of DNA copy number alterations in esophaeal cancer

P.I: Dr M D Bashyam

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	2892.00	Opening Balance	2892.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**2892.00**		**2892.00**
2892.00	Excess of Expenditure Over Income	2892.00	0.00	Closing Balance	0.00
2892.00		**2892.00**	**2892.00**		**2892.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-120: Effect of reactive oxygen species on macrophage signalosome: impact on antigen presentation functions and T Cell priming responses
P.I: Dr Sangita Mukhopadhyay
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	769484.00	Opening Balance	769484.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**769484.00**		**769484.00**
769484.00	Excess of Expenditure Over Income	769484.00	0.00	Closing Balance	0.00
769484.00		**769484.00**	**769484.00**		**769484.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-121: Identification and characterization of PTEN regulators
P.I: Dr M Subba Reddy
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	1130866.00	Opening Balance	1130866.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**1130866.00**		**1130866.00**
1130866.00	Excess of Expenditure Over Income	1130866.00	0.00	Closing Balance	0.00
1130866.00		**1130866.00**	**1130866.00**		**1130866.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-122: Understanding the role of Hox genes in anterior-posterior axis determination of the central nervous system
P.I: Dr Rohit Joshi
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
388692.00	Opening Balance	2951109.00			0.00
8005983.00	Grant In Aid	2722184.00	662020.00	Salaries - Manpower	194574.00
0.00		0.00	2843518.00	Consumables	3368228.00
0.00		0.00	32463.00	Contingencies	3377.00
0.00		0.00	44681.00	Travel	19369.00
0.00		0.00	483752.00	Overheads	513833.00
0.00		0.00	1254840.00	Equipment	1552788.00
0.00		0.00	122292.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
8394675.00		**5673293.00**	**5443566.00**		**5652169.00**
0.00	Excess of Expenditure Over Income	0.00	2951109.00	Closing Balance	21124.00
8394675.00		**5673293.00**	**8394675.00**		**5673293.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-123: Establish a Max Planck Partner Group for Genetic Diversity Studies at CDFD
P.I: Dr N Madhusudan Reddy
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
1402135.00	Opening Balance	771699.00		Opening Balance	0.00
1413360.00	Grant In Aid	1648000.00	395200.00	Salaries - Manpower	199277.00
0.00		0.00	886802.00	Consumables	428574.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	274360.00	Travel	186183.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	487434.00	Equipment	164978.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2815495.00		**2419699.00**	**2043796.00**		**979012.00**
0.00	Excess of Expenditure Over Income	0.00	771699.00	Closing Balance	1440687.00
2815495.00		**2419699.00**	**2815495.00**		**2419699.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-124: Preparation and characterization of peroxometal compounds and studies and their biological significance in cellular signalling

P.I: Dr Gayatri Ramakrishna

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	748411.00	Opening Balance	748411.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**748411.00**		**748411.00**
748411.00	Excess of Expenditure Over Income	748411.00	0.00	Closing Balance	0.00
748411.00		**748411.00**	**748411.00**		**748411.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-126: Rho-dependent transcription termination machinery: mechanism of action

P.I: Dr Ranjan Sen

Receipts and Payments Account from 01/04/2016to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
442524.00	Opening Balance	209670.00		Opening Balance	0.00
0.00	Grant In Aid	0.00	48729.00	Salaries - Manpower	49400.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	16919.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	167206.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
442524.00		**209670.00**	**232854.00**		**49400.00**
0.00	Excess of Expenditure Over Income	0.00	209670.00	Closing Balance	160270.00
442524.00		**209670.00**	**442524.00**		**209670.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-127: Systematic studies on the functional network of phosphatases in cell life and death
P.I: Dr M Subba Reddy
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	1895283.00	294516.00	Opening Balance	0.00
6736571.00	Grant In Aid	663747.00	432000.00	Salaries - Manpower	144000.00
0.00		0.00	3078989.00	Consumables	2182390.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	317282.00	Travel	0.00
0.00		0.00	384898.00	Overheads	232640.00
0.00		0.00	20707.00	Equipment	0.00
0.00		0.00	312896.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
6736571.00		**2559030.00**	**4841288.00**		**2559030.00**
0.00	Excess of Expenditure Over Income	0.00	1895283.00	Closing Balance	0.00
6736571.00		**2559030.00**	**6736571.00**		**2559030.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-128: Mechanism of iron acquisition and iron homeostasis in an opportunistic human pathogen Candida glabrata
P.I: Dr Rupinder Kaur
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	77108.00	Opening Balance	158488.00
0.00	Grant In Aid	0.00	1740.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	79640.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**158488.00**		**158488.00**
158488.00	Excess of Expenditure Over Income	158488.00	0.00	Closing Balance	0.00
158488.00		**158488.00**	**158488.00**		**158488.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-130: Comparative genetic analysis of sex chromosomes and sex determining genes in silkmoths
P.I: Dr J Nagaraju
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	869.00	2550050.00	Opening Balance	0.00
4024000.00	Grant In Aid	0.00	546581.00	Salaries - Manpower	125471.00
0.00		0.00	0.00	Consumables	17656.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	926500.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
4024000.00		**869.00**	**4023131.00**		**143127.00**
0.00	Excess of Expenditure Over Income	142258.00	869.00	Closing Balance	0.00
4024000.00		**143127.00**	**4024000.00**		**143127.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-131: Structural and functional studies of Acyl CoA Binding proteins from plasmodium falciparum
P.I: Dr Akash Ranjan
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
398632.00	Opening Balance	398632.00		Opening Balance	0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
398632.00		**398632.00**	**0.00**		**0.00**
0.00	Excess of Expenditure Over Income	0.00	398632.00	Closing Balance	398632.00
398632.00		**398632.00**	**398632.00**		**398632.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-132: Characterization of tumor suppressor function of ARIDIB, a component of the human SWI/SNF chromatin remodelling complex
P.I: Dr M D Bashyam, Dr Rohit Joshi
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	12199.00	Opening Balance	12199.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**12199.00**		**12199.00**
12199.00	Excess of Expenditure Over Income	12199.00	0.00	Closing Balance	0.00
12199.00		**12199.00**	**12199.00**		**12199.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-133: Investigating the role of Hox gene deformed in central nervous system patterning in Drosophila melanogaster
P.I: Dr Rohit Joshi
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
460117.00	Opening Balance	0.00		Opening Balance	702990.00
0.00	Grant In Aid	500000.00	206034.00	Salaries - Manpower	132600.00
0.00		0.00	946755.00	Consumables	988633.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	-25467.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	35785.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
460117.00		**500000.00**	**1163107.00**		**1824223.00**
702990.00	Excess of Expenditure Over Income	1324223.00	0.00	Closing Balance	0.00
1163107.00		**1824223.00**	**1163107.00**		**1824223.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-134: Exploration of wild silk moth biodiversity in Manipur and their genetic characterization using molecular markers

P.I: Dr K P Arun Kumar

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	77061.00	Opening Balance	77061.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**77061.00**		**77061.00**
77061.00	Excess of Expenditure Over Income	77061.00	0.00	Closing Balance	0.00
77061.00		**77061.00**	**77061.00**		**77061.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-135: Sys TB: A Network Program for Resolving the Intracellular Dynamics of Host Phthogen Interaction in TB Infection

P.I: Dr. Sanjeev Kholsa

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	357268.00	Opening Balance	336135.00
2430700.00	Grant In Aid	0.00	343200.00	Salaries - Manpower	343200.00
0.00		0.00	2000000.00	Consumables	423237.00
0.00		0.00	50000.00	Contingencies	0.00
0.00		0.00	16367.00	Travel	16184.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2430700.00		**0.00**	**2766835.00**		**1118756.00**
336135.00	Excess of Expenditure Over Income	1118756.00	0.00	Closing Balance	0.00
2766835.00		**1118756.00**	**2766835.00**		**1118756.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-136: Raf Kinase - a key target for modem-day theraphy against tumors
P.I: Dr Sunil Kumar Manna
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	292334.00	Opening Balance	196001.00
0.00	Grant In Aid	0.00	-43781.00	Salaries - Manpower	0.00
0.00		0.00	-20658.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	-31894.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**196001.00**		**196001.00**
196001.00	Excess of Expenditure Over Income	196001.00	0.00	Closing Balance	0.00
196001.00		**196001.00**	**196001.00**		**196001.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-138: Co-evaluation of Dnmt3l and Genomic imprinting
P.I: Dr Sanjeev Khosla
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	1353238.00	Opening Balance	1500300.00
0.00	Grant In Aid	0.00	12580.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	-48800.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	134482.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**1500300.00**		**1451500.00**
1500300.00	Excess of Expenditure Over Income	1451500.00	0.00	Closing Balance	0.00
1500300.00		**1451500.00**	**1500300.00**		**1451500.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-139: Evaluating the role of Sirtuins and epigenetic changes during cellular senescense in context of p53 status

P.I: Dr Gayatri Ramakrishna, Dr Sanjeev Khosla

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
20000.00	Opening Balance	20000.00		Opening Balance	0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
20000.00		**20000.00**	**0.00**		**0.00**
0.00	Excess of Expenditure Over Income	0.00	20000.00	Closing Balance	20000.00
20000.00		**20000.00**	**20000.00**		**20000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-140: Development of baculovirus resistant silkworms strains through synthetic miRNA based knockdown of essential viral genes

P.I: Dr K P Arun Kumar

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	403336.00	Opening Balance	608652.00
0.00	Grant In Aid	0.00	205316.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**608652.00**		**608652.00**
608652.00	Excess of Expenditure Over Income	608652.00	0.00	Closing Balance	0.00
608652.00		**608652.00**	**608652.00**		**608652.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-141: Evaluating the functional role of PTEN interacting proteins in cell survival signaling and tumor suppression

P.I: Dr M Subba Reddy

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	125000.00	Opening Balance	125000.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**125000.00**		**125000.00**
125000.00	Excess of Expenditure Over Income	125000.00	0.00	Closing Balance	0.00
125000.00		**125000.00**	**125000.00**		**125000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-142: Identification of H3K4 TRI Demethylase involved in erasing H3K4 trimethylation marks at E2F Responsive promoters

P.I: Dr Shweta Tyagi

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	280596.00	Opening Balance	81861.00
196800.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	(-2.00)	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	(0.00)	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
196800.00		**0.00**	**278661.00**		**81861.00**
81861.00	Excess of Expenditure Over Income	81861.00	0.00	Closing Balance	0.00
278661.00		**81861.00**	**278661.00**		**81861.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-143: Microarray based characterisation of squamous cell carcinoma of the tongue occuring in non smokers
P.I: Dr M D Bashyam
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	534504.00	Opening Balance	1381684.00
0.00	Grant In Aid	662545.00	205400.00	Salaries - Manpower	0.00
0.00		0.00	487500.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	154280.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**662545.00**	**1381684.00**		**1381684.00**
1381684.00	Excess of Expenditure Over Income	719139.00	0.00	Closing Balance	0.00
1381684.00		**1381684.00**	**1381684.00**		**1381684.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-144 : Tri-National Training Program for Psychiatric Genetics
P.I: Dr Ashwin B Dalal
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
424130.00	Opening Balance	122130.00		Opening Balance	0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	302000.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
424130.00		**122130.00**	**302000.00**		**0.00**
0.00	Excess of Expenditure Over Income	0.00	122130.00	Closing Balance	122130.00
424130.00		**122130.00**	**424130.00**		**122130.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-145: H3K4 HMT family regulatescell cycle progression
P.I: Dr Shweta Tyagi
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	3222.00	1112243.00	Opening Balance	0.00
1200000.00	Grant In Aid	0.00	72713.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	11822.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1200000.00		**3222.00**	**1196778.00**		**0.00**
0.00	Excess of Expenditure Over Income	0.00	3222.00	Closing Balance	3222.00
1200000.00		**3222.00**	**1200000.00**		**3222.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-146: Role of MLL in ribosomal RNA transcription
P.I: Dr Shweta Tyagi
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
433858.00	Opening Balance	59533.00		Opening Balance	0.00
0.00	Grant In Aid	0.00	107187.00	Salaries - Manpower	0.00
0.00		0.00	267138.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
433858.00		**59533.00**	**374325.00**		**0.00**
0.00	Excess of Expenditure Over Income	0.00	59533.00	Closing Balance	59533.00
433858.00		**59533.00**	**433858.00**		**59533.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-147: The Effect of Parental Education, Ethics of Research Participation and Array Comparative Genomic Hybridization in Subjects with Mental Retardation (MR) and /or Autism
P.I: Dr Ashwin B Dalal
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	677839.00	Opening Balance	272874.00
500000.00	Grant In Aid	0.00	82026.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	13009.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
500000.00		**0.00**	**772874.00**		**272874.00**
272874.00	Excess of Expenditure Over Income	272874.00	0.00	Closing Balance	0.00
772874.00		**272874.00**	**772874.00**		**272874.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-149: Role of SUMOylation in the pathobiology of Candida Glabrata
P.I: Dr Rupinder Kaur
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	1016335.00	Opening Balance	59917.00
1420800.00	Grant In Aid	0.00	153920.00	Salaries - Manpower	13084.00
0.00		0.00	300000.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	10182.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	280.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1420800.00		**0.00**	**1480717.00**		**73001.00**
59917.00	Excess of Expenditure Over Income	73001.00	0.00	Closing Balance	0.00
1480717.00		**73001.00**	**1480717.00**		**73001.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-151: Human Exome Sequencing to Identify Novel Genes for Medelian Disorders
P.I: Dr Ashwin B Dalal
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	375851.00	601366.00	Opening Balance	0.00
1756400.00	Grant In Aid	0.00	343200.00	Salaries - Manpower	28600.00
0.00		0.00	351886.00	Consumables	148114.00
0.00		0.00	25000.00	Contingencies	0.00
0.00		0.00	59097.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1756400.00		**375851.00**	**1380549.00**		**176714.00**
0.00	Excess of Expenditure Over Income	0.00	375851.00	Closing Balance	199137.00
1756400.00		**375851.00**	**1756400.00**		**375851.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-152 : Global transcriptomics of sex specific spilicing
P.I: Dr K P Arun Kumar
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
29100.00	Opening Balance	0.00		Opening Balance	30814.00
1931400.00	Grant In Aid	0.00	343200.00	Salaries - Manpower	483433.00
0.00		0.00	1648114.00	Consumables	592311.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	17421.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1960500.00		**0.00**	**1991314.00**		**1123979.00**
30814.00	Excess of Expenditure Over Income	1123979.00	0.00	Closing Balance	0.00
1991314.00		**1123979.00**	**1991314.00**		**1123979.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-153: An attractive and promising stragegy for early cancer diagnosis through the assembly of the human cancer volatome"

P.I: Dr H A Nagarajaram

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
641552.00	Opening Balance	0.00		Opening Balance	64305.00
0.00	Grant In Aid	1787000.00	358800.00	Salaries - Manpower	296400.00
0.00		0.00	70000.00	Consumables	0.00
0.00		0.00	80000.00	Contingencies	6049.00
0.00		0.00	197057.00	Travel	52330.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	206143.00
0.00		0.00	0.00	Transfer of Funds	0.00
641552.00		**1787000.00**	**705857.00**		**625227.00**
64305.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	1161773.00
705857.00		**1787000.00**	**705857.00**		**1787000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-154 : Rational design, synthetic strategies for developing organometallic anticancer compounds based on organotin and organoiron

P.I: Dr Sunil Kumar Manna

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
30832.00	Opening Balance	13510.00		Opening Balance	0.00
930000.00	Grant In Aid	0.00	297322.00	Salaries - Manpower	15097.00
0.00		0.00	600000.00	Consumables	432806.00
0.00		0.00	50000.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
960832.00		**13510.00**	**947322.00**		**447903.00**
0.00	Excess of Expenditure Over Income	434393.00	13510.00	Closing Balance	0.00
960832.00		**447903.00**	**960832.00**		**447903.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-155: Studies on thecellular roles of calcium signalling proteins in Neurospora crassa

P.I: Dr D P Kasbekar

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
335194.00	Opening Balance	335194.00		Opening Balance	0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
335194.00		**335194.00**	**0.00**		**0.00**
0.00	Excess of Expenditure Over Income	0.00	335194.00	Closing Balance	335194.00
335194.00		**335194.00**	**335194.00**		**335194.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-156 : Targeting microbial quorum sensing to demonstrate potential application of cell-cell signaling molecules from Xanthomonas group of plant pathogen in diesease control

PI : Dr Subhadeep Chatterjee

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	239949.00	175165.00	Opening Balance	0.00
1706000.00	Grant In Aid	0.00	345520.00	Salaries - Manpower	82680.00
0.00		0.00	1000000.00	Consumables	724735.00
0.00		0.00	-50000.00	Contingencies	24845.00
0.00		0.00	0.00	Travel	12812.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	-4634.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1706000.00		**239949.00**	**1466051.00**		**845072.00**
0.00	Excess of Expenditure Over Income	605123.00	239949.00	Closing Balance	0.00
1706000.00		**845072.00**	**1706000.00**		**845072.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-157 : Identification of novel antifungal drug and delineation of drug resistance mechanisms in an opportunistic human fungal pathogen Candida glabrata

PI : Dr Rupinder Kaur

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
204372.00	Opening Balance	0.00		Opening Balance	1361799.00
0.00	Grant In Aid	1638000.00	165813.00	Salaries - Manpower	109200.00
0.00		0.00	1402360.00	Consumables	42992.00
0.00		0.00	-23540.00	Contingencies	0.00
0.00		0.00	21538.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
204372.00		**1638000.00**	**1566171.00**		**1513991.00**
1361799.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	124009.00
1566171.00		**1638000.00**	**1566171.00**		**1638000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-158 : Modulation of host immune responses by a PPE Protein of Mycobacterium tuberculosis: Understanding its role in host - pathogen cross-talk

PI : Dr Sangita Mukhopadhyay

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	1379658.00	Opening Balance	2575346.00
0.00	Grant In Aid	2790992.00	100100.00	Salaries - Manpower	187200.00
0.00		0.00	1011202.00	Consumables	196820.00
0.00		0.00	23868.00	Contingencies	0.00
0.00		0.00	17338.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	43180.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**2790992.00**	**2575346.00**		**2959366.00**
2575346.00	Excess of Expenditure Over Income	168374.00	0.00	Closing Balance	0.00
2575346.00		**2959366.00**	**2575346.00**		**2959366.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-159 : Gene Targeting of microbial isolates to demonstrate potential plant growth promoting (PGP) traits by third generation sequencing
PI : Dr Subhadeep Chatterjee
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00		Opening Balance	300000.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	300000.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**300000.00**	**300000.00**		**300000.00**
300000.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	0.00
300000.00		**300000.00**	**300000.00**		**300000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-160 : Understanding the role of novel adhesins of Xanthomonas oryzae PV orzae in Virulence and colonization in Rice
PI : Dr Subhadeep Chatterjee
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
208333.00	Opening Balance	0.00	0.00	Opening Balance	41667.00
687200.00	Grant In Aid	0.00	187200.00	Salaries - Manpower	62400.00
0.00		0.00	750000.00	Consumables	43113.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
895533.00		**0.00**	**937200.00**		**147180.00**
41667.00	Excess of Expenditure Over Income	147180.00	0.00	Closing Balance	0.00
937200.00		**147180.00**	**937200.00**		**147180.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-162 : Characterization and design of inhibitors of Mycobacterium tuberculosis transcription
PI : Dr Ranjan Sen
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	316464.00	Opening Balance	1021767.00
0.00	Grant In Aid	699600.00	247673.00	Salaries - Manpower	117000.00
0.00		0.00	422026.00	Consumables	0.00
0.00		0.00	25000.00	Contingencies	0.00
0.00		0.00	10604.00	Travel	25000.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**699600.00**	**1021767.00**		**1163767.00**
1021767.00	Excess of Expenditure Over Income	464167.00	0.00	Closing Balance	0.00
1021767.00		**1163767.00**	**1021767.00**		**1163767.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-163 : Unravelling new functions for the H-NS family of proteins in Gram-negative bacterial pathogens
PI : Dr J Gowrishankar
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
1052471.00	Opening Balance	678659.00		Opening Balance	0.00
1062777.00	Grant In Aid	1483389.00	194480.00	Salaries - Manpower	117000.00
0.00		0.00	800000.00	Consumables	47378.00
0.00		0.00	30000.00	Contingencies	2229.00
0.00		0.00	342109.00	Travel	465102.83
0.00		0.00	70000.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2115248.00		**2162048.00**	**1436589.00**		**631709.83**
0.00	Excess of Expenditure Over Income	0.00	678659.00	Closing Balance	1530338.17
2115248.00		**2162048.00**	**2115248.00**		**2162048.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-164 : A Yeast based screen for discovery of novel sirtuin inhibitors as anticancer agents
PI : Dr Devyani Halder
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00		Opening Balance	29200.00
0.00	Grant In Aid	0.00	24671.00	Salaries - Manpower	0.00
0.00		0.00	4529.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	29200.**00**		29200.**00**
29200.00	Excess of Expenditure Over Income	29200.00	0.00	Closing Balance	0.00
29200.**00**		29200.**00**	29200.**00**		29200.**00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-165 : Identification and functional characterization of immune response genes in silkmoths
PI : Dr V V Satyavathi
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
330135.00	Opening Balance	1567830.00		Opening Balance	0.00
2858334.00	Grant In Aid	0.00	344600.00	Salaries - Manpower	297200.00
0.00		0.00	1000000.00	Consumables	407724.00
0.00		0.00	50000.00	Contingencies	0.00
0.00		0.00	15957.00	Travel	0.00
0.00		0.00	50000.00	Overheads	0.00
0.00		0.00	160082.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
3188469.00		**1567830.00**	**1620639.00**		**704924.00**
0.00	Excess of Expenditure Over Income	0.00	1567830.00	Closing Balance	862906.00
3188469.00		**1567830.00**	**3188469.00**		**1567830.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-166 : Sequencing analysis of transcriptome variants in early-onset sporadic rectal cancer
PI : Dr M D Bashyam
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
2165638.00	Opening Balance	35696.00		Opening Balance	0.00
574700.00	Grant In Aid	0.00	192400.00	Salaries - Manpower	354378.00
0.00		0.00	500000.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	30850.00
0.00		0.00	12242.00	Travel	19077.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	2000000.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2740338.00		**35696.00**	**2704642.00**		**404305.00**
0.00	Excess of Expenditure Over Income	368609.00	35696.00	Closing Balance	0.00
2740338.00		**404305.00**	**2740338.00**		**404305.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-167 : To elucidate the role of MLL complex in epigenetic specification of centromere
PI : Dr Shweta Tyagi
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
633780.00	Opening Balance	569787.00		Opening Balance	0.00
1500000.00	Grant In Aid	900000.00	137381.00	Salaries - Manpower	46800.00
0.00		0.00	885797.00	Consumables	617187.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	19362.00	Travel	25148.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	521453.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2133780.00		**1469787.00**	**1563993.00**		**689135.00**
0.00	Excess of Expenditure Over Income	0.00	569787.00	Closing Balance	780652.00
2133780.00		**1469787.00**	**2133780.00**		**1469787.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-168 : A Search for nucleus -limited genes in Neurospora
PI : Dr D P Kasbekar
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
788623.00	Opening Balance	0.00		Opening Balance	0.00
1000000.00	Grant In Aid	0.00	187200.00	Salaries - Manpower	0.00
0.00		0.00	1110910.00	Consumables	161318.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	25963.00	Travel	0.00
0.00		0.00	100000.00	Overheads	0.00
0.00		0.00	364550.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1788623.00		**0.00**	**1788623.00**		**161318.00**
0.00	Excess of Expenditure Over Income	161318.00	0.00	Closing Balance	0.00
1788623.00		**161318.00**	**1788623.00**		**161318.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-169 : Implementation of 3 year DNB Program in Medical Genetics by Department of Biotechnology in colloboration with National Board of Examination ag SGHR, NIBMG&CDFD
PI : Dr J Gowrishankar
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
1758108.00	Opening Balance	16915.00		Opening Balance	0.00
0.00	Grant In Aid	2535600.00	1300000.00	Salaries - Manpower	2529290.00
0.00		0.00	121193.00	Consumables	55242.00
0.00		0.00	20000.00	Contingencies	0.00
0.00		0.00	300000.00	Travel	300000.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1758108.00		**2552515.00**	**1741193.00**		**2884532.00**
0.00	Excess of Expenditure Over Income	332017.00	16915.00	Closing Balance	0.00
1758108.00		**2884532.00**	**1758108.00**		**2884532.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-170 : Women Scientist Scheme "Identification and character of deregulated micro RNAs in defined sub-set of early onset sporadic rectal cancer patients using transcriptome sequencing"

PI : Dr Mithu Ray Chaudhuri

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
277449.00	Opening Balance	0.00	0.00	Opening Balance	659867.00
0.00	Grant In Aid	1100000.00	587316.00	Salaries - Manpower	730000.00
0.00		0.00	300000.00	Consumables	78750.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	15246.00
0.00		0.00	50000.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
277449.00		**1100000.00**	**937316.00**		**1483863.00**
659867.00	Excess of Expenditure Over Income	383863.00	0.00	Closing Balance	0.00
937316.00		**1483863.00**	**937316.00**		**1483863.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-171 : Role of vesicle-mediated transport and chromatin remodelling in the virulence of Candida glabrata

PI : Dr Rupinder Kaur

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
1754447.00	Opening Balance	211423.00	0.00	Opening Balance	0.00
0.00	Grant In Aid	0.00	236080.00	Salaries - Manpower	553547.00
0.00		0.00	1011064.00	Consumables	895411.00
0.00		0.00	320.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	295560.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1754447.00		**211423.00**	**1543024.00**		**1448958.00**
	Excess of Expenditure Over Income	1237535.00	211423.00	Closing Balance	0.00
1754447.00		**1448958.00**	**1754447.00**		**1448958.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-172 : Molecular Characterization of early onset sporadic rectal cancer

PI : Dr M D Bashyam

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
1461747.00	Opening Balance	111850.00		Opening Balance	0.00
1200000.00	Grant In Aid	1000000.00	465412.00	Salaries - Manpower	423251.00
0.00		0.00	596335.00	Consumables	522522.00
0.00		0.00	0.00	Contingencies	5000.00
0.00		0.00	0.00	Travel	9207.00
0.00		0.00	100000.00	Overheads	0.00
0.00		0.00	1388150.00	Equipment	111850.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2661747.00		**1111850.00**	**2549897.00**		**1071830.00**
0.00	Excess of Expenditure Over Income	0.00	111850.00	Closing Balance	40020.00
2661747.00		**1111850.00**	**2661747.00**		**1111850.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-173 : Development and application of a next generation sequencing approach for molecular genetic analysis of lysosomal storage disorders

PI : Dr Ashwin B Dalal

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
584882.00	Opening Balance	487953.00		Opening Balance	0.00
699782.00	Grant In Aid	2107380.00	326006.00	Salaries - Manpower	387703.00
0.00		0.00	470705.00	Consumables	529500.00
0.00		0.00	0.00	Contingencies	6000.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1284664.00		**2595333.00**	**796711.00**		**923203.00**
0.00	Excess of Expenditure Over Income	0.00	487953.00	Closing Balance	1672130.00
1284664.00		**2595333.00**	**1284664.00**		**2595333.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-174 : Is non-canonical Wnt signalling a major player in early-onset sporadic rectal cancer

PI : Dr M D Bashyam

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
500000.00	Opening Balance	520542.00		Opening Balance	0.00
500000.00	Grant In Aid	0.00	210432.00	Salaries - Manpower	273420.00
0.00		0.00	260905.00	Consumables	37716.00
0.00		0.00	8121.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1000000.00		**520542.00**	**479458.00**		**311136.00**
0.00	Excess of Expenditure Over Income	0.00	520542.00	Closing Balance	209406.00
1000000.00		**520542.00**	**1000000.00**		**520542.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-175 : Multi Centri Collaborative study of the Clinical, Biochemical and Molecular Characterization of Lysosomal storage disorders in India - The initiative for research in Lysosomal Storage Disorders"

PI : Dr Ashwin B Dalal

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	509714.00	Opening Balance	1432672.00
0.00	Grant In Aid	2214648.00	396076.00	Salaries - Manpower	541200.00
0.00		0.00	500000.00	Consumables	345462.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	16983.00
0.00		0.00	26882.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**2214648.00**	**1432672.00**		**2336317.00**
1432672.00	Excess of Expenditure Over Income	121669.00	0.00	Closing Balance	0.00
1432672.00		**2336317.00**	**1432672.00**		**2336317.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-176 : International Atomic Energy Agency

PI : Dr K P Arun Kumar
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
200103.00	Opening Balance	200103.00		Opening Balance	0.00
0.00	Grant In Aid	207044.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	199130.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
200103.00		**407147.00**	**0.00**		**199130.00**
0.00	Excess of Expenditure Over Income	0.00	200103.00	Closing Balance	208017.00
200103.00		**407147.00**	**200103.00**		**407147.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-177 : Morphological and molecular taxonomy of the Phlebotomus argendtipes species complex in relation to transmission of Kala-azar in India"

PI : Dr J Gowrishankar
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00		Opening Balance	197394.00
225000.00	Grant In Aid	225000.00	0.00	Salaries - Manpower	0.00
0.00		0.00	400000.00	Consumables	147576.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	22394.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
225000.00		**225000.00**	**422394.00**		**344970.00**
197394.00	Excess of Expenditure Over Income	119970.00	0.00	Closing Balance	0.00
422394.00		**344970.00**	**422394.00**		**344970.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-178 : Understanding differential signaling via toll like receptor-2: A proteomics approach

PI : Dr Rameshwaram Nagender Rao

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00		Opening Balance	0.00
1000000.00	Grant In Aid	1000000.00	507419.00	Salaries - Manpower	660000.00
0.00		0.00	376554.00	Consumables	134050.00
0.00		0.00	0.00	Contingencies	15801.00
0.00		0.00	16027.00	Travel	5950.00
0.00		0.00	100000.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1000000.00		**1000000.00**	**1000000.00**		**815801.00**
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	184199.00
1000000.00		**1000000.00**	**1000000.00**		**1000000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-179 : Quality Assurance Programme for Molecular and Prenatal Diagnosis of Hemoglobin Opathies

PI : Dr Ashwin B Dalal

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00		Opening Balance	50000.00
50000.00	Grant In Aid	100000.00	0.00	Salaries - Manpower	0.00
0.00		0.00	100000.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
50000.00		**100000.00**	**100000.00**		**50000.00**
50000.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	50000.00
100000.00		**100000.00**	**100000.00**		**100000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-180 : Collaborative studies on genomic diversity among bombycoid silkmoths in Asia

PI : Dr Akash Ranjan

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	117886.00		Opening Balance	0.00
200000.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	4223.00
0.00		0.00	82114.00	Travel	50279.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
200000.00		**117886.00**	**82114.00**		**54502.00**
0.00	Excess of Expenditure Over Income	0.00	117886.00	Closing Balance	63384.00
200000.00		**117886.00**	**200000.00**		**117886.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-181 : To Conduct multilocational field trails transgenic BmNPV resistant silkworm strains to establish their efficacy and generate data for their regulatory approval

PI : Dr V V Satyavathi

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	1744000.00		Opening Balance	0.00
1744000.00	Grant In Aid	0.00	0.00	Salaries - Manpower	446512.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	74392.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1744000.00		**1744000.00**	**0.00**		**520904.00**
0.00	Excess of Expenditure Over Income	0.00	1744000.00	Closing Balance	1223096.00
1744000.00		**1744000.00**	**1744000.00**		**1744000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-182 : Ramalingaswami Fellowship

PI : Dr Mohan C Joshi

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00		Opening Balance	277500.00
0.00	Grant In Aid	2110000.00	277500.00	Salaries - Manpower	555000.00
0.00		0.00	0.00	Consumables	744226.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**2110000.00**	**277500.00**		**1576726.00**
277500.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	533274.00
277500.00		**2110000.00**	**277500.00**		**2110000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-183 : "Prevalence and predictors of vitamin B12 deficiency: genetic associations for low vitamin B12 levels-multi-center a pan India study",

PI : Dr G R Chandak

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00		Opening Balance	0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	1091800.00
0.00		**0.00**	**0.00**		**1091800.00**
0.00	Excess of Expenditure Over Income	1091800.00	0.00	Closing Balance	0.00
0.00		**1091800.00**	**0.00**		**1091800.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-184 : Computational Approaches to Understanding Peptide- Protein Interactions involved in the Regulatory Events in the Cell"

PI : Dr Raghavender Surya Upadhyayula

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	957742.00		Opening Balance	0.00
1060000.00	Grant In Aid	0.00	92258.00	Salaries - Manpower	660000.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	7948.00
0.00		0.00	10000.00	Overheads	0.00
0.00		0.00	0.00	Equipment	166729.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1060000.00		**9577 42.00**	**102258.00**		**834677.00**
0.00	Excess of Expenditure Over Income	0.00	957742.00	Closing Balance	123065.00
1060000.00		**957742.00**	**1060000.00**		**957742.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-185 : Investigating potential of mycobacterium tuberculosis protein PPE18 encapsulated nanoparticle as therapy for microbial sepsis

PI : Dr Sangita Mukhopadhyay

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	1632207.00		Opening Balance	0.00
1648000.00	Grant In Aid	0.00	0.00	Salaries - Manpower	195000.00
0.00		0.00	15793.00	Consumables	61376.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	20000.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	84421.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1648000.00		**1632207.00**	**15793.00**		**360797.00**
0.00	Excess of Expenditure Over Income	0.00	1632207.00	Closing Balance	1271410.00
1648000.00		**1632207.00**	**1648000.00**		**1632207.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-186 : In vivo corss-talks between Rho-dependent transcription termination and other biological processes

PI : Dr Ranjan Sen

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	2410000.00		Opening Balance	0.00
2410000.00	Grant In Aid	1841600.00	0.00	Salaries - Manpower	408871.00
0.00		0.00	0.00	Consumables	1182804.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	30000.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	2180896.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2410000.00		**4251600.00**	**0.00**		**3802571.00**
0.00	Excess of Expenditure Over Income	0.00	2410000.00	Closing Balance	449029.00
2410000.00		**4251600.00**	**2410000.00**		**4251600.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-187 : Understanding the mechanism of induction of innate immunity in plants by the Xanthomonas Diffusible signal factor (DSF)

PI : Dr Subhadeep Chatterjee

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00.00	Opening Balance	1368000.00		Opening Balance	0.00
1368000.00	Grant In Aid	0.00	0.00	Salaries - Manpower	50323.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	35000.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1368000.00		**1368000.00**	**0.00**		**85323.00**
0.00	Excess of Expenditure Over Income	0.00	1368000.00	Closing Balance	1282677.00
1368000.00		**1368000.00**	**1368000.00**		**1368000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-188 : Identification of Novel Genes for Intellectual Disability

PI : Dr Aneek Das Bhowmik
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
	Opening Balance	1450000.00		Opening Balance	0.00
1450000.00	Grant In Aid	0.00	0.00	Salaries - Manpower	605000.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	4620.00
0.00		0.00	0.00	Travel	7486.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1450000.00		**1450000.00**	**0.00**		**617106.00**
0.00	Excess of Expenditure Over Income	0.00	1450000.00	Closing Balance	832894.00
1450000.00		**1450000.00**	**1450000.00**		**1450000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-189 : Characterization of glycosylphosphatidylinositol-linked aspartyl proteases in Candida glabrata: role in pathosgenicity
PI : Dr Rupinder Kaur
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	16858467.00		Opening Balance	0.00
16858467.00	Grant In Aid	5629854.00	0.00	Salaries - Manpower	557793.00
0.00		0.00	0.00	Consumables	3352016.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	94351.00
0.00		0.00	0.00	Overheads	460416.00
0.00		0.00	0.00	Equipment	600000.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
16858467.00		**22488321.00**	**0.00**		**5064576.00**
0.00	Excess of Expenditure Over Income	0.00	16858467.00	Closing Balance	17423746.00
16858467.00		**22488321.00**	**16858467.00**		**22488321.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-190 : Exploring mycobacteriophages to source novel factors / regulators of bacterial transcription machinery

PI : Dr Shweta Singh

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	1100000.00		Opening Balance	0.00
1100000.00	Grant In Aid	0.00	0.00	Salaries - Manpower	616155.00
0.00		0.00	0.00	Consumables	188819.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	50000.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1100000.00		**1100000.00**	**0.00**		**854974.00**
0.00	Excess of Expenditure Over Income	0.00	1100000.00	Closing Balance	245026.00
1100000.00		**1100000.00**	**1100000.00**		**1100000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-191 : "Human Frontier Science Program Reseearch Grant - A comprehensive approach towards the chemistry & biology of polyphosphate: the forgotten biopolymer

PI : Dr Rashna Bhandari

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00		Opening Balance	0.00
0.00	Grant In Aid	7765092.00	0.00	Salaries - Manpower	1144105.00
0.00		0.00	0.00	Consumables	500000.00
0.00		0.00	0.00	Contingencies	177341.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	186051.00
0.00		0.00	0.00	Equipment	39060.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**7765092.00**	**0.00**		**2046557.00**
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	5718535.00
0.00		**7765092.00**	**0.00**		**7765092.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-192 : Design of peptide inhibitor(s) for the bacterial trancription terminator Rho, a potent drug target

PI : Dr Ranjan Sen

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00		Opening Balance	0.00
0.00	Grant In Aid	3819000.00	0.00	Salaries - Manpower	254800.00
0.00		0.00	0.00	Consumables	1105283.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	2000000.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**3819000.00**	**0.00**		**3360083.00**
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	458917.00
0.00		**3819000.00**	**0.00**		**3819000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-193 : Screening for male infertility markers in the human Yq12 heterochromatic block

PI : Dr Ashwin B Dalal

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00		Opening Balance	0.00
0.00	Grant In Aid	1050000.00	0.00	Salaries - Manpower	44032.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	4621.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**1050000.00**	**0.00**		**48653.00**
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	1001347.00
0.00		**1050000.00**	**0.00**		**1050000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-194 : Mechanisms and regulation of iron transportin the pathogenic yeast Candida glabrata

PI : Dr Rupinder Kaur

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00		Opening Balance	0.00
0.00	Grant In Aid	500000.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	289966.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**500000.00**	**0.00**		**289966.00**
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	210034.00
0.00		**500000.00**	**0.00**		**500000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-195 : Molecular and biophysical characterization of the ESAT-6: 2M complex and its effect on intracellular iron concentration and macrophage anti-mycobacterial effector responses"

PI : Dr Sangita Mukhopadhyay

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00		Opening Balance	0.00
0.00	Grant In Aid	1285000.00	0.00	Salaries - Manpower	109200.00
0.00		0.00	0.00	Consumables	288596.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	15000.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**1285000.00**	**0.00**		**412796.00**
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	872204.00
0.00		**1285000.00**	**0.00**		**1285000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

p-196 : Exploring the volatome of noncommunicable diseases as a promising, innovative and integrating approach for its rapid diagnostics"

PI : Dr H A Nagarajaram

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00		Opening Balance	0.00
0.00	Grant In Aid	1281744.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	117723.30
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**1281744.00**	**0.00**		**117723.30**
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	1164020.70
0.00		**1281744.00**	**0.00**		**1281744.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-197 : National Post Doctoral Fellowship

PI : Dr Madhu Babu Battu

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00		Opening Balance	0.00
0.00	Grant In Aid	960000.00	0.00	Salaries - Manpower	330000.00
0.00		0.00	0.00	Consumables	46270.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**960000.00**	**0.00**		**376270.00**
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	583730.00
0.00		**960000.00**	**0.00**		**960000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-198 : Whole Genome Sequencing for characterization of novel genes and de novo balanced chromosomal rearrangements in human genectic disorders"

PI : Dr Ashwin Dalal

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00		Opening Balance	0.00
0.00	Grant In Aid	2556000.00	0.00	Salaries - Manpower	62400.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**2556000.00**	**0.00**		**62400.00**
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	2493600.00
0.00		**2556000.00**	**0.00**		**2556000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-199 : Investigating cellular processes and pathways controlled by phosphatases

PI : Dr M Subba Reddy

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00		Opening Balance	0.00
0.00	Grant In Aid	4013536.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**4013536.00**	**0.00**		**0.00**
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	4013536.00
0.00		**4013536.00**	**0.00**		**4013536.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-200 : Characterization of divergent functions of ARID1A and ARID1B: the two alternative DNA binding constituents of the human SWI/SNF chromatic remodelling complex

PI : Dr M D Bashyam

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00		Opening Balance	0.00
0.00	Grant In Aid	1830000.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	23801.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**1830000.00**	**0.00**		**23801.00**
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	1806199.00
0.00		**1830000.00**	**0.00**		**1830000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-201 : Defining the functions of MLL in mitosis

PI : Dr Shweta Tyagi

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00		Opening Balance	0.00
0.00	Grant In Aid	1241000.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**1241000.00**	**0.00**		**0.00**
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	1241000.00
0.00		**1241000.00**	**0.00**		**1241000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-202 : To decipher the role of MLL Complex in the process of cytokinesis

PI : Dr Shweta Tyagi
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00		Opening Balance	0.00
0.00	Grant In Aid	603000.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**603000.00**	**0.00**		**0.00**
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	603000.00
0.00		**603000.00**	**0.00**		**603000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-203 : Investigation of a potential novel function of fission yeast sirtuin family histone deacetylase Hst4 in regulation of DNA replication

PI : Dr Devyani Haldar
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00		Opening Balance	0.00
0.00	Grant In Aid	1186706.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**1186706.00**	**0.00**		**0.00**
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	1186706.00
0.00		**1186706.00**	**0.00**		**1186706.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
COE1/CORE : COE for Genetics and Genomics of silkmoths

PI : Dr. J. Nagaraju
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	11970751.00	Opening Balance	12271928.00
8335000.00	Grant In Aid	8768000.00	7219530.00	Salaries - Manpower	6942349.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
8335000.00		**8768000.00**	**19190281.00**		**19214277.00**
10855281.00	Excess of Expenditure Over Income	10446277.00	0.00	Closing Balance	0.00
19190281.00		**19214277.00**	**19190281.00**		**19214277.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
COE1/P-I : Comparative and function genomics of silkmoths.

PI : Dr. J. Nagaraju
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	355503.00	Opening Balance	410893.00
638000.00	Grant In Aid	775000.00	193390.00	Salaries - Manpower	143520.00
0.00		0.00	500000.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
638000.00		**775000.00**	**1048893.00**		**554413.00**
410893.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	220587.00
1048893.00		**775000.00**	**1048893.00**		**775000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE1/P-II : Development of RNA interference (RNAi) based nuclear polyhedrosis virus (NPV) resistant transgenic silkmoths.

PI : Dr. J. Nagaraju

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	419966.00	Opening Balance	593919.00
491000.00	Grant In Aid	643000.00	364953.00	Salaries - Manpower	193527.00
0.00		0.00	300000.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
491000.00		**643000.00**	**1084919.00**		**787446.00**
593919.00	Excess of Expenditure Over Income	144446.00	0.00	Closing Balance	0.00
1084919.00		**787446.00**	**1084919.00**		**787446.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE1/P-III : Identification and Characterization of micro RNAs and their targets in silkmoth genome.

PI : Dr. J. Nagaraju

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	475030.00	Opening Balance	448230.00
1086000.00	Grant In Aid	1090000.00	709200.00	Salaries - Manpower	225358.00
0.00		0.00	350000.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1086000.00		**1090000.00**	**1534230.00**		**673588.00**
448230.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	416412.00
1534230.00		**1090000.00**	**1534230.00**		**1090000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
COE-I/P-IV : Identification and characterization ofimmune response genes of silkmoths.

PI : Dr. J. Nagaraju
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	21563.00	Opening Balance	30963.00
331000.00	Grant In Aid	442000.00	140400.00	Salaries - Manpower	107640.00
0.00		0.00	200000.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
331000.00		**442000.00**	**361963.00**		**138603.00**
30963.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	303397.00
361963.00		**442000.00**	**361963.00**		**442000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
COE2/CORE : DBT Centre of Excellence for Microbial Biology

PI : Dr J Gowrishankar, Dr K Anupama, Dr Abhijit A Sardesai, Dr R
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	23840815.00	Opening Balance	23840815.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**23840815.00**		**23840815.00**
23840815.00	Excess of Expenditure Over Income	23840815.00	0.00	Closing Balance	0.00
23840815.00		**23840815.00**	**23840815.00**		**23840815.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2/P-A : Occurrence of R-loops (RNA-DNA hybrids) from nascent untranslated transcripts i E. Coli

PI : Dr. J Gowrishankar, Dr.K. Anupama

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	1354252.00	Opening Balance	1354252.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**1354252.00**		**1354252.00**
1354252.00	Excess of Expenditure Over Income	1354252.00	0.00	Closing Balance	0.00
1354252.00		**1354252.00**	**1354252.00**		**1354252.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2/P-B : Molecular genetic approaches to dissect the physiology of osmoadptation in Escherichia coli

PI : Dr. J. Gowrishankar, Dr. Abhijit A Sardesasi

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	1275609.00	Opening Balance	1275609.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**1275609.00**		**1275609.00**
1275609.00	Excess of Expenditure Over Income	1275609.00	0.00	Closing Balance	0.00
1275609.00		**1275609.00**	**1275609.00**		**1275609.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2/P-C : Functional role and mechanisms of the ArgO exporter and the transcriptional regulator ArgP in E. Coli

PI : Dr. J Gowrishankar, Dr. Ranjan Sen

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	473354.00	Opening Balance	473354.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**473354.00**		**473354.00**
473354.00	Excess of Expenditure Over Income	473354.00	0.00	Closing Balance	0.00
473354.00		**473354.00**	**473354.00**		**473354.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2/P-1 : Addressing functiional properties of E. coli through genome-wide protein-protein linkage analysis

PI : Dr. J Gowrishankar

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	684083.00	Opening Balance	684083.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**684083.00**		**684083.00**
684083.00	Excess of Expenditure Over Income	684083.00	0.00	Closing Balance	0.00
684083.00		**684083.00**	**684083.00**		**684083.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
COE2/P-2 : Mechanism of transcription termination and antitermination in Escherichia coli

PI : Dr. Ranjan Sen
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	1441181.00	Opening Balance	1441181.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**1441181.00**		**1441181.00**
1441181.00	Excess of Expenditure Over Income	1441181.00	0.00	Closing Balance	0.00
1441181.00		**1441181.00**	**1441181.00**		**1441181.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
COE2-II-Core : DBT Centre of Excellence for Microbiology - Phase II

PI : Dr J Gowrishankar
Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
9523323.00	Opening Balance	1736568.00		Opening Balance	0.00
0.00	Grant In Aid	3447000.00	4137634.00	Salaries - Manpower	2455983.00
0.00		0.00	832837.00	Consumables	91924.00
0.00		0.00	20593.00	Contingencies	0.00
0.00		0.00	11018.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	2134673.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	650000.00	Transfer of Funds	0.00
9523323.00		**5183568.00**	**7786755.00**		**2547907.00**
0.00	Excess of Expenditure Over Income	0.00	1736568.00	Closing Balance	2635661.00
9523323.00		**5183568.00**	**9523323.00**		**5183568.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2-II/P-A : Role of R-loops (RNA-DNA hybrids) in generatin of transcription -replication conflicts in E.Coli

PI : Dr J Gowrishankar

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
803300.00	Opening Balance	0.00		Opening Balance	26068.00
0.00	Grant In Aid	1061000.00	629368.00	Salaries - Manpower	928535.00
0.00		0.00	200000.00	Consumables	330000.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
803300.00		**1061000.00**	**829368.00**		**1284603.00**
26068.00	Excess of Expenditure Over Income	223603.00	0.00	Closing Balance	0.00
829368.00		**1284603.00**	**829368.00**		**1284603.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2-II/P-B : Role of the ArgP transcriptional regulator and metabolism of basic amino acids Arg and Lys in E.coli

PI : Dr J Gowrishankar

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
300000.00	Opening Balance	0.00		Opening Balance	510077.00
0.00	Grant In Aid	488000.00	610077.00	Salaries - Manpower	603955.00
0.00		0.00	200000.00	Consumables	350000.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
300000.00		**488000.00**	**810077.00**		**1464032.00**
510077.00	Excess of Expenditure Over Income	976032.00	0.00	Closing Balance	0.00
810077.00		**1464032.00**	**810077.00**		**1464032.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2-II/P-C : Investigating global RNA turnover mechanisms and their interplay with Rho-dependent transcription termination in E. coli

PI : Dr K Anupaman

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
803300.00	Opening Balance	577635.00		Opening Balance	0.00
0.00	Grant In Aid	1061000.00	25665.00	Salaries - Manpower	0.00
0.00		0.00	200000.00	Consumables	330000.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
803300.00		**1638635.00**	**225665.00**		**330000.00**
0.00	Excess of Expenditure Over Income	0.00	577635.00	Closing Balance	1308635.00
803300.00		**1638635.00**	**803300.00**		**1638635.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2-II/P-D : Molecular, genetic and biochemical studies on physiology of K+ION homeostatis and the regulatory mechanisms mediating avoidance of its imbalance in Escherichia coli

PI : Dr Abhijit A Sardesai

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
500000.00	Opening Balance	300000.00		Opening Balance	0.00
0.00	Grant In Aid	496000.00	0.00	Salaries - Manpower	0.00
0.00		0.00	200000.00	Consumables	357000.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
500000.00		**796000.00**	**200000.00**		**357000.00**
0.00	Excess of Expenditure Over Income	0.00	300000.00	Closing Balance	439000.00
500000.00		**796000.00**	**500000.00**		**796000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2-II/P-E : Understanding (p) ppGpp-mediated functions in E.Coliby deciphering the physiology of strain lacking (p)ppGpp OR altered in its metabolism

PI : Dr J Gowrishankar

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
1076226.00	Opening Balance	713939.00		Opening Balance	0.00
0.00	Grant In Aid	866000.00	301291.00	Salaries - Manpower	326400.00
0.00		0.00	60996.00	Consumables	271000.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1076226.00		**1579939.00**	**362287.00**		**597400.00**
0.00	Excess of Expenditure Over Income	0.00	713939.00	Closing Balance	982539.00
1076226.00		**1579939.00**	**1076226.00**		**1579939.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2-II/P-1 : In vivo studies on molecular mechanism of Rho-dependent transcription termination

PI : Dr Ranjan Sen

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
2071265.00	Opening Balance	504781.00		Opening Balance	0.00
650000.00	Grant In Aid	2100000.00	1056820.00	Salaries - Manpower	1049273.00
0.00		0.00	1000000.00	Consumables	591000.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	159664.00	Equipment	15503.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2721265.00		**2604781.00**	**2216484.00**		**1655776.00**
0.00	Excess of Expenditure Over Income	0.00	504781.00	Closing Balance	949005.00
2721265.00		**2604781.00**	**2721265.00**		**2604781.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P.I: Others

Receipts and Payments Account from 01/04/2016 to 31/03/2017

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00		Opening Balance	0.00
0.00	Grant In Aid	2028298.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**2028298.00**	**0.00**		**0.00**
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	2028298.00
0.00		**2028298.00**	**0.00**		**20285298.00**

फोटो गैलरी
Photo Gallery

Flag hoisting at CDFD Uppal Campus on the occasion of Independence Day

EU-Indian Cooperation (INDIGO) Meeting on Human Volatome

EU-Indian Cooperation (INDIGO) Meeting on Human Volatome

Meeting on Molecular Microbiology (Mcube)

Meeting on Molecular Microbiology (Mcube)

Hindi Day

OUTREACH ACTIVITY

DST Inspire Camp

Kendriya Vidyalaya Regional Level National Children Science Congress, 2016

Conference on Cancer Biology at Silver Jubilee Government College, Kurnool

Inauguration of the CDFD Uppal Campus

Inauguration of the CDFD Uppal Campus

Second India International Science Festival (IISF-2016)

Second India International Science Festival (IISF-2016)

Foundation Day Lecture by Dr Rajesh S Gokhale, NII, New Delhi

Glimpses of the CDFD Foundation Day Celebrations