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नामपल्ली, हैदराबाद - 500 001

Centre for DNA Fingerprinting and Diagnostics

Nampally, Hyderabad - 500 001

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अधिदेश
Mandate

अघिदेश

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 प्रयोगशालाओं, 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;

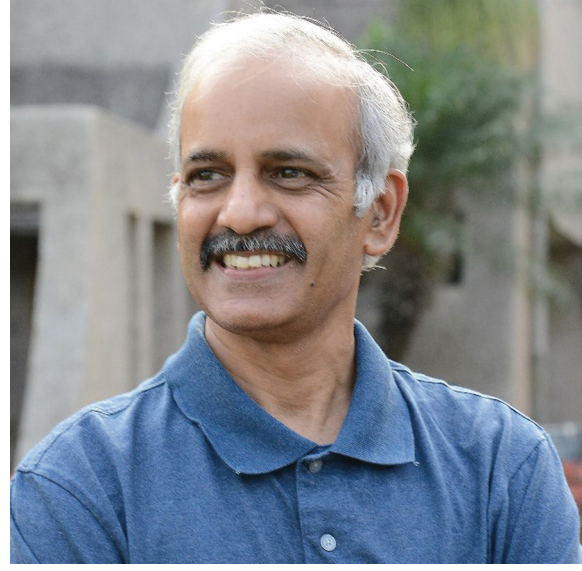
- xv. For investing the funds of or money entrusted to the Centre, to open such securities or in such manner as may from time to time be determined by the Governing Council and to sell or transpose such investment;
- xvi. To do all such other lawful acts as may be necessary, incidental or conducive to the attainment of all or any of the above objectives;
- xvii. To institute Professorships, other faculty positions, fellowships including visiting fellowships, research and cadre positions, scholarships, etc. for realizing the objectives of the Centre;
- xviii. To establish, maintain and manage laboratories, workshops, stores, library, office and other facilities for scientific and technological work of the Centre;
- xix. To acquire or transfer technical know-how from/to entrepreneurs and industries; and
- xx. To register patents, designs & technical know-how that may be developed by the Centre and transfer any portion of such patents/designs/technical know-how in the interest of the Centre.

निदेशक का संदेश
From the Director's Desk

निदेशक का संदेश

मुझे डीएनए फिंगरप्रिंटिंग एवं निदान केन्द्र (सीडीएफडी), हैदराबाद की वार्षिक रिपोर्ट करते हुए अत्यंत हर्ष का अनुभव हो रहा है। यह संस्थान 1996 में स्थापित किया गया था और इसने तब से विविध गतिविधियों में उत्कृष्टता प्राप्त की है। संस्थान द्वारा डीएनए फिंगरप्रिंटिंग, मानव आनुवंशिक विकारों के लिए नैदानिक परीक्षणों और शुद्धता के लिए बासमती चावल के विश्लेषण के क्षेत्रों में सेवाएं प्रदान की जाती हैं तथा यह आधुनिक जीव विज्ञान के विभिन्न विषयों में मूलभूत अनुसंधान गतिविधियों में भी संलग्न है। इस वर्ष केन्द्र के कुछ समूहों की प्रमुख उपलब्धियों और अनुसंधान प्राप्तियों का सारांश आगे प्रस्तुत किया गया है, जिसके विवरण इस रिपोर्ट में संलग्न अलग अलग प्रयोगशालाओं द्वारा दिए गए विवरणों में शामिल हैं।

वर्ष 2013-14 की अवधि के दौरान डीएनए फिंगरप्रिंटिंग सेवा प्रयोगशाला को देश भर की न्यायिक और कानून प्रवर्तन एजेंसियों द्वारा लगभग 350 मामले अग्रेषित किए गए थे, जो पिछले वर्षों से 100 प्रतिशत की वृद्धि दर्शाते हैं। सीडीएफडी ने अनेक राज्य सरकारों के साथ डीएनए रूपांश सेवाएं प्रदान करने तथा विधि विज्ञान वैज्ञानिकों को प्रशिक्षण देने के समझौता ज्ञापनों (एमओयू) पर हस्ताक्षर किए हैं। इस प्रयोगशाला ने पिछले वर्ष अनेक प्रमुख मामलों में डीएनए फिंगर प्रिंटिंग सेवाएं प्रदान की, जैसे उत्तराखण्ड में भारतीय वायुसेना के हैलीकॉप्टर की दुर्घटना और आंध्र प्रदेश के महबूब नगर जिले में बस में आग लगने की दुर्घटना में पीड़ितों की पहचान। एक अन्य बड़ा मामला जो अभी जारी है, जून 2013 में उत्तराखण्ड में बाढ़ और भूस्खलन की त्रासदी में लगभग 575 व्यक्तियों की पहचान का है। नैदानिक सेवाओं के क्षेत्र में सीडीएफडी के नैदानिक प्रभाग द्वारा विभिन्न आनुवंशिक विकारों वाले 3500 रोगियों को आनुवंशिक मूल्यांकन प्रदान किया गया। इसे निजाम इंस्टीट्यूट ऑफ मेडिकल साइंसिस, हैदराबाद के साथ समझौता ज्ञापन के तहत नजदीकी और सफल सहयोग द्वारा पूरा किया गया जिसके तहत यहां एक चिकित्सा आनुवंशिक विभाग स्थापित किया गया है। क्लिनिकल कोशिका आनुवंशिकी और



क्लिनिकल आण्विक आनुवंशिकी में अध्येतावृत्ति कार्यक्रम आरंभ किए गए हैं। विभिन्न लाइसोसोमल चरण के विकारों में अभिज्ञात नए उत्परिवर्तनों के कार्यात्मक विश्लेषण से इन भिन्नताओं के बेहतर लाक्षणिकरण में सहायता मिली है। मायोपैथी के एक रोगी में गुणसूत्र के सफल मानचित्रण का कार्य पूरा किया गया है और 280 के बी क्षेत्र तक सफलता बिन्दु का क्षेत्र ज्ञात किया गया है। बासमती डीएनए विश्लेषण के लिए एपिडा-सीडीएफडी केन्द्र में वर्ष के दौरान 200 से अधिक बासमती चावल के नमूनों का आँकलन शुद्धता हेतु किया गया।

कोशिका चक्र नियमन प्रयोगशाला ने दर्शाया है कि एमएलएल प्रोटीन एस चरण के आगे बढ़ने और उचित पृथक्करण का नियमन करता है तथा कोशिका चक्र के एम चरण के दौरान एक नई प्रक्रिया द्वारा साइटोकाइनेसिस होता है तथा एमएलएल के माइटोटिक कार्यों में प्रोटीन डब्ल्यूआरएडी भी भाग लेते हैं। कोशिका मृत्यु और उत्तरजीविता प्रयोगशाला के अनुसंधानकर्ता कोशिका उत्तरजीविता तथा मृत्यु की प्रक्रियाओं में विभिन्न प्रोटीनों के नियमन के अंतःक्रियात्मक नेटवर्क के कार्यात्मक लाक्षणिकरण और इसे अभिज्ञात करने के लिए कार्यरत हैं।

कोशिका संकेतन प्रयोगशाला ने प्रदर्शित किया है कि आरएनए पॉलीमरेज 1 के पायरोफॉस्फोराइलेशन करने वाले घटकों द्वारा ईस्ट में राइबोसोम बायोजेनेसिस के नियमन सहित इनोसिटॉल पायरोफॉस्फेट आईपी7 अनेक कार्य करता है; समजात पुनर्योजन द्वारा डीएनए की मरम्मत; तथा प्लेटलेट कार्य का नियमन करता है। इनोसिटॉल पायरोफॉस्फेट तथा इनोर्गनिक पॉलीफॉस्फेट के बीच ईस्ट और चूहों में उपापचय लिंक संरक्षित है। ऊतक विशिष्ट प्रोटीन-प्रोटीन अंतःक्रिया नेटवर्कों पर कार्य करते समय अभिकलनात्मक जीव विज्ञान प्रयोगशाला ने दर्शाया है कि स्प्लाइस परिवर्तियों से भरपूर नोड आम तौर पर नेटवर्क में केन्द्रीय स्थानों पर रहते हैं।

न्यूरोस्पोरा आनुवंशिकी प्रयोगशाला द्वारा इस कवक में जोड़ा विहिन डीएनए द्वारा समसूत्री साइलेंसिंग की असामान्य घटना की प्रक्रिया की जांच का अध्ययन किया गया है और इसमें हिटेरोकेरियाँ में नाभिक सीमित' व्यवहार की संकल्पना की जांच की गई है। कवक रोगाणुजनन प्रयोगशाला द्वारा दर्शाया गया है CgMED2 जीन, जो आरएनए पॉलीमरेज 2 मीडिएटर कॉम्प्लेक्स की सिरे की उप इकाई के लिए कोड करता है, यह कैडिडा ग्लेआब्राटा में एजोल कवक रोधी द्वारा आधारभूत और अर्जित दोनों प्रतिरोधकताओं के लिए आवश्यक है, इसके अलावा कवक की सिरे की उप इकाई का मीडिएटर कॉम्प्लेक्स पहली बार फैले हुए कैडीडियासिस के म्यूरिन मॉडल में मानव मैक्रोफेज में उत्तरजीविता और रोगाणुजनकता तथा एपिथिलियल कोशिकाओं के साथ जुड़ाव के लिए निहित किए गए हैं।

स्तनी आनुवंशिकी प्रयोगशाला में किए गए अनुसंधान से कैसर जनन और विकास में डीएनए मेथिल ट्रांसफरेज़ डीएनएमटी31 और डीएनएमटी 2 की भूमिका का पता चला है। इस समूह ने माइक्रोबैक्टीरियम ट्यूबरकुलोसिस से चुनौती देने पर पोषी कोशिका में होने वाले एपिजेनेटिक बदलावों को भी अभिज्ञात किया है। आण्विक कोशिका जीव विज्ञान प्रयोगशाला के अध्ययनों से प्रकट हुआ है कि ऑक्सीडेटिव तनाव मैक्रोफेज में सह उद्दीपक संकेतन के मॉड्यूलन के बिना एमएचसी वर्ग 2 प्रतिबंधित एंटीजन प्रस्तुतीकरण का संदमन करता है। इस समूह के अनुसंधान से आगे इन परिस्थितियों के तहत

वर्ग 2 एंटीजन प्रस्तुतीकरण के डाउन रेगुलेशन में कैलमोडुलिन सी आरईएल सिगनलिंग की महत्वपूर्ण भूमिका का संकेत मिला। ये यह साक्ष्य भी प्रदान करते हैं कि एम. ट्यूबरकुलोसिस के पीपीई18 प्रोटीन से टीएनएफ-अल्फा और आरईएल-1 बीटा स्तर में कमी आती है और ई. कोलाई से उद्दीपित सेप्टिक आघात में चूहों की उत्तरजीविता बढ़ जाती है, इस प्रकार इस प्रस्ताव को समर्थन मिलता है कि पीपीई18 सेप्टिक आघात के नियंत्रण के लिए एक प्रतिरक्षी मॉड्यूलटर के रूप में इस्तेमाल किया जा सकता है।

अनुलेखन प्रयोगशाला ई. कोलाई में आरएचओ कारक आश्रित अनुलेखन समापन और समापन रोधी आण्विक आधार को समझने में संलग्न है। समूह ने दर्शाया है कि आरएचओ का प्राथमिक रूप से आरएनए बंधन कार्य जीवे रूप से अनावश्यक है और यह कि एनयूएसए प्रोटीन आरएचओ कार्य के सामान्य एंटागोनिस्ट्स के रूप में कार्य करता है। आण्विक ओंकोलॉजी प्रयोगशाला के अध्ययनों में पहली बार लिंच सिंड्रोम से जुड़े कोलोरेक्टल कार्सिनोमा की उपश्रेणी के अस्तित्वों का पता चला है जो डीएनए में बेमेल मरम्मत के लिए प्रवीण हैं।

जीवाण्विक आनुवंशिकी प्रयोगशाला ने जीवाणु में अनुलेखन संबद्ध आरएनए-डीएनए हाइब्रिड (आर-लूप्स) से उत्पन्न विपथित गुणसूत्र द्विगुणन के नए मॉडल का प्रस्ताव दिया है। पादप-सूक्ष्मजीव अंतःक्रिया प्रयोगशाला में प्रदर्शित किया है कि यह बैक्टीरिया कोरम सेंसिंग प्रतिक्रिया में अपनी न बदलने योग्य गैर आनुवंशिक विषम जनकता दर्शाता है, अतः यह बैट हैजिंग उत्तरजीविता कार्यनीति के तौर पर कार्य कर सकती है। इस समूह ने जैथोमोनास ओरिजी पीवी. ओरिजीकोला नामक चावल के नॉन वेस्कुलर रोगाणु में लौह अधिग्रहण के नियमन में कोरम सेंसिंग की एक नई भूमिका भी अभिज्ञात की है और दर्शाया है कि पौधे की वृद्धि में लौह अधिग्रहण तंत्र एक महत्वपूर्ण भूमिका निभाता है और यह इस रोगाणु की रोगाणुजनक विशेषता है।

आण्विक आनुवंशिकी प्रयोगशाला में किए गए कार्य से यह सुझाने का सशक्त साक्ष्य मिलता है कि रेशमकीट सेरोइन एक नए सूक्ष्मजीव रोधी प्रोटीन के तौर पर कार्य

करता है जो वायरस और बैक्टीरिया दोनों के खिलाफ रक्षा में संलग्न है। संक्रमण के आरंभिक चरण में वायरस द्वारा कोड किए गए माइक्रो आरएनए (बीएमएनपीवी - एमआईआर-3) से वायरस के विलंबित जीन का नियमन होता है, जो मेजबान की आरंभिक प्रतिरक्षा प्रतिक्रिया से वायरस के पलायन को सक्षम बनाता है। यह प्रयोगशाला पारजीनी वायरस रोधी रेशम कीट के बहु स्थानिक क्षेत्र परीक्षणों का आयोजन भी करती है, जिन्हें सरकार की आनुवंशिक प्रकटन समीक्षा समिति (आरसीजीएम) द्वारा अनुमोदन हाल ही में प्रदान किया गया है।

इस वर्ष भी पिछले वर्षों के समान सीडीएफडी संकाय और अध्येताओं को प्रतिष्ठित पुरस्कार और सम्मान प्राप्त हुए हैं। इसमें अन्य के अलावा शामिल हैं युवा जैव चिकित्सा वैज्ञानिक के लिए आईसीएमआर अध्येतावृत्ति, कैरियर विकास के लिए राष्ट्रीय जैव विज्ञान पुरस्कार, वरिष्ठ नवाचारी युवा जैव प्रौद्योगिकीविद पुरस्कार, भारतीय राष्ट्रीय विज्ञान अकादमी (इंसा) और भारतीय विज्ञान अकादमी की अध्येतावृत्तियां, भारत-अमेरिकी अनुसंधान अध्येतावृत्ति, गुहा अनुसंधान सम्मेलन सदस्यता, एपी अकादमी ऑफ साइंस का यंग साइंटिस्ट पुरस्कार, इंसा यंग साइंटिस्ट पदक, डीएसटी-रमन चरपक अध्येतावृत्ति

और के वी राव अनुसंधान पुरस्कार। इस अवधि के दौरान आठ अनुसंधान अध्येताओं को पीएचडी डिग्री प्रदान की गई। अनेक पोस्ट डॉक्टरल अध्येताओं, परियोजना सहयोगियों तथा ग्रीष्म कालीन प्रशिक्षुओं ने सीडीएफडी के लिए कार्य किया और केन्द्र की गतिविधियों में महत्वपूर्ण भूमिका निभाई।

इस वर्ष केन्द्र के स्थायी परिसर के लिए सरकार की वित्त व्यय समिति का अनुमोदन प्रदान किया गया था और निर्माण की गतिविधि तेजी से जारी हैं। हमें आशा है कि आने वाले वर्ष में जंतु सुविधा कार्यशील होगी।

मैं अपार सहयोग के प्रति आभार व्यक्त करता हूँ जो केन्द्र को शासी परिषद, अनुसंधान क्षेत्र पैनल - वैज्ञानिक सलाहकार समिति, शैक्षिक / वित्त / भवन समितियों और जैव प्रौद्योगिकी विभाग से इसकी सभी गतिविधियों के लिए प्राप्त हुआ। मैं हमारी गतिविधियों और उपलब्धियों में समर्थन देने के लिए सभी सदस्यों और अधिकारियों के समय और प्रयास हेतु धन्यवाद देता हूँ।

मैं सीडीएफडी परिवार के प्रति भी हार्दिक आभार व्यक्त करता हूँ, जिन्होंने केन्द्र के कार्यक्रमों और विकास में महत्वपूर्ण भूमिका निभाई है।

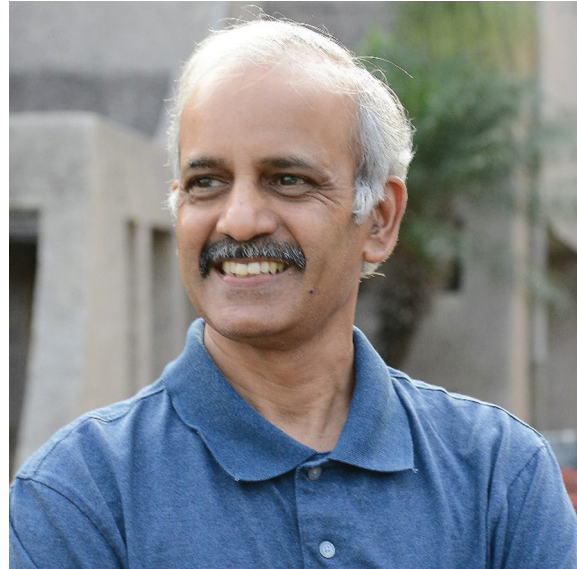
ज गौरीशंकर

31 मार्च, 2014

Director's Message

I have great pleasure in presenting the Annual Report of the Centre for DNA Fingerprinting and Diagnostics (CDFD), Hyderabad. The institute was established in 1996 and has since excelled its diverse activities. The institute provides services in the areas of DNA fingerprinting, diagnostic tests for human genetic disorders and analysis of basmati rice for purity, and is also engaged in basic research activities in different disciplines of modern biology. A summary of the major achievements and research findings from some groups of the Centre this year is given below, the details of which are covered in the descriptions by the individual laboratories that are enclosed in this Report.

During the 2013-14 period, the Laboratory of DNA Fingerprinting Services was forwarded about 350 cases by the judiciary and law enforcing agencies across the country, which represents a 100% increase above that in previous years. CDFD has entered into memoranda of understanding (MoUs) with several State Governments to provide DNA profiling services and to impart training to forensic scientists. This laboratory provided DNA fingerprinting services in several prominent cases last year, such as identification of victims of an Indian Air Force helicopter crash in Uttarakhand and of a bus fire mishap in Mahabubnagar district of Andhra Pradesh. Another major case that is currently ongoing is that of identification of around 575 victims of the floods-and-landslide tragedy at Uttarakhand in June 2013. In the area of diagnostic services, the Diagnostics division of CDFD provided genetic evaluation to around 3500 patients for various genetic diseases. This was achieved in close and successful collaboration under an MoU with the Nizam's Institute of Medical Sciences, Hyderabad under which a Medical Genetics department has been established there. Fellowship programs in Clinical Cytogenetics and Clinical Molecular Genetics have been initiated. Functional analysis of novel mutations identified in various



lysosomal storage disorders helped in better characterization of these variations. Chromosomal breakpoint mapping in a patient with myopathy is under way and the breakpoint region has been narrowed to a 280 kb region. The APEDA-CDFD Centre for Basmati DNA Analysis tested over 200 basmati rice samples for their purity during the year.

The Laboratory of Cell Cycle Regulation has shown that the MLL protein regulates S phase progression and proper segregation and cytokinesis during M phase of the cell cycle by a novel mechanism, and that the protein WRAD also participates in the mitotic functions of MLL. Researchers in the Laboratory of Cell Death & Cell Survival are working to identify and functionally characterize the interacting network of various proteins in regulation of cell survival and cell death processes.

The Laboratory of Cell Signalling has demonstrated that the inositol pyrophosphate IP_7 performs multiple functions, including regulation of ribosome biogenesis in yeast by pyrophosphorylating components of RNA polymerase I; DNA repair by homologous recombination; and regulation of platelet function. The metabolic link between inositol pyrophosphates and inorganic

polyphosphate is conserved in yeast and mice. While working on tissue-specific protein-protein interaction networks, the Laboratory of Computational Biology has shown that nodes enriched with splice variants usually occupy central positions in the networks.

The Laboratory of Neurospora Genetics is undertaking studies to examine the mechanism of the unusual phenomenon of meiotic silencing by unpaired DNA in this fungus, and to test the hypothesis of “nucleus-limited” behaviour in heterokaryons. The Laboratory of Fungal Pathogenesis showed that the *CgMED2* gene, which codes for a tail subunit of the RNA polymerase II mediator complex, is required for both basal and acquired resistance to azole antifungals in *Candida glabrata*; additionally, a tail subunit of the fungal Mediator complex has been implicated for the first time in adherence to epithelial cells, survival in human macrophages, and virulence in a murine model of disseminated candidiasis.

Work in the Laboratory of Mammalian Genetics has dissected the role of DNA methyltransferases *Dnmt3l* and *Dnmt2* in carcinogenesis and development. The group has also identified epigenetic changes that host cells undergo when challenged with *Mycobacterium tuberculosis*. Studies in the Laboratory of Molecular Cell Biology have revealed that oxidative stress inhibits MHC class II-restricted antigen presentation without modulating the co-stimulatory signaling in macrophages. The research of this group further indicates an important role of calmodulin-c-rel signaling in the down-regulation of class II antigen presentation under these conditions. They also provide evidence that PPE18 protein of *M. tuberculosis* reduces TNF- α and IL-1 β levels and increases survival in mice subjected to *E. coli*-induced septic shock, thus, supporting the proposal that PPE18 can find use as an immunomodulator to control septic shock.

The Laboratory of Transcription is engaged in understanding the molecular basis of Rho factor-dependent transcription termination and antitermination in *E. coli*. The group has shown that primary RNA binding function of Rho is redundant *in vivo*, and that the NusA protein functions as a general antagonist of Rho function. Studies in the Laboratory of Molecular Oncology have revealed for the first time the existence of a sub-category of Lynch syndrome-associated colorectal carcinomas that are proficient for DNA mismatch repair.

The Laboratory of Bacterial Genetics has proposed a new model for aberrant chromosome replication arising from transcription-associated RNA-DNA hybrids (R-loops) in bacteria. The Laboratory of Plant-Microbe Interactions has demonstrated that bacteria exhibit reversible non-genetic heterogeneity in their quorum sensing response, which may therefore serve as a bet-hedging survival strategy. The group has also identified a new role for quorum sensing in regulation of iron uptake in *Xanthomonas oryzae* pv. *oryzicola*, a non-vascular pathogen of rice, and have shown that the ferric uptake system plays an important role in the *in planta* growth and virulence characteristics of this pathogen.

Work carried out in the Laboratory of Molecular Genetics provides strong evidence to suggest that silkworm serpins function as novel antimicrobial proteins that are involved in defense against viruses as well as bacteria. A virus-encoded microRNA (bmnsv-miR-3) apparently regulates viral late genes in the early stage of infection to enable the virus escape the early immune response of the host. This laboratory is also co-ordinating multi-locational field trials of transgenic virus-resistant silkworms, for the conduct of which approval has recently been accorded by the Review Committee on Genetic Manipulation (RCGM) of the Government.

This year too as in previous years, several of the CDFD faculty and scholars have been recipients of prestigious awards and honours. These include, amongst others, the ICMR Fellowship for Young Biomedical Scientists, National Bioscience Award for Career Development, Senior Innovative Young Biotechnologist Award, Fellowships of the Indian National Science Academy (INSA) and the Indian Academy of Sciences, Indo-US Research Fellowship, Guha Research Conference Membership, Young Scientist Award of the AP Akademi of Sciences, INSA Young Scientist Medal, DST-Raman Charpak Fellowship, and KV Rao Research Award. During this period, eight research scholars were conferred with PhD degrees. Many postdoctoral fellows, project associates and summer trainees work for CDFD and play significant roles in the Centre's activities.

Approval of the Governments' Expenditure Finance Committee for the Centre's permanent campus was

accorded this year, and the construction activities are progressing in full swing. We expect that the Animal facility would be functional in the coming year.

I take this opportunity to acknowledge the unstinted co-operation which the Centre has received all along for its activities from the Governing Council, Research Area Panels-Scientific Advisory Committee, Academic / Finance / Building Committees and, of course, the Department of Biotechnology. I wish to thank all the members and officials for their time and effort in supporting our activities and achievements.

I also express my gratitude to the CDFD family who have played a crucial role in the programs and development of the Centre.

J Gowrishankar

March 31, 2014

सेवाएँ
Services

LABORATORY OF DNA FINGERPRINTING SERVICES

Faculty	Madhusudan Reddy Nandineni	Staff Scientist
Other Members	SPR Prasad	Senior Technical Officer
	Ch V Goud	Technical Officer
	Devinder Kumar	Technical Officer
	Sanjukta Mukerjee	Technical Officer
	S Naveenchandra	Technical Officer (Since Feb. 2014)
	Neelima Thota	Technical Officer (Since Dec. 2013)
	Pooja Tripathi	Technical Officer (Since Mar. 2014)
	Girnar Vijay Amrutarao	Technical Assistant
	Shruti Dasgupta	Technical Assistant
	**Devinder Singh Negi	Technical Officer
	**Chandra Shekhar Singh	Technical Assistant
Coordinator	D P Kasbekar	Haldane Chair

(**Presently posted at DNA Profiling Laboratory of CDFD (DPL-CDFD) at the Institute of Life Sciences, Bhubaneswar, Odisha)

Objectives

- 1) To provide DNA fingerprinting services in cases forwarded by law-enforcing agencies / judiciary of State and Federal Governments, relating to murder, rape, paternity, maternity, child swapping, body identification and 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; and
- 5) To create DNA marker databases of different populations of India.

Summary of services provided until the beginning of this reporting year (upto March 31, 2013)

A total number of 186 cases were received for DNA fingerprinting examination during the reporting period (2012 – 2013). Of these, 70 cases related to paternity / maternity, 79 cases related to identification of deceased, 19 cases were pertaining to sexual assault (rape), 13 cases were related to murder and 5 cases pertaining to biological relationship (Kidney transplantation). Seventeen states and Union Territories of India have availed DNA fingerprinting services of CDFD during

this period. Andhra Pradesh forwarded the highest number of cases (104) followed by Odisha (33), Chhattisgarh (13), Punjab (10), Karnataka (5), Kerala (4), Bihar (2), Goa (2), Jammu & Kashmir (2) Tamil Nadu (2), Uttar Pradesh (2), Chandigarh (1), Delhi (1), Madhya Pradesh (1), Puducherry (1), Sikkim (1) and West Bengal (1)

Details of services provided in the current reporting year (April 1, 2013 - March 31, 2014)

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

Biological Relationship	005
Identity of Deceased	255
Murder	011
Paternity/Maternity	051
Sexual Assault (Rape)	036
Total number of cases	<u>358</u>

A total number of 358 cases were received for DNA fingerprinting examination during the current reporting period (2013 – 2014). Of these 255 cases were related to identification of deceased, 51 cases were related to paternity / maternity, 36 cases were pertaining to sexual assault (rape), 11 cases were related to murder and 5 cases were pertaining to biological relationship (kidney transplantation). Fifteen states, Union Territories of India and one foreign country (from East Timor) have availed DNA fingerprinting services of CDFD during this period. Andhra Pradesh forwarded the highest number of cases (233) followed by Madhya Pradesh (53), Chhattisgarh (18), Punjab (14), Delhi (7), Goa (6),

Uttar Pradesh (6), Karnataka (5), Maharashtra (5), Bihar (2), Kerala (2), Puducherry (2), Uttarakhand (2), Jammu & Kashmir (1), and Timor (1) (Fig.1). The cases involving identification of the deceased (71%) constituted the bulk of the cases received (Fig.2).

- Several cases from National Investigation Agency (NIA) involving national security and public safety.

Deposition of evidence in Courts of Law

During this reporting year, the DNA experts defended their reports in 17 cases in various Honorable Courts throughout the country.

Summary of the state-wise break-up of DNA fingerprinting cases:

State/Union Territory	Biological Relationship	Identity of Deceased	Maternity/ Paternity	Murder	Sexual Assault (Rape)	No. of Cases
Andaman & Nicobar	-	-	01	-	-	001
Andhra Pradesh	03	209	14	02	05	233
Bihar	-	-	02	-	-	002
Chhattisgarh		011	06		01	018
Delhi	-	004	02	01	-	007
Goa	-	002	04	-	-	006
Jammu & Kashmir	-		01	-	-	001
Karnataka	02	001	02	-	-	005
Kerala	-	001	01	-	-	002
Madhya Pradesh	-	017	08	08	20	053
Maharashtra	-	-	04	-	01	005
Puducherry	-	-	02	-	-	002
Punjab	-	005	03	-	06	014
Uttar Pradesh	-	003	-	-	03	006
Uttarakhand	-	002	-	-	-	002
International (East Timor)	-	-	01	-	-	001
Total number of cases	05	255	51	11	36	358

During this reporting period, an amount of **Rs. 25,98,889/-** (Rupees twenty five lakhs ninety eight thousand eight hundred and eighty nine only) has been received towards DNA fingerprinting analysis charges, which is inclusive of service charge as levied by Govt. of India.

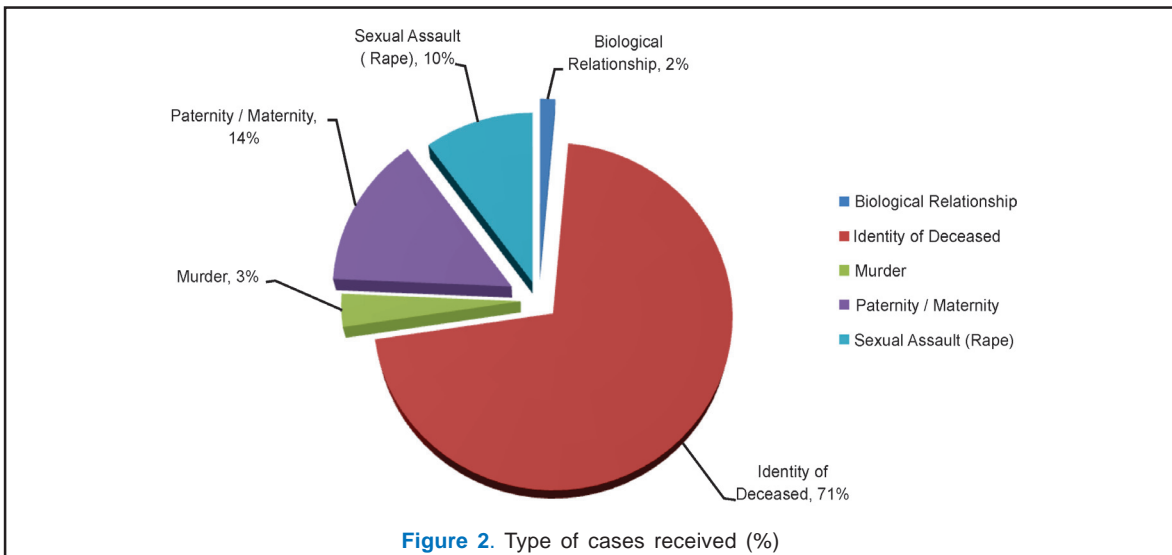
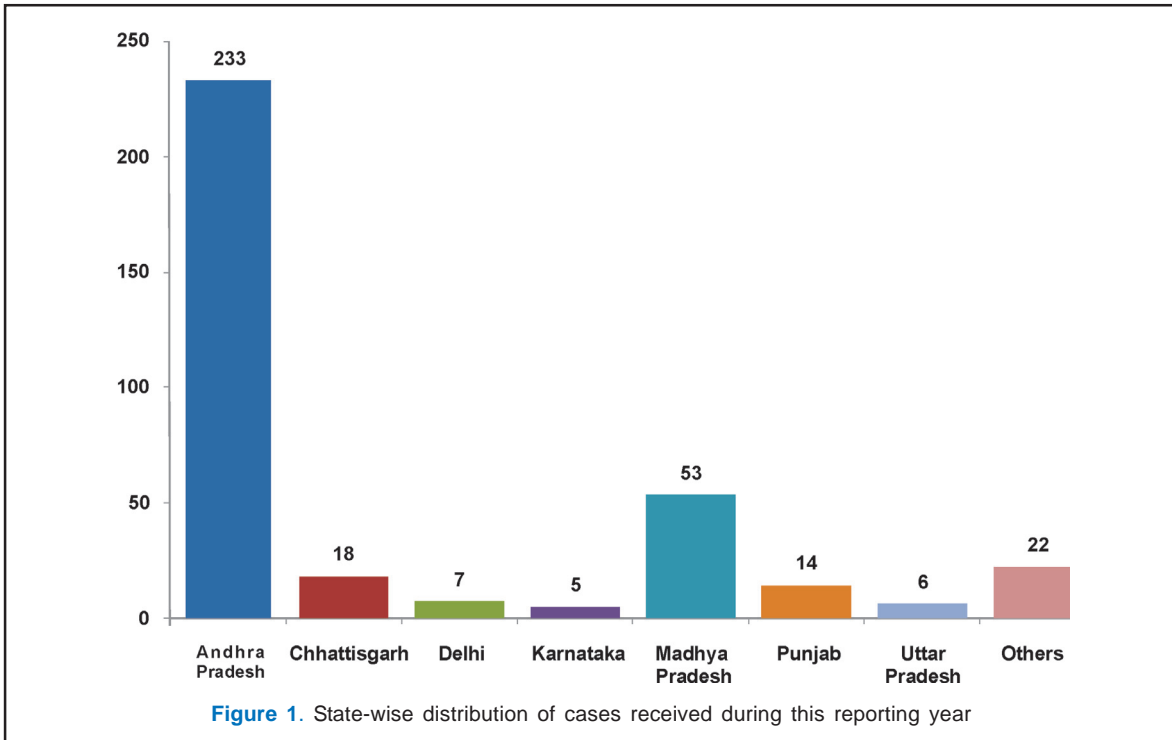
Some prominent cases reported by CDFD during April 1, 2013 to March 31, 2014

- Identification of victims of the Uttarakhand flash floods of June 2013.
- Identification of victims of the Indian Air Force Helicopter crash during Uttarakhand flood relief work of June 2013.
- Identification of victims of bus fire accident case in Mahabubnagar District, A.P. in October, 2013.

Training/Lectures/Workshops on DNA fingerprinting examination

Training

- Training on DNA fingerprinting techniques to personnel from Armed Forces Medical College, Dept. of Forensic Medicine, (AFMC), Pune from 08.07.2013 to 11.07.2013, 04.08.2013 to 15.09.2013 and 17.02.2014 to 30.03.2014.
- Training on DNA fingerprinting techniques to personnel from Andhra Pradesh Forensic Science Laboratory, Hyderabad from 05.08.2013 to 04.09.2013.
- Training on DNA fingerprinting techniques to personnel from Forensic Science Laboratory, Delhi from 07.10.2013 to 14.10.2013.



4. Training on DNA fingerprinting techniques to scientific personnel from Central Police Forensic Science Laboratory, Nepal from 16.12.2013 to 28.02.2014 and 03.03.2014 to 01.04.2014.
5. Training on DNA fingerprinting techniques to personnel from Forensic Science Laboratory, Sagar, Madhya Pradesh from 01.03.2014 to 15.03.2014.
6. Dr Devinder Kumar was deputed to Kedarnath to assist Uttarkhand officials for collection of skeletal remains of the victims of Uttarkhand floods for DNA testing.
7. Ms Sanjukta Mukerjee was deputed to Bodh Gaya, Bihar to assist the National Investigation Agency (NIA) personnel in collection of samples from the crime scene for DNA testing.

Lectures/Workshops

1. Lecture delivered for the benefit of the Post Graduate Students from Jain University, Bangalore, Karnataka on 03.03.2014.
2. Lecture delivered for the benefit of the Post Graduate Students from Karnataka University, Dharwad, Karnataka on 04.03.2014.
3. Lecture delivered for the benefit of new recruited Asst. Director & Scientific Assistants of Andhra Pradesh Forensic Science Laboratory, Hyderabad on 23.04.2013.
4. Lecture delivered for the benefit of the B.Tech Students from Integral University, Lucknow, Uttar Pradesh on 04.06.2013.
5. Lecture delivered for the benefit of Post Graduate Students from Amrita Institute of Medical Sciences, Kochi, Kerala on 20.07.2013.
6. Lecture delivered for the benefit of Dy. Sr. Superintendent of Polices from North Eastern Police Academy (NEPA), Umsaw, Meghalaya on 16.09.2013.
7. Lecture delivered for the benefit of Students and Faculty members from Vivekanand College, Kolhapur, Maharashtra State on 07.01.2014.
8. Lecture delivered for the benefit of the Post Graduate Students from Jain University, Bangalore, Karnataka on 10.02.2014.
9. Lecture delivered for the benefit of Air Force Officers from Air Force Intelligence School, Pune on 11.02.2014.
10. Lecture delivered for the benefit of the 55 Police Officers from different countries, coordinated by National Crime Records Bureau (NCRB), New Delhi on 12.02.2014.
11. Lecture delivered for the benefit of the Post Graduate Students from Dept. of Microbiology, SGB, Amravati University, Maharashtra on 24.02.2014.
12. Lecture delivered for the benefit of the Post Graduate Students from School of Sciences, Kathmandu University, Nepal on 28.02.2014.
13. Lecture delivered for the benefit of the Post Graduate Students of Osmania University, Hyderabad on 10.03.2014.

DIAGNOSTICS DIVISION

Faculty	Ashwin B Dalal	Staff Scientist
Adjunct Faculty	Prajnya Ranganath Shagun Aggarwal	Assistant Professor, NIMS Assistant Professor, NIMS
PhD Students	Anusha Uttarilli Ashish Bahal Anjana Kar	Senior Research Fellow Senior Research Fellow Senior Research Fellow
Other Members	Aneek Das Bhowmik T Nageswara Rao Divya Matta Savita Wangnekar V Subhash P Rajitha GR Savithri Angalena R A Sobhan Babu Usha Rani Dutta M Muthulakshmi S Jamal Md Nurul Jain Bhagwati Sharan Sharma S Vasantha Rani C Krishna Prasad R Sudheer Kumar	Research Associate DBT-Research Associate (Till Jan. 2014) Project-Junior Research Fellow Research Assistant (Till Oct. 2013) Project Assistant (Till May 2013) Technical Officer Senior Technical Officer Senior Technical Officer Technical Officer (Since Nov. 2013) Technical Officer Technical Officer (Since Mar. 2014) Technical Officer Technical Assistant (Till Aug. 2013) Technical Officer (Since Jan. 2014) Technician Technician

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; and
4. To impart training in genetic evaluation of patients with genetic disorders.

Details of services provided in the current reporting year (April 1, 2013 - March 31, 2014)

Clinical Genetics

A total of 3496 patient samples were analysed for genetic testing, during the year 2013-14. These consisted of patients with chromosomal disorders, monogenic disorders, mental retardation, congenital malformations, inborn errors of

metabolism, and other familial disorders. A fellowship program for training in Clinical Cytogenetics and Clinical Molecular Genetics has been initiated.

The Department of Medical Genetics established at Nizam's Institute of Medical Sciences, Hyderabad is running successfully. A total of 2304 patients were examined and counseled in the unit during 2013-14.

Diagnosics Research

Project 1: Cloning, characterization and analysis of chromosomal rearrangements in human genetic disorders.

Summary of work done until the beginning of this reporting year (upto March 31, 2013)

Structural chromosomal rearrangements alter the genome architecture and result in various human disease phenotypes. Cloning the breakpoint region provides opportunity of identifying the disease gene in patients with such rearrangements.

Genetic investigations done during 2013-14

Investigation	Total cases	Positives
Cytogenetics	1251	99 (8%)
Proband	1141	96 (8.4%)
Prenatal	0110	3 (2.7%)
Molecular Genetics	1307	447 (34%)
Proband	1224	427 (34.9%)
Prenatal	0083	20 (24.1%)
Biochemical Genetics	0938	256 (27.2%)
Proband	0924	253 (27.3%)
Prenatal	0014	3 (21.4%)

Cytogenetics

Disease	Abnormality	No. of cases
Down Syndrome	Trisomy 21	42
	46,XY,rob(21;21) +21	1
	46,XX,rob(21;21) +21	1
	46,XX,rob(14;21)+21	1
	46,SC,rob(14;21)+21	1
	47,XX+21[32]/46,XX[18]	1
Edward syndrome	47,XX,+18	1
Turner syndrome	Monosomy X (45,X)	11
	Mosaic 45,X/ 46,XX	3
	46,Xr(X)/45,X	1
Klinefelter Syndrome	47,XXY	8
Triple X Syndrome	mos 47,XXX/46,XX	1
Sex reversal	Phenotypic female with 46,XY	2
	Phenotypic male with 46,XX	1
	47,XY+marker	1

Fluorescence *in situ* Hybridization (FISH)

Disease/translocation	Probe	No of tests
Prader-Willi Syndrome	SNRPN(15q11)/PML(15q24)	2
1p36 syndrome	1p36 probe	20
Di-George Syndrome	TUPLE(22q11.2)/ARSA(22q13)	8
Williams-Beuren	ELN(7q11)/Control(7q22)	5
Marker chromosome	WCP-Y, WCP-15SE(14)/(22), SE(X)/(Y), Acro-p-arm	18
Spectral karyotyping		3

Quantitative Fluorescent PCR (QF-PCR)

QF-PCR kit	Patients	Positives
Prenatal QF-PCR	28	1

Structural chromosomal abnormalities

Inversions	
46,XY,inv(9)	2
46,XX,inv(11)	4
46,X,inv(Y)	2
46,X,inv(Y)/46,XY	1
Deletions	
46,X,del(Y)	2
46,XX,del(10)(p)	1
46,X,del(X)(p11.2>pter)	1
Translocations	
45,XY,rob(13;14)	3
46,XY,t(6;8)(p24;q14)	1
46,XY,t(8;22)(q21.2;q13.3)	1
46,XX,t(14;15)(q12;q26)	1
46,XY,der15t(14;15)mat	1
46,XX,t(11;22)(q24;q13)	1
46,XY,t(2;6)(q31;q27)	1
46,XX,t(6;13)(p24;q14)	1
Polymorphic variants	
46,XX,9qh+, 46,XY,9qh+ 46,XX,9qh-, 46,XX,1qh+ 46,XY,1qh+, 46,XX,1qh+ 46,XX,22p+, 46,XY,16qh+ 46,XY,21p+, 46,XX,15p+ 46,XY,14p+,46,X,Yqh-	34

Biochemical Genetics

Disease/Test	Positives
Urine & Blood Metabolic Screening tests (N=266)	61
Amino acid disorders (N=195)	70
Maple syrup urine disease	1
Non Ketotic Hyperglycinemia	12

Hyperornithinemia	5
Tyrosinemia	2
Phenylketonuria	2
Other amino acid disorders	48
Lysosomal storage disorders (N=463)	122
Hurler syndrome(25)	13
Hunter syndrome(18)	6
Sanfilippo B (14)	3
Morquio A disease (42)	25
Arylsulphatase B (8)	4
Sly disease (25)	0
GM1-Gangliosidosis (70)	12
Fucosidosis (2)	0
Gaucher disease (54)	10
Krabbe disease (26)	0
Pompe disease (3)	1
Nieman Pick disease (33)	12
Mucopolidosis(15)	15
Metachromatic Leukodystrophy (68)	8
Fabry disease(11)	5
Mannosidase (6)	2
Hexosaminidase A/B (42)	
Tay Sachs disease	1
Sandhoff disease	4
Multiple sulphatase deficiency(1)	1
Prenatal diagnosis (14)	3
Niemann Pick disease (3)	2
Sly disease (1)	1

Molecular Genetics

Disorders	Cases	Positive	Negative		
DMD/BMD (till Jan'14)	240	177	63		
DMD Carrier Analysis	29	16	13		
Spinal Muscular Atrophy	100	55	45		
SMA Carrier Analysis	48	17	31		
		Normal	Homozygous	Heterozygous	Compound Heterozygous
β-thalassemia/Sickle cell	139	10	56	54	19
Factor V Leiden	186	178	-	08	-
Factor II mutation	121	121	-	-	-
Cystic Fibrosis	99	92	03	04	-
Triplet Repeat Disorders		Positive	Negative		
Friedreichs Ataxia	57	18	39		
Myotonic Dystrophy	13	10	03		
Huntington Disease	43	24	19		
SCA Panel (1,2,3,6 & 7)	86	21	65		
DRPLA	05	-	05		
Fragile X Syndrome	58	11	47		
Prenatal Diagnosis					
DMD	04	03	01		
Spinal Muscular Atrophy	15	03	04	08	
Huntington Disease	01	01	-		
Cystic Fibrosis	02	-	02		
		Normal	Homozygous	Heterozygous	Cpd. Heterozygous
β-thalassemia	61	13	10	35	03

Cpd Heterozygous= Compound Heterozygous

Details of progress made in the current reporting year (April 1, 2013 - March 31, 2014)

We performed molecular characterization of chromosomal breakpoint 46, XX, t (2; 22) (q33; q11.2) (Fig.1A) in a patient with delayed motor milestones and myopathy. Parents karyotype was normal. Lymphoblastoid cell lines of the patient were established and WCP FISH confirmed the balanced translocation (Fig.1B). Array CGH studies confirmed that this translocation is not associated with any gains or losses at the breakpoints and elsewhere in the genome (Fig.1C). We followed a positional cloning approach for

mapping the chromosomal breakpoints 2; 22.

Delineation of the breakpoint region using BACs

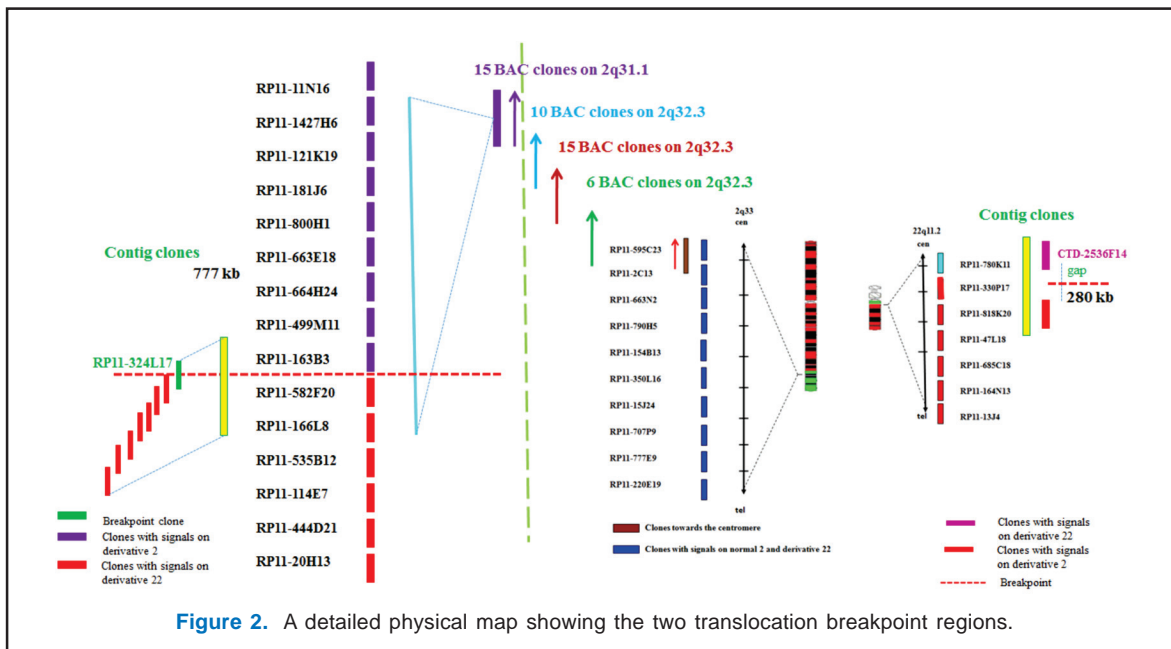
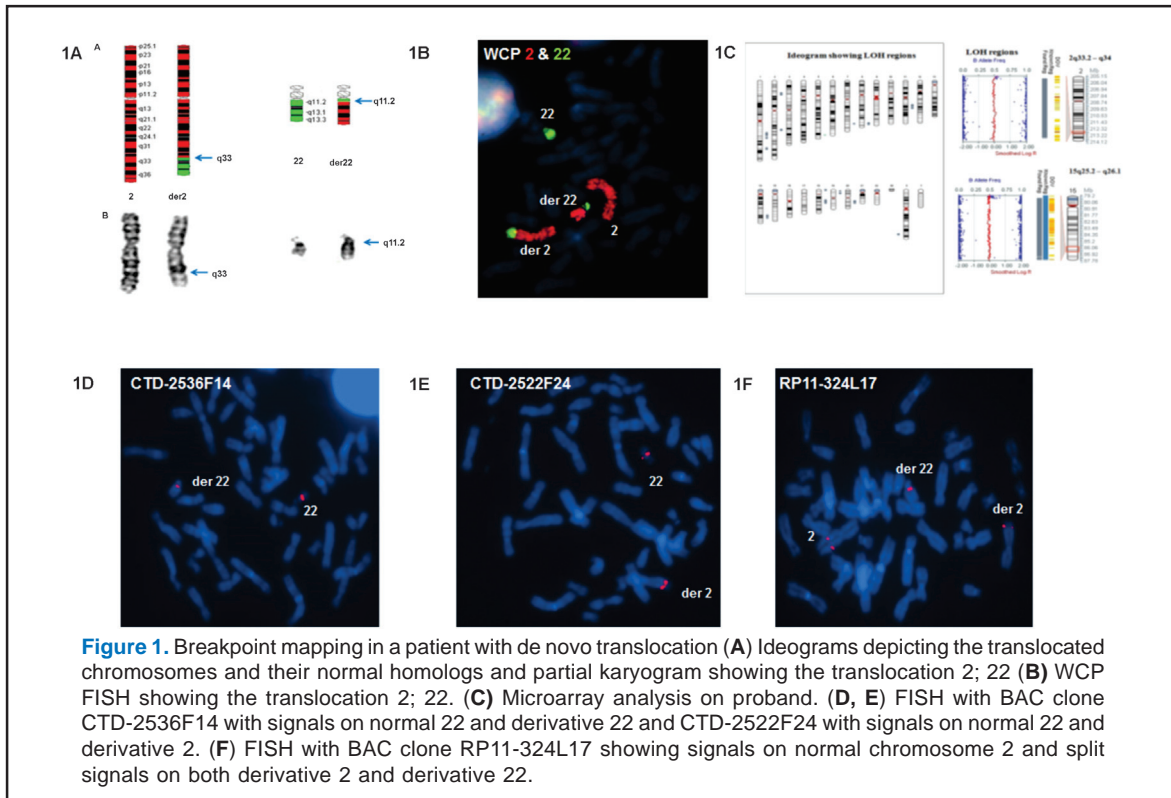
Sixty four BAC clones from the 2q region and 20 from the 22q region were selected. FISH was performed with all the clones (Fig.2). On 22q region, FISH with BAC clone CTD-2536F14 showed signals on normal 22 and derivative 22 (Fig.1D) whereas CTD-2522F24 showed signals on normal 22 and derivative 2 (Fig.1E). The breakpoint region was narrowed down to 280 kb. Due to the gap in the genome further identification was stopped and focused on the other breakpoint region on 2q. Due

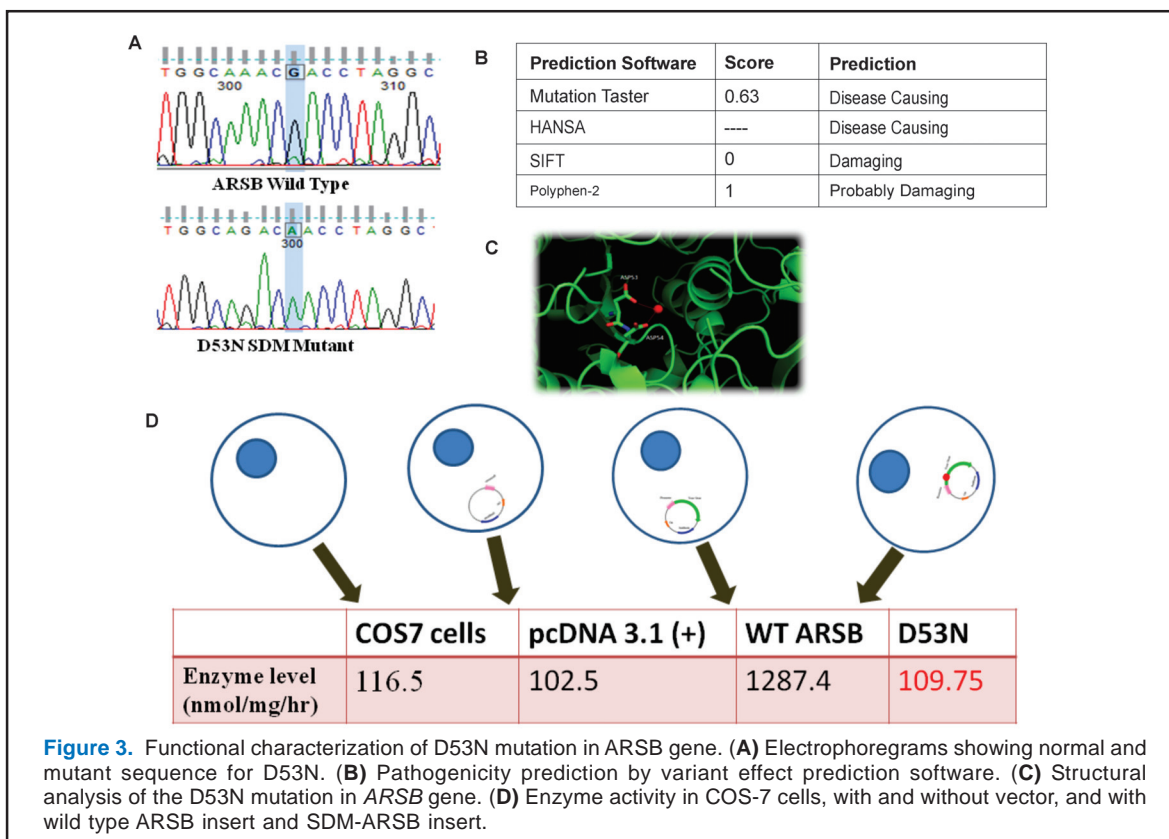
to the big subchromosomal and euchromatic region, detailed chromosomal walking was performed from 2q33.1 to 2q31.1 region. Finally the breakpoint spanning BAC clone RP11-324L17 showing signals on normal 2, and split signals on derivative 2 and derivative 22 was identified (Fig.1F). Further identification of the candidate or new genes is underway.

Project 2: Clinical, biochemical and molecular analysis of common lysosomal storage disorders.

Summary of work done until the beginning of this reporting year (upto March 31, 2013)

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 common lysosomal storage disorders.

Details of progress made in the current reporting year (April 1, 2013 - March 31, 2014)

Over last four years we have been able to identify mutations in 165 patients with different lysosomal storage diseases (LSDs) (Table 1). This study has revealed the mutation spectrum in patients with LSDs in the Indian population.

Further the novel mutations identified in the MPS VI patients were functionally characterized by use

Lysosomal storage disorder	Total Patients	Number of mutations	Number of novel mutations
MPS I / Hurler syndrome	30	16	9
MPS II / Hunter syndrome	38	16	5
MPS VI / Maroteaux-Lamy syndrome	36	21	16
Niemann-Pick disease	38	30	22
Metachromatic leucodystrophy	20	20	10
Sialidosis	3	3	3
Total cases	165	106	65

Table 1. Data sheet showing all the mutations detected in different patients.

of molecular techniques of RNA isolation, Reverse transcriptase PCR & cDNA synthesis, cloning and site directed mutagenesis (SDM). Total RNA was isolated from human cultured fibroblasts and reverse transcription PCR (RT-PCR) amplification was carried out followed by amplification of ARSB cDNA. The amplified full length 1.7kb ARSB cDNA was cloned into pcDNA3.1(+) vector to produce pcDNA3.1 - ARSB. COS-7 cells were maintained in DMEM supplemented with 10% FBS and 1% penicillin/streptomycin (Gibco) at 37°C and 5% CO₂. For transfection, 4x10⁶ cells were grown up to 70 to 90% confluence on 100 mm plates. Cells were transfected with 2 µg of plasmid DNA and 4 µl of Lipofectamine 2000 (Invitrogen, Heidelberg, Germany). Cells were harvested after 48 hr after transfection. SDM-ARSB cDNA mutant constructs were functionally characterized by transient transfection into cultured COS-7 cells. Cell lysate was prepared using chemical cell lysis method and ARSB enzyme assays were performed. Enzyme assays have been done for six different SDM cDNA mutant constructs namely D53N, D54N, S320R, A237D, P313A & c.1208delC. COS-7 cells with all the mutated cDNA constructs showed a significant (less than 10% activity) decrease in the enzyme activity as compared to wild type revealing the pathogenic nature of the detected mutations.

Publications

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2. Bashyam MD, Chaudhary AK, Kiran M, Reddy V, Nagarajaram HA, Dalal A, Bashyam L, Suri D, Gupta A, Gupta N, Kabra M, Puri R, Rama Devi R, Kapoor S and Danda S (2013). Molecular analyses of novel *ASAH1* mutations causing Farber lipogranulomatosis: analyses of exonic splicing enhancer inactivating mutation. **Clinical Genetics** Nov 8. Doi.10.1111/cge.12316.
3. Dutta UR, Pidugu VK, Goud ChV, Hoefers C, Hagemann M and Dalal A (2013). Identification and molecular cytogenetic characterization of a novel complex Y chromosome rearrangement in a boy with disorder of sexual development. **Gene** 519: 374-380.
4. Dutta UR, Rajitha P, Pidugu VK and Dalal AB (2013). Chromosomal abnormalities in amenorrhea: A retrospective study and review of 637 Patients in South India. **Archives of Iranian Medicine** 16: 267-270.
5. Dutta UR, Rajitha P, Pidugu VK and Dalal AB (2013). Partial proximal trisomy 14: Identification and molecular characterization in a girl with global developmental delay. **Genetic Counseling** 24: 207-216.
6. Kantaputra PN, Kayserili H, Güven Y, Kantaputra W, Balci MC, Tanpaiboon P, Uttarilli A and Dalal A (2013). Oral manifestations of 17 patients affected with mucopolysaccharidosis type VI. **Journal of Inherited Metabolic Diseases** 37: 263-268.
7. Kesari A, Dalal A, Lal G and Pandey SN (2013). Molecular diagnostics. **BioMed Research International** 2013: 387486.
8. Muthugaduru DJ, Sahu C, Ali MJ, Dalal A and Jalali S (2013). Report on ocular biometry of microphthalmos, retinal dystrophy, flash electroretinography, ocular coherence tomography, genetic analysis and the surgical challenge of entropion correction in a rare case of Hallermann-Streiff-Francois syndrome. **Documenta Ophthalmologica** 127: 147-153.
9. Muthuswamy S, Agarwal S and Dalal A (2013). Diagnosis and genetic counseling for Friedreich's Ataxia: A time for consideration of TP-PCR in an Indian Setup. **Hippokratia** 17: 38-41.
10. Ranganath P and Dalal AB (2013). Congenital metacarpal pseudoarthrosis, cleft palate, short stature, advanced bone age, and genu valgum: a new syndrome or a variant of Devriendt syndrome? **Clinical Dysmorphology** 22: 73-75.
11. Bashyam MD, Chaudhary AK, Kiran M, Nagarajaram HA, Devi RR, Ranganath P, Dalal A, Bashyam L, Gupta N, Kabra M, Muranjan M, Puri RD, Verma IC, Nampoothiri S and Kadandale JS (2014). Splice, insertion-deletion and nonsense mutations that perturb the phenylalanine hydroxylase transcript cause phenylketonuria in India. **Journal of Cellular Biochemistry** 115: 566-574.
12. Dutta UR, Vempally S, Ranganath P and Dalal A (2014). A novel combined 15q11.2 duplication and a bisatellited supernumerary marker derived from chromosome 22: molecular characterization of the marker. **Gene** 539: 162-167.

13. Love JM, Prosser D, Love DR, Chintakindi KP, Dalal AB and Aggarwal S (2014). A novel glycine decarboxylase gene mutation in an Indian Family with nonketotic hyperglycinemia. ***Journal of Child Neurology*** 29: 122-127.
 14. Aggarwal S, Coutinho MF, Dalal AB, Mohamed Nurul Jain SJ, Prata MJ and Alves S. Prenatal skeletal dysplasia phenotype in severe MLII alpha/beta with novel GNPTAB mutation. ***Gene*** (In press).
 15. Anusha U, Ranganath P, Md Nurul Jain SJ, Krishna Prasad C, Sinha A, Verma IC, Phadke SR, Puri RD, Danda S, Muranjan MN, Jevalikar G, Nagarajaram HA and Dalal AB. Novel mutations of the ARSB gene in Indian patients with Mucopolysaccharidosis Type VI. ***Indian Journal of Medical Research*** (In press).
 16. Dutta UR, R Ponnala and A Dalal. A novel de novo balanced reciprocal translocation t(18;22) associated with recurrent miscarriages: A case report. ***Journal of Reproduction and Infertility*** (In press).
 17. Kantaputra PN, Kayserili H, Guven Y, Kantaputra W, Balci MC, Tanpaiboon P, Tananuvat N, Uttarilli A and Dalal A. Clinical manifestations of 17 patients affected with mucopolysaccharidosis type VI and eight novel ARSB mutations. ***American Journal of Medical Genetics*** (In press).
 18. Rajashree N, Phadke SR, Dalal AB and Ranganath P. Novel mutations in the PRG4 gene in two Indian families with the Camptodactyly- arthropathy- coxa vara-pericarditis syndrome. ***Indian Journal of Medical Research*** (In press).
 19. Stephen J, Shukla A, Dalal A, Girisha KM, Shah H, Gupta N, Kabra M, Dabadghao P and Phadke SR. Mutation spectrum of COL1A1 and COL1A2 genes in Indian patients with osteogenesis imperfecta. ***American Journal of Medical Genetics*** (In press).
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- Other publications**
1. Rajashree N, Kumar SR, Ranganath P and Dalal A (2013). Banking of genetic material: A key to the future. ***Genetic Clinics*** 6: 12-15.
 2. Dalal A (2014). Phenylketonuria: Past, present and future. ***Genetic Clinics*** 7: 19-24.
 3. Ranganath P and Dalal A (2014). Quality issues in medical genetics. ***Genetics in Clinical Practice*** 1st Edition: 237-243.

APEDA-CDFD CENTRE FOR BASMATI DNA ANALYSIS

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Objectives

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; and
2. Fine mapping and characterization of the candidate genes of grain appearance traits of Basmati rice and studies on QTLs from a promising region of chromosome 5.

Summary of the work done until the beginning of this reporting year (upto March 31, 2013)

Previously, 47 QTLs governing 18 economically important traits of Basmati rice have been identified in a mapping population of 189 F₂ individuals of a cross between Basmati370 and Jaya by screening with 134 polymorphic microsatellite markers. The F₂ material has been advanced to F₇ generation comprising 155 Recombinant Inbred Lines (RILs)

where phenotyping of 18 traits were recorded. DNA was isolated from the 155 RILs and screened with polymorphic Simple Sequence Repeats (SSR) markers which have been used for screening F₂. About 74 SSR markers have been screened during this period.

Details of progress made in the current reporting year (April 1, 2013 - March 31, 2014)

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 period under report, a total of 219 (EIC samples) Basmati samples were analyzed and the number of samples indicating the percentage of adulteration with non-basmati rice is shown in Figure 1.

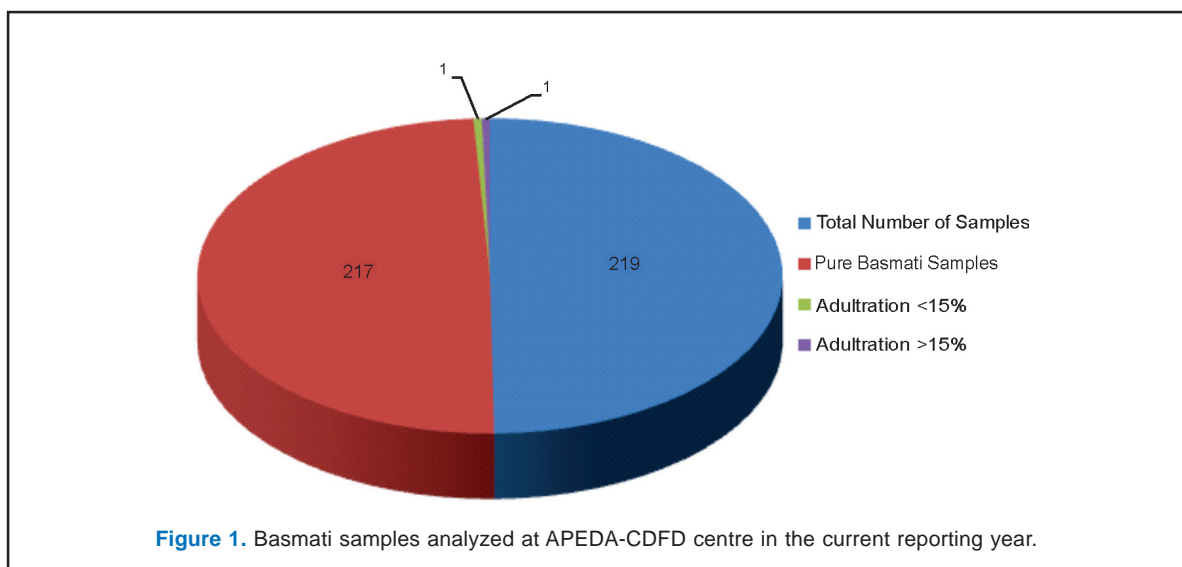


Figure 1. Basmati samples analyzed at APEDA-CDFD centre in the current reporting year.

Objective 2: Fine mapping and characterization of the candidate genes of grain appearance traits of Basmati rice and studies on QTLs from a promising region of chromosome 5.

Screening of mapping population of 155 RILs with the remaining 60 polymorphic SSR markers was completed for confirmation and fine mapping of the grain size QTL identified in the F₂ population. The preliminary analysis of RILs indicated narrowing down genetic distance of the flanking markers harboring QTL cluster from 26.5cM to 16.5cM with physical distance of 17.66-18.60 Mb. To identify the candidate gene(s) underlying the major QTL within this marker interval, association mapping approach was employed using diverse rice germplasms. In all, three markers namely, RM18582, RM430 and RM18616 were found to be strongly associated with grain size. These markers have the potential to be used in marker-assisted improvement of the grain size in Basmati rice.

One of the ultimate goals of genetic mapping of complex traits is to isolate candidate genes at QTL regions. In the present study, one QTL region at marker interval of RM430 and RM18600 on chromosome 5 spanning a physical distance of

327.1kb was chosen for identification of candidate genes. Based on the rice genome sequence information, about 50 genes were identified in this region. Out of 50 genes, two genes which were reported to be involved in controlling the seed size and seed weight in *Arabidopsis* as well as rice were found. Hence, the two genes, *AP2* transcription factor (Os05g32270) and *RING E3 ligase* (Os05g32570) were chosen for further studies. Out of the two predicted candidate genes, *AP2* domain transcription factor was amplified using both gDNA and cDNA of Basmati370 and aligned to *indica* and *japonica* reference sequences. The *AP2* transcription factor of *japonica* shows two splice variants. The single nucleotide polymorphisms (SNPs) identified within the intronic regions were represented in Figure 2. The identified SNPs are being validated. Future work in this project would involve i) further narrowing down of targeted QTL region through association mapping, ii) prediction of candidate genes controlling grain size and their structural and functional analysis and iii) use of SoLiD sequencing data to align genomic sequence of QTL region with rice reference genome sequences to check for the variations at genomic level.

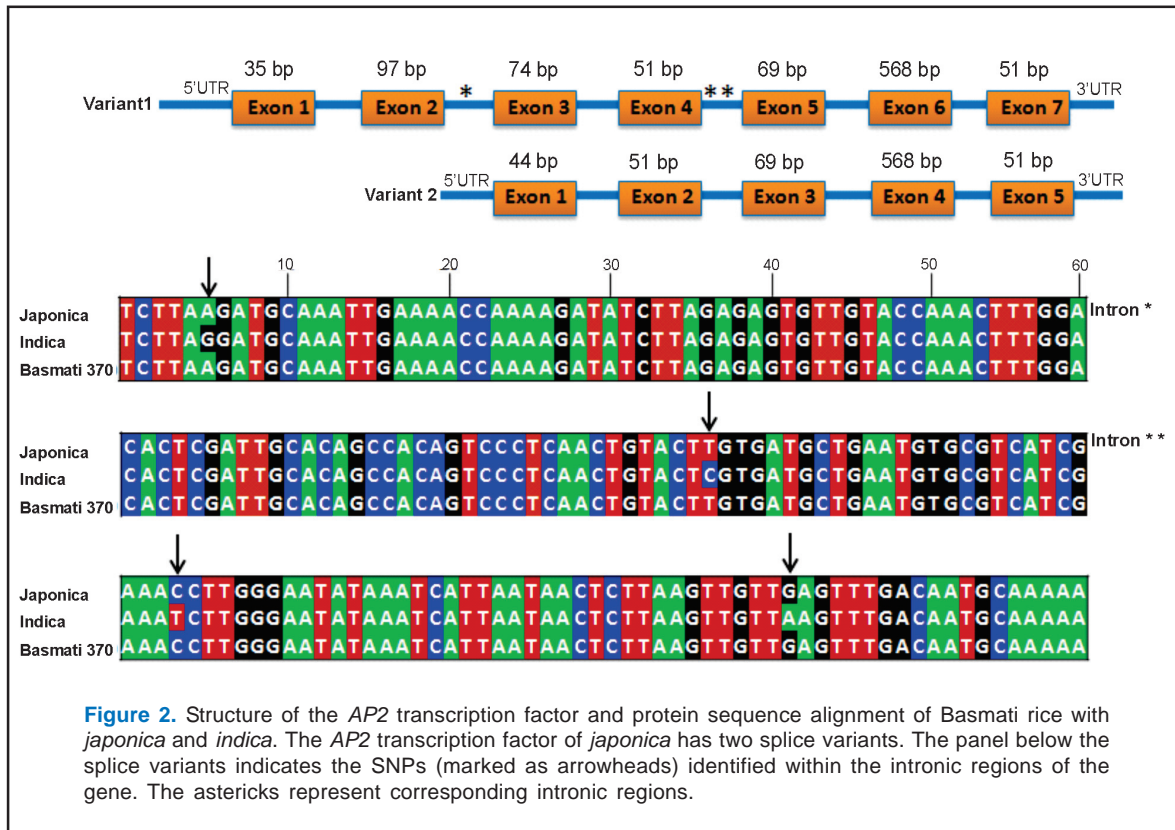


Figure 2. Structure of the *AP2* transcription factor and protein sequence alignment of Basmati rice with *japonica* and *indica*. The *AP2* transcription factor of *japonica* has two splice variants. The panel below the splice variants indicates the SNPs (marked as arrowheads) identified within the intronic regions of the gene. The asterisks represent corresponding intronic regions.

Publications

1. Malathi S, Lakshminarayan RV, Hameedunnisa B, Purushotham Reddy B, Neetasri C, Nagaraju J, Anwar SY and Siddiq EA (2013). Population structure and genetic analysis of different utility types of mango (*Mangifera indica* L.) germplasm of Andhra Pradesh state of India using microsatellite markers. *Plant Systematics and Evolution* 299: 1215-1229.
2. Archak S and Nagaraju J (2014). Computational analyses of protein coded by rice (*Oryza sativa japonica*) cDNA (GI: 32984786) indicate lectin like Ca(2+) binding properties for Eicosapenta Peptide Repeats (EPRs). *Bioinformation* 10: 63-67.

शोध
Research

LABORATORY OF BACTERIAL GENETICS

Studies on Gene Regulation, Transcription Termination, and Amino Acid and Ion-Transport in *Escherichia coli*

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	Noor Md Shaik	CDFD-IKP Fellow (Till Jan. 2014)

The Laboratory of Bacterial Genetics comprises three faculty groups engaged in research 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 in Microbial Biology. The work undertaken in this reporting year is described below under the following objectives:

Objectives

1. To understand the role of Rho-dependent transcription termination in prevention of excessive genome-wide RNA-DNA hybrids (R-loops);
2. To characterize a novel pathway for potassium translocation;
3. To determine mechanisms of export of basic amino acids;
4. To understand genetic interactions between (p)ppGpp and tm-RNA/SmpB;

5. To delineate the role of (p)ppGpp in cell division;
6. Suppressor studies of mutants lacking (p)ppGpp hydrolase SpoT; and
7. Role of transketolases in *E. coli* physiology.

Summary of work done until the beginning of this reporting year (upto March 31, 2013)

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

Details of progress made in the current reporting year (April 1, 2013 - March 31, 2014)

1. Role of Rho-dependent transcription termination in avoidance of R-loops.

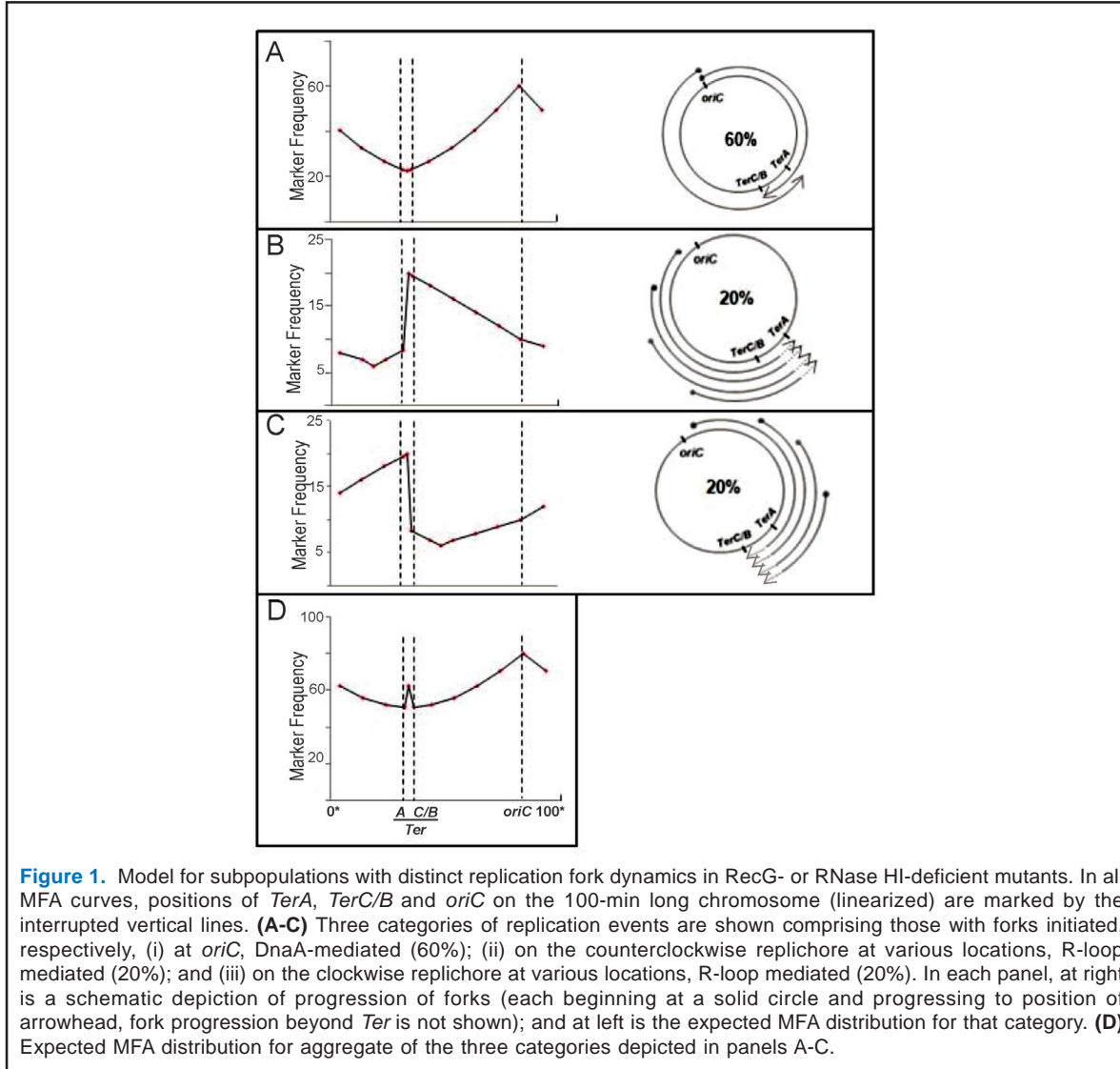
Transcription and translation are coupled processes in all bacteria, and Rho-dependent transcription termination (RDTT) in *E. coli* is a mechanism by which synthesis of transcripts (other than ribosomal RNA) which are not being simultaneously

translated is prematurely terminated. Previous work from this laboratory has established that an essential function of RDTT is to prevent the formation of toxic R-loops, presumably by aborting the synthesis of nascent untranslated transcripts which would otherwise be prone to re-anneal with upstream DNA to form RNA-DNA hybrids. Lethality associated with loss of Rho or NusG proteins (that are required for RDTT) is rescued by ectopic expression of an R-loop helicase UvsW, and a whole-genome R-loop mapping approach has been employed to demonstrate the occurrence in a *nusG* mutant of excessive R-loops across the genome from both sense and antisense transcripts.

In the current year, we have completed the development of a model to explain the phenomenon of R-loop initiated constitutive stable DNA

replication (cSDR). Replication of the circular chromosome in *E. coli* is normally initiated from *oriC* with the aid of the protein DnaA, and then proceeds bidirectionally across the clockwise and counterclockwise replichores to terminate by merger of the opposing forks in an antipodal terminus region which is characterized by discrete *Ter* sequences that act as polar arrest sites for replication fork progression (see Fig. 1A). Deletion of *oriC* or *dnaA* results in inviability, unless cSDR is induced in these cells as an alternative mechanism of chromosomal replication by abolition of either of two R-loop removing enzymes present in *E. coli*, RNase HI and RecG.

The new model for cSDR developed by us is based in part on the earlier demonstration by our group of genome-wide R-loops in RDTT-deficient mutants,



and in part on recent published data of chromosomal marker frequency analysis (MFA) by two other groups in RNase HI- and RecG-deficient mutants, respectively. The latter have both reported that cSDR is associated with a distinct and unusual marker frequency peak in the chromosomal terminus region, but they have offered different explanations for the same, namely abnormal replication initiation from (i) R-loops at discrete *oriK* sites, or (ii) fork collisions in the terminus region.

In our model, we propose that the common MFA curve patterns obtained by the two groups can be explained on the assumption that this pattern is a composite of distributions from subpopulations with three different kinds of replication initiation events, as represented in Figure 1. For the purpose of this simple depiction, 60% of replication initiations are envisaged to have occurred from *oriC* (Fig. 1A), and 20% each from R-loops in the counter-clockwise and clockwise replicore arms, respectively (Figs. 1B and 1C); nevertheless, a single cell could harbour more than one category of replication event.

For cSDR events initiated from site(s) on the counter-clockwise replicore arm (Fig. 1B), it is expected that cSDR origins occur at a uniform, but low, probability across the genome, since the R-loops are assumed to be fairly evenly distributed. Hence, the marker frequency of an arbitrary locus on the counterclockwise arm will be proportional to its distance from *oriC* (that is, the opposite of that with DnaA-mediated initiations from *oriC*; compare Figures 1A and 1B). The mirror symmetrically reverse situation would apply for the subpopulation of cSDR initiation events on the clockwise replicore arm, as shown in Fig. 1C.

The composite MFA distribution derived from the three categories is shown in Figure 1D. Two features of the observed MFA for the mutant lacking RecG or RNase HI are recapitulated in this composite curve, namely, a peak in the mid-terminus region and a shallower MFA distribution, that is, a smaller magnitude of enrichment of *oriC*-proximal to *oriC*-distal markers (compare Figs. 1A and 1D).

The published data are therefore consistent with a model of distributed occurrence of R-loops across the genome as has been proposed by us earlier. Our current efforts are directed towards validation of R-loop occurrence in the genome-wide high-ranking clusters previously identified by our bisulphite mapping approach, and to ask whether cSDR can be demonstrated in RDTT-deficient

mutants. The dynamics of replication fork progression and termination during cSDR, as also the possibility of occurrence of replication fork reversals when opposing replisomes meet, are other subjects that are sought to be investigated by us in future studies.

2. Evidence for a novel cryptic pathway for potassium translocation in *E. coli*.

In *E. coli*, components of the phosphotransferase system (PTS) mediate uptake of carbohydrates wherein transport of the incoming sugar is coupled to its phosphorylation. In each of these systems, a phosphate moiety is transferred from phosphoenolpyruvate (PEP) to the particular sugar via a multi-protein phosphorelay mechanism. *E. coli* also possesses a paralogous PTS comprised of the proteins PtsP-PtsO-PtsN, with PEP-dependent phosphorelay operating in the same sequence. However, the phosphorylation substrate of PtsN is unknown. We have been examining a physiological link between potassium (K^+) metabolism and the PtsP-PtsO-PtsN phosphorelay and have previously reported that consistent with earlier reports a strain lacking PtsN was progressively rendered K^+ sensitive (K^S) as the external K^+ concentration ($[K^+]_e$) was raised above 20 mM in a synthetic glucose minimal medium. The *ptsN* mutant however grew at rates comparable to the parent in a medium of low (1 mM) $[K^+]_e$. An independent study by others has postulated that the K^S phenotype associated with a deficiency of PtsN occurs due to a growth inhibitory increase in intracellular K^+ content, resulting from unfettered activity of TrkA the regulatory subunit of the TrkG/H K^+ uptake proteins. Our studies on the other hand lend support to an alternative model wherein the K^S of the *ptsN* mutant paradoxically results due to a K^+ limitation in media of high (≥ 30 mM) $[K^+]_e$. Our earlier observation that expression of the K^+ carrier proteins TetA and a truncated KdpA polypeptide (KdpA') suppressed the K^S of the *ptsN* mutant appears consistent with the latter suggestion. In the current year we tested the effects of heterologous overexpression of a K^+ uptake protein Kup and found that its overexpression suppressed the K^S phenotype of a *ptsN* mutant, lending support to the K^+ limitation model.

Recently in literature the transport properties of a variant of the ammonia uptake channel AmtB bearing the H168D/H318E double substitutions in AmtB (AmtB^K) have been described. These studies have shown that in contrast to AmtB, AmtB^K at the

expense of mediating uptake of its natural substrate, the NH_4^+ ion (or its analogue methylammonium), mediates the uptake of K^+ with the outcome that in a wild-type strain expression of *amtB^K* (but not *amtB*) leads to a K^{S} phenotype in a medium of high $[\text{K}^+]_{\text{e}}$ and in a triple K^+ transporter deficient strain leads to both a K^{S} phenotype and a K^+ sparing phenotype such that the latter strain displays an enhanced ability to grow in media containing less than 20 mM $[\text{K}^+]_{\text{e}}$. The K^{S} phenotype of *amtB^K* expression is known to correlate with elevated cellular K^+ content. We constructed chromosomal single copy derivatives of *amtB* and *amtB^K* under the expression control of the P_{trc} promoter that were placed at the *attB* site on the chromosome. We found consistent with the recent report that expression of *amtB^K* (but not *amtB*) with 1 mM IPTG conferred (i) a K^{S} phenotype to the parent in a high $[\text{K}^+]_{\text{e}}$ medium (ii) a K^+ sparing phenotype to a triple K^+ transporter deficient strain. On the other hand the simultaneous presence of the two perturbations that are known to cause a K^{S} phenotype in one strain namely, absence of PtsN and expression of *amtB^K* led to the annihilation of the K^{S} phenotype, indicating (i) the two perturbations causing K^{S} must have different causalities and (ii) implying that the K^{S} in the *ptsN* mutant may result due to K^+ limitation. Additionally, we found that the K^{S} of the *ptsN* mutant persisted in an exacerbated fashion in strains that lacked all K^+ uptake systems and exacerbation in the K^{S} was also seen in strains lacking either the Trk or the Kup K^+ transporters.

Previously we found that the K^{S} phenotype of the *ptsN* mutant was suppressed in a strain bearing a knockout mutation in the gene *ycgO* encoding a predicted inner membrane protein of unknown function and also showed that gratuitous overexpression of *ycgO* expressed from a P_{trc} promoter conferred a K^{S} phenotype in the *ptsN⁺* parent which was suppressed by plasmid borne expression of *tetA* and was independent of the *ptsPO* status of the strains. In the current year we observed that as seen in the case of the *ptsN* mutation, the K^{S} phenotype exhibited by overexpression of *ycgO* was also exacerbated in strains bearing null mutations in genes encoding TrkA and Kup K^+ transporters. We constructed using recombineering a chromosomal allele of *ycgO* that encodes a C-terminally 3XFLAG epitope tagged version of the YcgO polypeptide (YcgO_{CFL}). The tagging appeared to have inactivated YcgO as a strain bearing the *ptsN* null mutation and

expressing YcgO_{CFL} was not rendered K^{S} . Notwithstanding this alteration of YcgO function, we found that the level of YcgO_{CFL} was comparable in the parent and its *ptsN* derivative, indicating that the K^{S} phenotype caused by the *ptsN* mutation may not occur through overexpression of YcgO, rather overexpression may provoke activation of YcgO to yield a K^{S} phenotype similar to that exhibited by a strain lacking PtsN.

Overall our studies are consistent with a scenario wherein the absence of PtsN leads to enhancement in the activity of YcgO with $[\text{K}^+]_{\text{e}}$ serving as coactivator of YcgO, to mediate cytoplasmic K^+ release. In this model one of the two forms of PtsN, most likely dephospho-PtsN, represses the activity of YcgO, rendering it cryptic. We propose thus that in the *ptsN* mutant an unfettered K^+ release activity mediated by YcgO, is manifested that operates above the uptake capacity of the Trk and Kup K^+ uptake systems, is subjected to coactivation by $[\text{K}^+]_{\text{e}} > 30$ mM and against a background of the known $[\text{K}^+]_{\text{e}}$ mediated inhibition of the Kdp system, leads to K^+ limitation and hence causes the K^{S} phenotype.

In our earlier studies we have found that a strain doubly defective for the nucleoid protein H-NS and either thioredoxin 1, TrxA or thioredoxin reductase, TrxB (TH mutant) also displays a K^{S} phenotype similar to that displayed by a *ptsN* mutant in the sense that the K^{S} in both instances is exacerbated by the absence of TrkA and Kup and is suppressed by overproduction of Kup or by expression of *kdpA⁺* and *tetA*. Furthermore like that seen for the *ptsN* mutant the K^{S} of the TH mutant was also suppressed by absence of YcgO. In order to understand the interrelationship between the *trx hns* mutations on one hand and the *ptsN* mutation on the other, we have performed a test of the notion that the K^{S} phenotype of the TH strain may simply result because the *trx hns* mutations may cause a reduction in the level of PtsN and hence lead to a K^{S} phenotype. Towards this end we constructed a strain expressing a chromosomally encoded PtsN polypeptide (PtsN_{CFL}) with 3XFLAG epitope tag abutted to its C-terminal end and showed that the epitope tag did not alter the function of PtsN. Immunoblotting studies with anti-FLAG antibodies showed that strains singly deficient for H-NS, TrxA or TrxB or those bearing double deficiencies for TrxA H-NS or TrxB H-NS, displayed equivalent levels of PtsN_{CFL}, thus discounting the aforementioned notion. Our current studies are

directed towards determining cellular K⁺ levels in the *ptsN* mutant and its various suppressor derivatives to test the K⁺ limitation model. Furthermore, we will also be testing the notion that dephospho-PtsN may directly interact with YcgO and fetter its activity, for which we are constructing additional epitope tagged versions of YcgO that retain biological activity. Finally we are also examining the interrelationship, by employing alternate approaches, between the *ptsN* and the *trx hns* mutations.

3. Studies on basic amino acid export in *E. coli*.

Towards studies on regulation of basic amino acid export in *E. coli* we have previously reported characterization of the ORF *yggA* (*argO*) that encodes a novel arginine (Arg) exporter ArgO in *E. coli*, whose expression is regulated by the transcription factor ArgP. By employing certain gain-of-function variants of ArgP (ArgP^d) that cause high and constitutive expression of ArgO, leading to increased excretion of Arg into the culture medium, we have devised a process for microbial production of Arg that has also been patented by CDFD. Furthermore, under a program of co-operation with the IKP Knowledge Park, Hyderabad, improvements were undertaken to maximize the output of the patented process and the technology has been licensed by CDFD to a start-up company for commercialization. Towards understanding the mechanism of Arg export mediated by ArgO we have previously conducted mutagenesis and second-site suppressor studies on ArgO and have assessed its topology in the inner membrane. Furthermore we have reported the identification of *ybjE* (*lysO*), a gene whose product mediates export of L-Lysine (Lys) and another gene *ydhE* whose product appears to encode a second Arg exporter in *E. coli*.

In this year we extended studies on the genetic regulation of *lysO*. Previously we had identified the transcriptional start site of *lysO* and using this information we determined the location of the -10 and -35 promoter region of *lysO* and by performing site-specific mutagenesis studies we ascertained the authenticity of the -10 and -35 boxes. Earlier we had observed that the expression of a *lysO-lac* transcriptional fusion was impaired two-fold in Arg supplemented minimal medium in an ArgR dependent manner. This feature correlated with the presence of two putative binding sites for the ArgR repressor (ARG box), positioned (roughly centred at) -56 (box 1), with box 2 lying in a probable overlap

with the -35 hexamer sequence. We performed site specific mutagenesis of a T residue located in the 7th position of box 1 to find that Arg dependent ArgR repression of *lysO-lac* was eliminated suggesting that box 1 at least may represent one binding site for ArgR in the *lysO* regulatory region. In agreement with the above we found that purified ArgR bound to the *lysO* regulatory region encompassing the promoter and upstream sequences in an Arg dependent manner. Current studies in this regard are directed towards more detailed determination of the binding site for ArgR and the mechanism by which ArgR mediates its repressive effect on *lysO* expression. Furthermore we are also performing experiments to demonstrate LysO mediated L-lysine export into the culture medium.

Earlier studies from this laboratory on ArgP the transcriptional regulator of *argO* indicate that ArgP appears to be somewhat enigmatic in the sense that is capable of mediating specific transcriptional regulation and also may play a role as a global nucleoid structural protein. Currently we are engaged in examining the genome-wide transcriptional regulation imparted by ArgP following its binding to multiple targets on the *E. coli* chromosome and to identify new gene targets of ArgP. Towards this end we are engaged in a collaboration with Dr Tomohiro Shimada of the Tokyo Institute of Technology, Japan, who has obtained a genome-wide catalogue of binding sites for the transcription factor ArgP in presence and absence of its small molecule co-effectors namely L-arginine and L-lysine using the SELEX technology. Recently we have performed comparative phenotype microarrays (PM) studies on a strain that lacks ArgP and its parent in order to obtain biological correlates of the SELEX data. The analyses of the PM and the SELEX data is ongoing at present.

4. Genetic interactions between (p)ppGpp and tm-RNA/SmpB.

In work described in earlier reports the lethal phenotype observed during the combined deficiency of (p)ppGpp and tmRNA or SsrA (synthetic lethality) was genetically characterized and the following were inferred,

- a) Absence of (p)ppGpp –mediated modulation of transcription contributes to the observed synthetic lethality
- b) Genetic suppression studies using biochemically characterized RNAP mutants defective for elongation properties indicated that the synthetic lethal phenotype could be a

consequence of increased elongation rate and implied a possible role for (p)ppGpp in the modulation of transcription elongation *in vivo*.

- c) Studies done using various mutant alleles of *ssrA* indicated that its ribosome rescue function but not that of peptide tag addition is necessary for supporting cell survival in a (p)ppGpp deficient strain.

We had earlier reported that the *rho-4* allele which encodes a hypomorphic Rho protein confers suppression of synthetic lethality. Further studies have shown that suppression is also seen using the antibiotic bicyclomycin which inhibits Rho function or a defective *nusG* allele (*nusG*-G146D) that is compromised for rho-dependent termination. These results indicate that uncoupling of transcription from translation contributes to the synthetic phenotype through rho-dependent termination of transcription. Since it has been previously shown that the ribosome rescue function of SsrA was sufficient for rescue of synthetic lethality it can be reasoned that ribosome rescue prevents uncoupling in the ppGpp⁰ strain. This line of reasoning would imply that the SsrA rescued ribosomes are not at the 3' end of mRNA (non-stop mRNA) because under those conditions even following ribosome rescue productive coupling of translation with transcription cannot be reestablished. Our results favor the model that SsrA mediated rescue occurs on the pioneer ribosome (the ribosome that follows the RNAP) on an intact mRNA, and thereby allowing subsequent ribosomes to reestablish coupling and prevent premature termination of transcription. The following genetic evidence also support this idea of SsrA rescue of ribosome on intact mRNA,

- (i) Generation of non-stop mRNA-ribosome complex *in vivo* can be mediated by the action of certain mRNA endoribonucleases that make up the toxin component of the toxin-antitoxin systems. A strain lacking 10 toxin-antitoxin loci did not show any significant alleviation of the *ssrA* ppGpp⁰ synthetic lethality indicating that generation of non-stop mRNA may not be an important determinant in the synthetic phenotype.
- (ii) There is evidence in the literature that ribosome pausing at rare codons lead to SsrA-mediated rescue even without the generation of non-stop mRNA. To study if such ribosome pausing events contribute to the synthetic phenotype,

plasmid pRARE was used. pRARE is a p15A based plasmid carrying 10 tRNA genes that decode the rare codons in *E. coli*. The presence of pRARE increases the abundance of these tRNA's and facilitates translation of rare codons. We found that the ppGpp⁰ *ssrA* synthetic phenotype was suppressed in the presence of pRARE but not by control plasmid pACYC184, suggesting that rescue of ribosomes paused at rare codons by SsrA is important for the survival of the ppGpp⁰ strain.

A recent study proposed physical coupling between RNAP and ribosomes and that the coupling coordinates the speed of transcription with translation. The ribosomes assist in the forward translocation of RNAP by physically hindering the backtracking activity of RNAP. Based on this study, the time required for the synthesis of β -galactosidase following IPTG induction, in the presence or absence of sub-inhibitory concentration of chloramphenicol (to reduce the rate of translation) was monitored for the wild-type and ppGpp⁰ strain. As reported, a lag in β -galactosidase synthesis time is seen for the wild-type strain after chloramphenicol addition, however the same is not observed in the ppGpp⁰ strain. We do not have an explanation for this observation, but clearly, (p)ppGpp seems to be important for the observed lag in β -galactosidase synthesis in the presence of chloramphenicol, furthermore, that chloramphenicol addition can result in the disappearance of cellular (p)ppGpp is a well documented phenomenon.

5. (p)ppGpp and modulation of cell division.

During our studies using the ppGpp⁰ strain we identified that deletion of the *lon* gene resulted in loss of cell viability. Two well studied targets of Lon protease are RcsA and Sula. Increase in levels of RcsA, a positive transcriptional regulator of capsular polysaccharide, results in increased capsular polysaccharide synthesis through its interaction with RcsB. Increase in Sula levels inhibits cell division by inhibiting the activity of the cell division protein FtsZ. We deleted *rscB* or *sula* to find out the role of each in the inviability conferred by *lon* deletion in the ppGpp⁰ strain. Deletion of *sula*, but not *rscB*, suppressed the inviability indicating that inhibition of FtsZ could be the reason for the inviability of the ppGpp⁰ *lon* mutant. The *sula* gene is part of the SOS regulon and following

DNA damage, endogenous SulA level increases due to the activation of the regulon and inhibits FtsZ activity. We therefore tested if endogenous DNA damage in the ppGpp⁰ strain resulted in increased SulA levels by using *sulA-lacZ* reporter fusions. No significant difference in reporter activity was observed between the wild-type and ppGpp⁰ strains, suggesting that activation of the SOS regulon may not be the reason for SulA dependent inviability of the ppGpp⁰ *lon* mutant. It was observed that the *lexA3* allele, encoding the non-cleavable LexA protein that constitutively represses the SOS regulon conferred suppression of ppGpp⁰ *lon* inviability. These results are consistent with the following interpretation; basal levels of SulA protein (in the absence of DNA damage) when stabilized due to absence of Lon protease is sufficient to inhibit the growth of the ppGpp⁰ strain, and the *lexA3* allele allows survival by further lowering of the SulA levels.

6. Isolation and characterization of mutations that tolerate deletion of the *spoT* gene.

Wild-type *E. coli* loses viability upon deletion of *spoT* gene, however, it is possible to delete *spoT* gene in a Δ *relA* mutant, suggesting that SpoT performs an essential function in the presence of RelA activity. Since the only known function of RelA is (p)ppGpp synthesis, it was reasoned that deletion of *spoT* results in (p)ppGpp accumulation and loss of viability, although this is yet to be proved. Furthermore, the functions targeted by accumulation of (p)ppGpp is not known. Using transposon mutagenesis an attempt was made to screen for mutations in genes other than in *relA* that would allow viability in a Δ *spoT* background. A Δ *spoT* strain carrying the shelter plasmid pRC-*spoT* was mutagenized to select for mutants that can survive in the absence of shelter plasmid and subsequently screened for growth in minimal media to identify mutations in genes other than *relA*, because a Δ *relA* Δ *spoT* strain does not grow in minimal media. Mutants so obtained were further tested for their ability to grow in the presence of Serine, Methionine and Glycine (SMG resistance) a test for functionality of RelA protein. The genomic location of the mutations identified by sequencing the transposon junctions revealed two kinds of mutation, one at the end of the *rlmD* ORF and the second within the *relA* ORF after the 496th codon. We believe these to be hypomorphic alleles of *relA* that can survive *spoT* deletion and confer SMG resistance. Interestingly, the SMG resistance conferred by the mutations, especially the one

within the *relA* ORF is seen only in the Δ *spoT* background and lost in the *spoT*⁺ background. We have characterized the (p)ppGpp accumulation pattern in the *relA*⁺ Δ *spoT* background and preliminary results indicate that it is different from that seen during a stringent response.

7. Role of transketolases in *E. coli* physiology.

Genetic studies carried out in transketolase deficient strain i.e., a *tktA tktB* double mutant and previously reported, revealed that transketolase activity is essential to sustain the growth of *E. coli* in LB media and that the growth defect can be partially compensated by: a) elimination of the purine/pyrimidine salvage pathways through inactivation of DeoB (phosphopentomutase) resulting in reduced ribose-5-phosphate pool; b) activation of *glpK*, coding for glycerol-3-phosphate kinase; c) presence of the *pntAB* genes, coding for the subunits of the membrane bound pyridine nucleotide trans-hydrogenase on multi-copy plasmid and presumed to increase its intracellular activity; or d) glucose supplementation to LB media. Based on the premise that the loss of transketolase activity results in altered pyridine cofactor levels, their levels were measured in the wild-type, transketolase mutant and the different suppressor strains. The results clearly indicate that with the exception of NADP⁺ the intracellular levels of the other cofactors are dramatically lowered in the transketolase deficient strain as compared to the wild-type strain, and the presence of the suppressor mutations by and large restored the cofactor levels to that observed under permissive growth condition in the *tktA tktB* double mutant.

To understand the metabolic consequences of transketolase deficiency, we also carried out metabolomic analyses by GC-MS and compared the profiles of the wild-type strain with that of the transketolase deficient strain following growth in LB. The analyses revealed the relative concentrations of a limited set of compounds. A predicted consequence of transketolase deficiency is accumulation of pentose sugars, and this is seen in the metabolic profile; the four pentose sugars detected in the analysis, namely, ribulose-5-P, ribitol, arabinose, and ribose were 20-fold, 5-fold, 2.4-fold and 2.2-fold higher in the mutant as compared to that in the wild-type. Analysis of the data after grouping metabolic intermediates of different pathways indicate significant decrease in the levels of Krebs's cycle intermediates in the transketolase mutant. Relative to the wild-type

strain, citrate, oxoglutarate and succinate levels were 4.8, 5.7 and 7.6 fold lower in the transketolase deficient strain, suggesting lower flux in the Krebs cycle in the mutant and provides a possible explanation for the lowered levels of NADH and NADPH we had noted earlier. Another compound significantly lower in the transketolase mutant was the cysteine-glycine dipeptide (13-fold less). We think the dipeptide could be the breakdown product of glutathione (γ -L-glutamyl-L-cysteinyl-glycine) and indicative of lowered glutathione pools in the transketolase mutant. It is also possible that this may be related to the reduced levels of NADPH in the transketolase mutant since glutathione synthesis from glutathione disulphide requires the NADPH utilizing glutathione reductase. Unfortunately, the metabolomic analysis did not provide the relative concentrations of any of the pyridine nucleotides.

To find further support for the idea that transketolase activity is required for maintaining the balance of pyridine nucleotides and that pyridine nucleotide imbalance is the cause for the growth defect in a transketolase deficient strain, we tested the ability of a glycolytic enzyme with a pyridine cofactor requirement different from the *E. coli* enzyme to suppress growth defect. We replaced the *E. coli gapA* gene coding for glyceraldehyde-3-phosphate dehydrogenase (GAPDH) with that from *Streptococcus mutants* that carries out the same reaction but using NADP⁺ as the cofactor instead of NAD⁺ that is used by the *E. coli* enzyme, and interestingly, we observed growth suppression. The following possibilities are being studied to explain suppression of growth defect, namely, activation of the lower half of the glycolytic pathway or restoration of NADPH pools or both. The transketolase deficient strain has low NAD⁺ levels, while the NADP⁺ levels are not perturbed significantly, this could potentially reduce the *E. coli* GAPDH activity which uses NAD⁺ as cofactor

but not that of *S. mutants* since it uses NADP⁺ as cofactor. In cells, NADPH is produced by the reduction of NADP⁺, which in turn is mainly produced by the phosphorylation of NAD⁺. The most prominent function of NADP⁺ is to maintain a pool of NADPH as reducing equivalents for metabolic reactions and the cellular concentration of NADPH is higher than that of NADP⁺. However, strikingly, in the transketolase mutant the NADPH drops to undetectable levels. It is therefore possible that suppression of growth defect by the *S. mutants gapA* is due to NADPH synthesis.

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1. Gowrishankar J, Krishna Leela J and Anupama K (2013). R-loops in bacterial transcription: Their causes and consequences. ***Transcription*** 4: 153-157.
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3. Sarkar P, Sardesai AA, Murakami KS and Chatterji D (2013). Inactivation of the bacterial RNA polymerase due to acquisition of secondary structure by the ω subunit. ***Journal of Biological Chemistry*** 288: 25076-25087.
4. Phulera S, Akif M, Sardesai AA and Mande SC (2014). Redox proteins of *Mycobacterium tuberculosis*. ***Journal of the Indian Institute of Science*** 94: 127-137.

Patents

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Indian Patent Application No.5465/CHE/2013
Date of filing: November 27, 2013

LABORATORY OF CELL CYCLE REGULATION

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

Faculty	Shweta Tyagi	Ramalingaswami Fellow
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	Zaffer Ullah Zargar	Senior Research Fellow
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	B Sumani Bharadwaj	Project-Junior Research Fellow (Since Jun. 2013)
	SJ Yogaspoorthi	Project Assistant (Since Jan. 2014)

Objectives

1. Identification of new effector proteins involved in regulation of E2F-responsive promoters; and
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 (upto March 31, 2013)

We decided to take a candidate based approach to look for effector proteins. We had expressed and purified E2F4 as GST fusion to homogeneity. We also cultured HeLa Spinner cells which can be used for producing large amounts of cells in suspension cultures. HeLa cells were grown in Joklik's media and growth conditions were standardized.

Details of the progress made in the current reporting year (April 1, 2013–March 31, 2014)

Once we isolate our E2F4 interacting partners, we would want to identify the domain of E2F4 that mediated this interaction. For this we cloned and expressed E2F4 deletions as GST-fusion protein.

We are also expressing E2F4 as triple-epitope-tagged fusion protein for tandem affinity purification from HeLa spinner cells.

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. We have shown that MLL-family of H3K4 histone methyltransferases is linked to cell cycle regulation by interacting with E2F1. 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 (upto March 31, 2013)

To find out the role of MLL complex in cell cycle regulation, in our previous report we demonstrated that loss of WDR5 resulted in delay in progression of mitosis.

Details of the progress made in the current reporting year (April 1, 2013 –March 31, 2014)

Here we show that the MLL has a regulatory role during multiple phases of the cell cycle. RNAi mediated knockdown reveals that MLL regulates S phase progression and, proper segregation and cytokinesis during M phase. Using deletions and mutations, we narrow the cell-cycle regulatory role to the C subunit of MLL.

MLL siRNA-treated U2OS cells displayed a pronounced decrease in cell growth (Fig. 1A). Further, to determine the proliferative status of individual MLL depleted cells, we assayed the cells

for S phase entry using long-term bromodeoxyuridine (BrdU) incorporation by indirect immunofluorescence staining (IFS). When stained with BrdU antiserum, about 50% of MLL depleted cells were deficient in incorporating BrdU, 72 hours after siRNA treatment (see arrowheads Figs. 1B & 2A, sample 2). Our results show that MLL is required for mammalian cells to proliferate.

When examined, the MLL siRNA-treated cells displayed high number of binucleated cells (see closed arrowhead in Fig. 1C and panel a) as opposed to control siRNA-treated U2OS cells, probably arising from defective cytokinesis. We also noticed the presence of micronuclei in MLL depleted cells. Micronuclei are recognized as small distinct bodies of chromatin in the cytoplasm of interphase mammalian cells and considered as

marker of chromosome loss during mitotic segregation (see open arrowhead in Fig. 1C and panel b).

When quantified, U2OS cells displayed 2-4% defective cells even before any siRNA treatment, and control siRNA treatment did not exacerbate the count. In contrast, MLL siRNA treatment resulted in significantly higher number of cells displaying mitotic defects (Fig. 2B, sample 2). Our results indicate that MLL may regulate multiple steps in mitosis and depletion of MLL results in segregation and cytokinesis defects.

To identify the regions of MLL required to promote S and M phase progression, a set of recombinant Flag epitope-tagged MLL protein deletions were stably expressed in U2OS cells (Fig. 2). We were

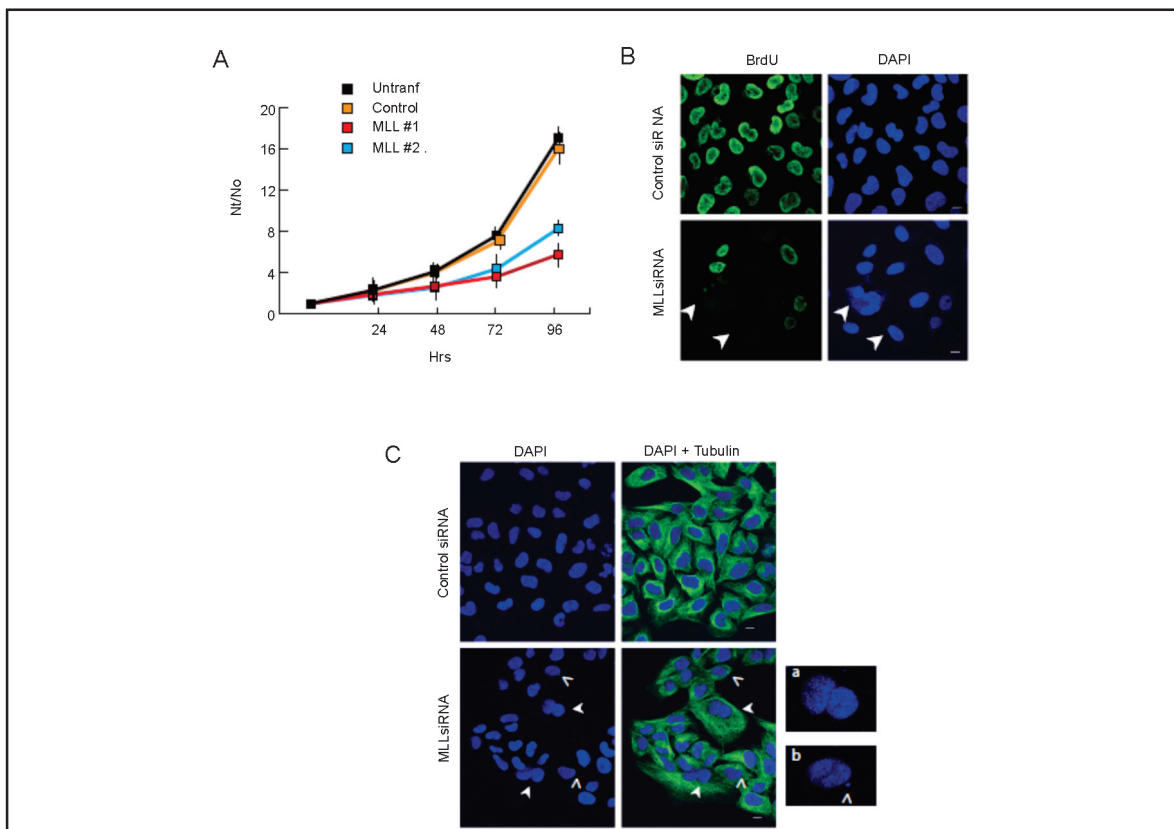
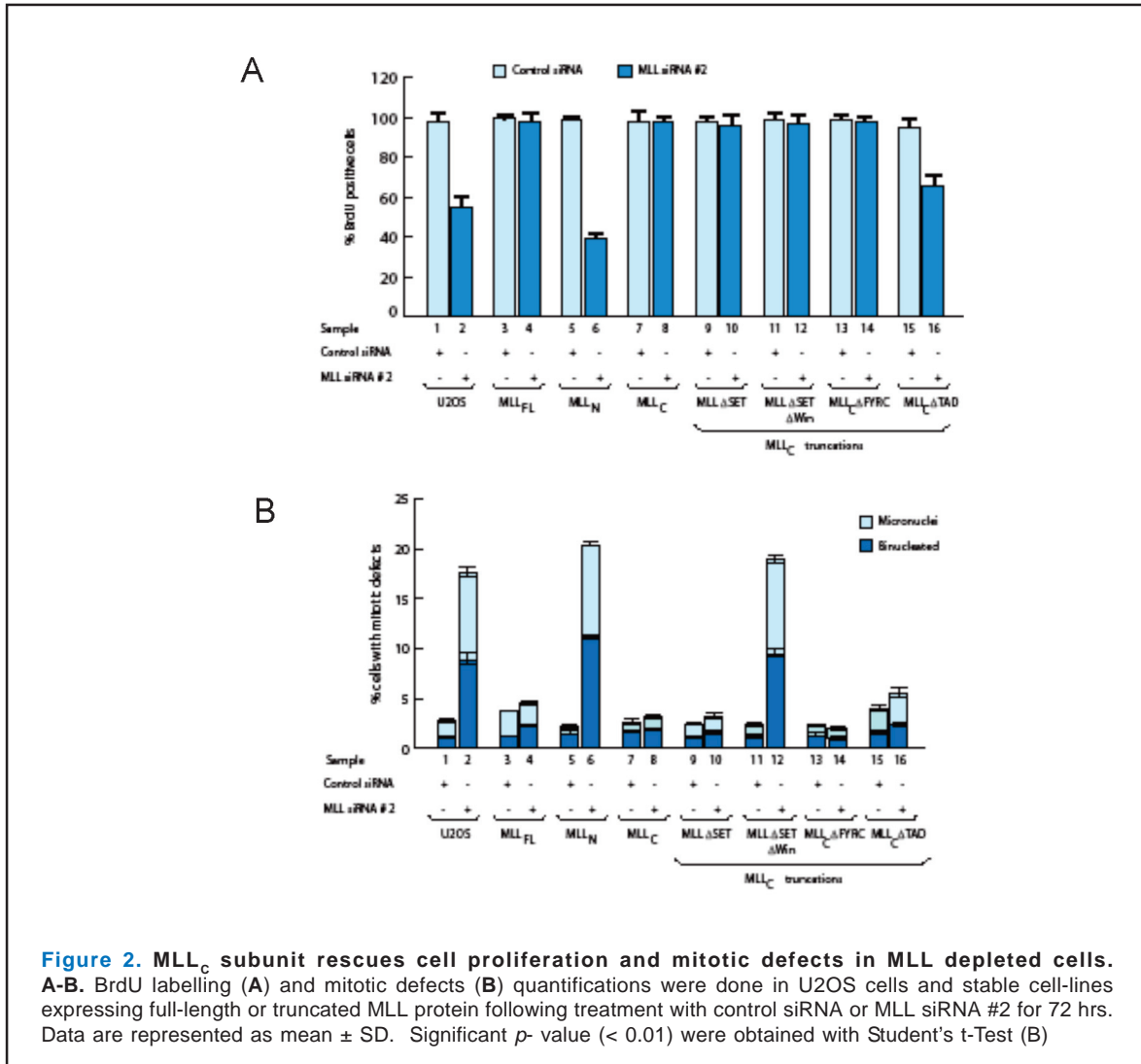


Figure 1. MLL loss-of-function leads to growth arrest and mitotic defects.

- Growth curves of untransfected (Black), control siRNA-transfected (Orange), or MLL siRNA [siRNA #1(red) or #2 (blue)] transfected U2OS cells were generated by plotting the total number of live cells (N_t) divided by number of cells seeded on day 0 (N₀). The cells were harvested at 24 hrs, 48 hrs, 72 hrs, and 96 hrs from duplicate experiments after siRNA treatment, stained with trypan blue, counted, and averaged results are shown.
- Immunofluorescence analysis of BrdU incorporation in control or MLL siRNA transfected cells was done by staining cells with anti-BrdU antibody and DAPI. Arrowheads point to BrdU negative cells. Scale 5µm.
- Immunofluorescence analysis showing mitotic defects (binucleation and micronuclei) upon MLL depletion in U2OS cells. The cells were stained with DAPI and anti-Tubulin antibody. Closed arrowheads and panel a show binucleated cells; open arrowheads and panel b show cells with micronuclei. Scale 5µm.

able to narrow the required region to 9 amino acid in transcriptional activation (TA) domain of MLL indicating that the transcriptional activity of TA domain of MLL and not the methyltransferase activity of SET domain is required for passage of cells into S phase. The region of MLL involved in mitosis and cytokinesis, turned out to be the WDR5

interacting (Win) motif in MLL C construct, where by changing arginine 3765 to alanine, we were able to abolish the binding of the whole WDR5, RbBP5, Ash2L, and Dpy30, (WRAD) complex to MLL. These mitotic functions of WRAD are independent of SET-domain of MLL and, therefore, define a new role of WRAD in subset of MLL functions.



LABORATORY OF CELL DEATH & CELL SURVIVAL

Functional protein networks controlling cell life and death

Faculty	Maddika V Subba Reddy	Scientist & WT-DBT India Alliance Intermediate Fellow
PhD Students	Neelam Chaudhary PV Vivek Reddy G Narmadha Reddy Swapnil R Shinde Parveen Kumar Varun J Shah	Senior Research Fellow Senior Research Fellow Senior Research Fellow Senior Research Fellow Senior Research Fellow Junior Research Fellow
Other Members	J Kiranmai M Prathyusha KVS Rammohan Chowdary K Nanci Rani	Project-Junior Research Fellow Project-Junior Research Fellow Project- Junior Research Fellow (Since Sept. 2013) Technical Assistant

Objectives

1. To dissect the functional network of phosphatases regulating cell life and death;
2. To identify and characterize novel protein complexes in maintaining genomic stability; and
3. 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, 2013)

Phosphatases play a crucial role in biological functions and controls 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 the cell. In this work we have already identified and characterized WWP2, PNUTS and TOPK as novel functional partners of PTEN (Maddika *et al.*, Nature Cell Biol. 2011, Kavela *et al.*, Cancer Res. 2013, Shinde *et al.*, Cell Signal 2013). In addition to PTEN interactome, we also started developing networks of other cellular phosphatases. We identified a deubiquitinase complex WDR48-USP12 as a regulator of another tumor suppressor phosphatase PHLPP1 (Gangula NR & Maddika S., JBC 2013).

Details of progress made in the current reporting year (April 1, 2013-March 31, 2014)

Project 1: Functional studies on phosphatase networks.

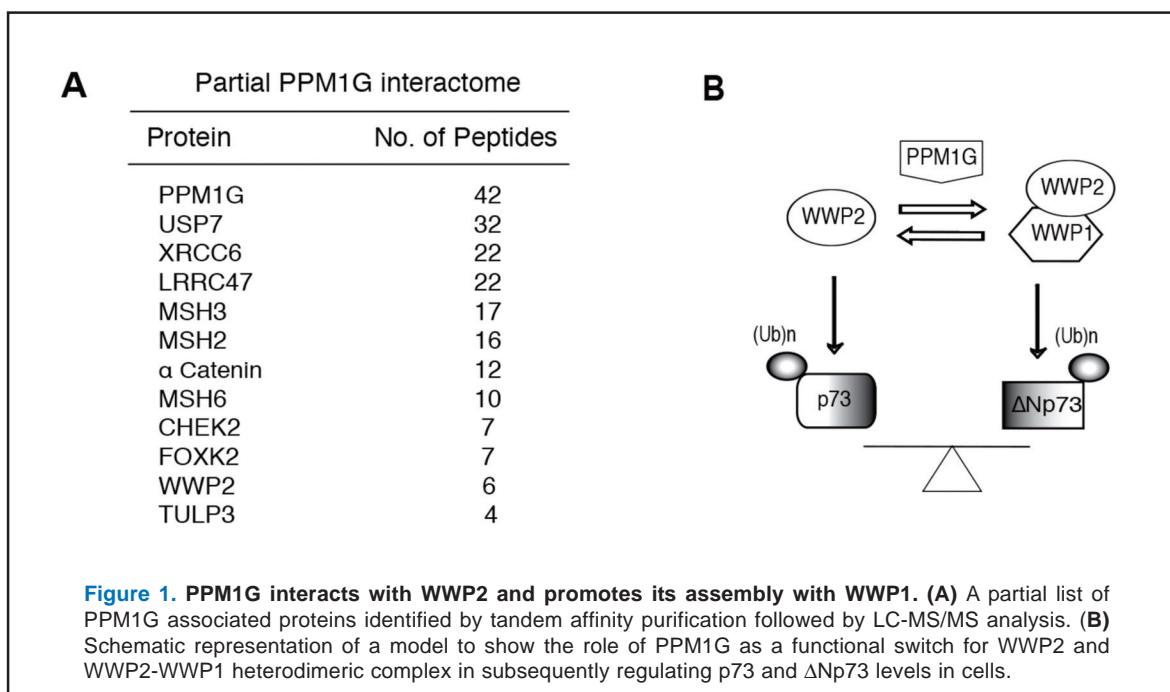
Currently, we are focused on actively expanding the network of all the available phosphatases in cell. In this direction, we already acquired a cDNA library that contains 160 phosphatases. We are now systematically purifying the complexes of phosphatases from different families such as lipid phosphatases, dual specificity phosphatases, non-receptor tyrosine phosphatases, receptor tyrosine phosphatases, PPM family of phosphatases etc. So far we cloned 110 phosphatases into a triple tagged mammalian expression vector and confirmed their expression in cells. Further, by using tandem affinity purification approach followed by LC-MS/MS analysis we identified and mapped the complexes of 52 phosphatases until now. While we continue to purify the complexes of remaining phosphatases in the library and aim to finally build the whole 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 the interaction of PPM family of phosphatases in particular PPM1G with its newly identified associated proteins.

PPM family of phosphatases play critical role in regulating the stress response, cell-cycle

progression, apoptosis, Ca²⁺ signalling, metabolism, RNA splicing, mitochondrial function and lipid transfer. Several members of this phosphatase family have been attributed to function either as tumor suppressors or oncogenes. PPM1G also known as PP2C γ is a Mg²⁺/Mn²⁺ dependent nuclear serine/threonine phosphatase that plays an important role in different functions such as nucleosome assembly, cell survival control, mRNA splicing and DNA damage response. Tandem affinity purification followed by mass spectrometry analysis allowed us to identify several novel associated proteins in PPM1G complex (Fig. 1A). Among these proteins, E3 ligase WWP2 caught our immediate attention as we have recently shown WWP2 as a negative regulator of PTEN phosphatase and thus a potential oncogene (Maddika et al., Nature Cell Biol 2011). We

confirmed the association of PPM1G and WWP2 in cells. We found that PPM1G acts as a functional molecular switch that controls the balance between monomeric WWP2 and its heterodimeric complex form with another E3 ligase WWP1. The switch between WWP2 and WWP2/WWP1 complex is crucial for maintaining the balance between protein levels of transcription factor p73 and its functionally opposing N-terminal truncated Δ Np73 isoform, which subsequently is necessary for cell survival (Fig. 1B).

Our further studies in this project are focused on mapping the functional networks of other phosphatases in cells such as lipid phosphatases, dual specificity phosphatases, non-receptor protein tyrosine phosphatases and receptor protein tyrosine phosphatases.



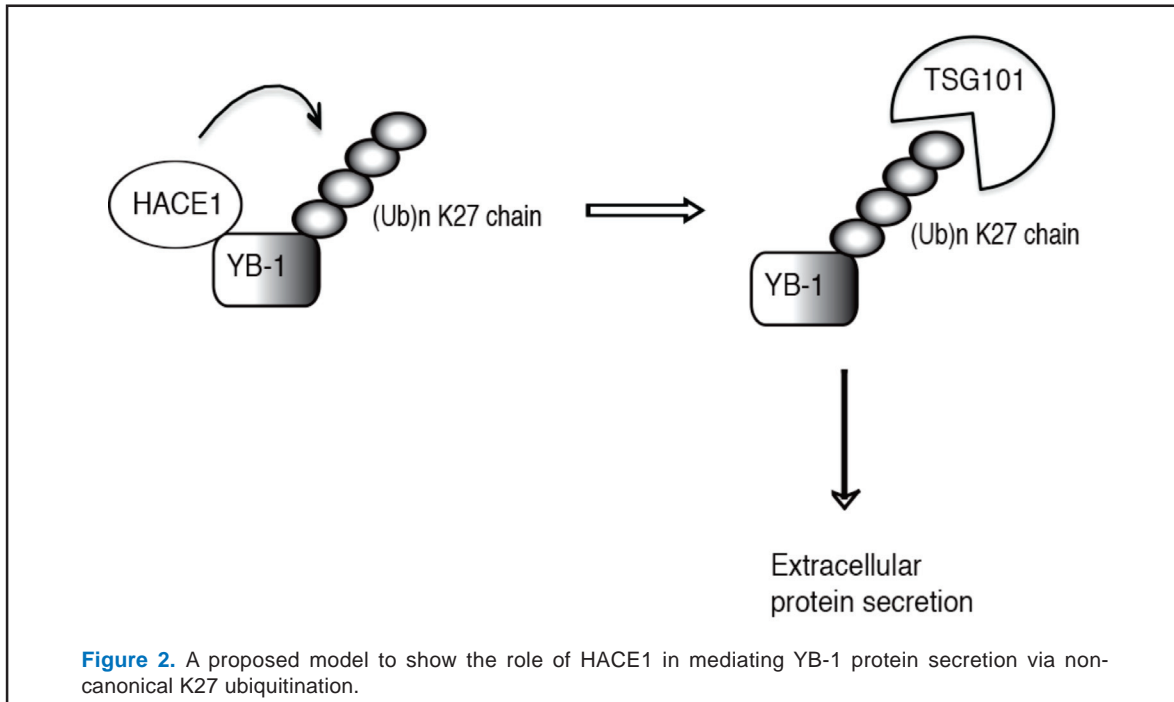
Project 2: Roles of canonical and non-canonical ubiquitination in cells.

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.

While the importance of ubiquitination in controlling the fate and the intracellular functions of various proteins was widely studied, its role in extracellular protein secretion has been unexplored so far. In this study, by using YB-1 (Y-box Binding protein 1) as a model protein, we showed that ubiquitination is required for its extracellular secretion. We also identified HACE1 as a specific E3 ligase that polyubiquitinates YB-1 but through non-canonical K27 linked ubiquitin chains. Formation of these ubiquitin linkages on YB-1 is necessary for its interaction with Tumor Susceptibility Gene 101 (TSG101), a component

of the Multi Vesicular Body (MVB) pathway, which facilitates its secretion. Finally, we demonstrated that extracellular secreted YB-1 is a functional protein that acts to inhibit Transforming Growth

Factor-Beta mediated epithelial to mesenchymal transition. In summary, we identified a novel functional role for non-canonical ubiquitin linkages in mediating protein secretion (Fig. 2).



Project 3: Identification and characterization of novel protein complexes in maintaining genomic stability.

In this work, we are particularly interested in identifying novel functional protein complexes that regulate DNA replication and cell cycle progression both of which are critical for maintaining genomic stability. By using proteomics based approach, we recently identified protein kinase SNF1LK as a novel regulator of eukaryotic DNA replication, which functions by associating with GINS complex at the replication forks. We found that SNF1LK phosphorylates the MCM components and is required for MCM helicase activity.

Simultaneously, in this project we are trying to delineate the non-canonical functions of splicing factors in regulating genomic stability. Several studies have indicated that many of the splicing proteins are involved in maintaining genomic stability independent of their splicing function. We recently initiated our studies in this direction since

the molecular mechanisms of their role in this process is completely unknown.

Publications

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2. Shinde SR, Gangula NR, Kavela S, Pandey V and Maddika S (2013). TOPK and PTEN participate in CHFR mediated mitotic checkpoint. *Cellular Signalling* 25: 2511-17.
3. Zhang J, Zhang P, Wei Y, Piao HL, Wang W, Maddika S, Wang M, Chen D, Sun Y, Hung MC, Chen J and Ma L (2013). Deubiquitylation and stabilization of PTEN by USP13. *Nature Cell Biology* 15: 1486-94.

LABORATORY OF CELL SIGNALLING

Investigating the role of inositol pyrophosphates in eukaryotic cell physiology

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Other Members	L Padmavathi Ruth Manorama Ravoori Dharmika Kumar Somadri Ghosh CP Unnikannan Ujjwal Banerjee Rahul Gopalam	Scientist Technical Assistant Project-Senior Research Fellow (Till May 2013) Project-Senior Research Fellow (Till Sep. 2013) Project-Senior Research Fellow Project-Junior Research Fellow (Till Nov. 2013) Project-Junior Research Fellow (Since Dec. 2013)
Collaborators	Satish Kumar Nagaraj Balasubramanian Roop Mallik Kana M Sureshan	CCMB, Hyderabad IISER, Pune TIFR, Mumbai IISER, Thiruvananthapuram

Objectives

Inositol pyrophosphates are derivatives of inositol that contain pyrophosphate or diphosphate moieties in addition to monophosphates. They include diphosphoinositol pentakisphosphate (PP-IP₅, or IP₇) and bis-diphosphoinositol tetrakisphosphate ([PP]₂-IP₄ or IP₈), which participate in diverse biological functions, including DNA recombination, vesicular trafficking, apoptosis 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-IP₅ (IP₇) is synthesised from inositol hexakisphosphate (IP₆) and ATP by IP₆ kinases. Mammals have three isoforms of IP₆ kinase, IP6K1, IP6K2 and IP6K3, whereas *Saccharomyces cerevisiae* have a single IP₆ kinase, Kcs1.

Our aim is to understand the biochemical link between protein pyrophosphorylation and cellular phenomena 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. Examine the role of inositol pyrophosphates in yeast physiology;
2. Understand the cellular functions of mammalian inositol hexakisphosphate kinase 1 (IP6K1); and
3. Study the role of inositol pyrophosphates in whole animal physiology.

Summary of work done until the beginning of this reporting year (upto March 31, 2013)

We observed that *S. cerevisiae* strains lacking Kcs1 display slow growth, increased sensitivity to translation inhibitors and decreased protein synthesis compared with wild type yeast. These phenotypes can be reversed upon the expression of enzymatically active Kcs1, but not the inactive form. *kcs1Δ* yeast exhibit reduced levels of ribosome subunits, suggesting that they are defective in ribosome biogenesis. The rate of rRNA synthesis, the first step of ribosome biogenesis, is decreased in *kcs1Δ* yeast, suggesting that RNA polymerase I (Pol I) activity may be reduced in these cells. We determined that the Pol I subunits, A190, A43 and A34.5 are pyrophosphorylated by IP₇ on serine residues falling within mobile regions on the surface of the enzyme.

To understand the cellular functions of IP₇ in mammals, we use mouse embryonic fibroblasts (MEFs) derived from IP6K1 knockout (*Ip6k1*^{-/-}) embryos, which have 70% reduced levels of IP₇ compared with wild type (WT) MEFs. These cells provide an excellent model to study specific cellular functions of inositol pyrophosphates that may be biochemically linked with protein pyrophosphorylation. In earlier reports, and in a recent 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 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 knock-out MEFs, implying that inositol pyrophosphates are required for HR-mediated repair.

To study the role of inositol pyrophosphates in whole animals, we have established a colony of *Ip6k1*^{+/-} heterozygous mice and are breeding them to obtain wild type and knockout litter-mates. Budding yeast lacking the IP₆ kinase Kcs1 display substantially lowered levels of inorganic polyphosphate (polyP), a linear polymer of orthophosphate moieties linked by phosphoanhydride bonds. We therefore used *Ip6k1*^{-/-} mice as a model system to determine whether the link between inositol pyrophosphates and polyP is conserved in mammals. PolyP of chain length 60-100 phosphate units is present in dense granules of mammalian platelets, and regulates the blood clotting cascade at multiple stages. We noted that *Ip6k1*^{-/-} platelets have lower levels of polyP compared with WT mouse platelets. Analysis of haematologic parameters in WT and *Ip6k1*^{-/-} mice revealed no difference in platelet count, platelet size or other blood parameters between these groups. Platelet activation by thrombin was unchanged, but platelet aggregation upon thrombin stimulation of washed platelets was lower in *Ip6k1*^{-/-} mice. Plasma clotting time in presence of platelet releasates was lengthened in *Ip6k1*^{-/-} mice. These data established that the metabolic link between inositol pyrophosphates

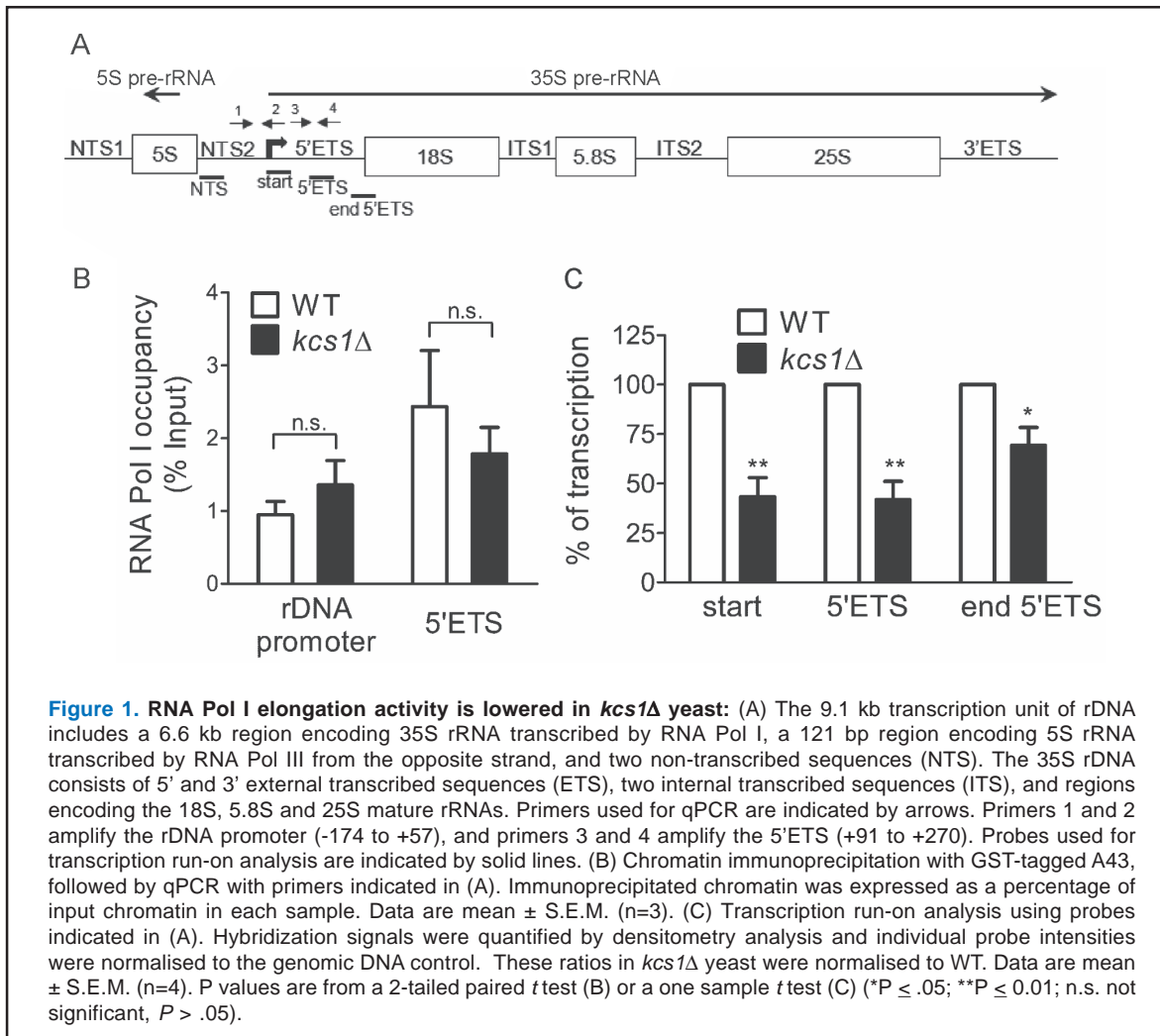
and polyP is conserved between yeast and mammals, and suggested that *Ip6k1*^{-/-} mice may display defects in haemostasis.

Details of progress made in the current reporting year (April 1, 2013 - March 31, 2014)

Project 1: Regulation of yeast ribosome biogenesis by IP₇.

To determine whether IP₇-mediated pyrophosphorylation of one or more serine residues is required for RNA Pol I activity, we generated several Ser to Ala mutants in A190, A34.5 and A43. However, none of the individual mutants displayed any decrease in growth, temperature sensitivity or protein synthesis (data not shown). It is possible that pyrophosphorylation at multiple sites is necessary for optimal RNA Pol I function.

IP₇-mediated pyrophosphorylation may be required for Pol I binding to the rDNA promoter, transcription initiation, or elongation. In *S. cerevisiae*, each rDNA unit encodes a 35S pre-rRNA transcribed by Pol I, which can be divided into regions coding for the mature 18S, 5.8S and 25S rRNA, two external transcribed regions (ETS) and two internal transcribed regions (ITS) which are cleaved during 35S pre-rRNA processing (Fig. 1A). We used chromatin immunoprecipitation assays to monitor recruitment of the Pol I complex to the rDNA promoter. There is no difference in promoter binding of Pol I from WT and *kcs1Δ* yeast (Fig. 1B). To examine the levels of active elongating Pol I, we measured Pol I bound to the 5' external transcribed sequence (5'ETS), that occurs approximately 200 bp downstream of the promoter. There is no significant difference in Pol I occupancy of this region in the rDNA locus in WT and *kcs1Δ* yeast. These results suggest that IP₇-mediated pyrophosphorylation does not influence the recruitment of Pol I to the rDNA locus. We measured the elongation efficiency of Pol I in a nuclear run-on assay, which measures transcription by RNA polymerase that is already bound to DNA, while preventing recruitment of new polymerase molecules to DNA. The levels of nascent transcript were significantly lower in *kcs1Δ* yeast compared with WT (Fig. 1C), indicating a reduction in the rate of transcription elongation by Pol I in yeast lacking inositol pyrophosphates. We conclude that IP₇-mediated pyrophosphorylation of three different subunits is required to maintain optimal elongation efficiency in Pol I. This project has now been completed and a manuscript is in preparation.



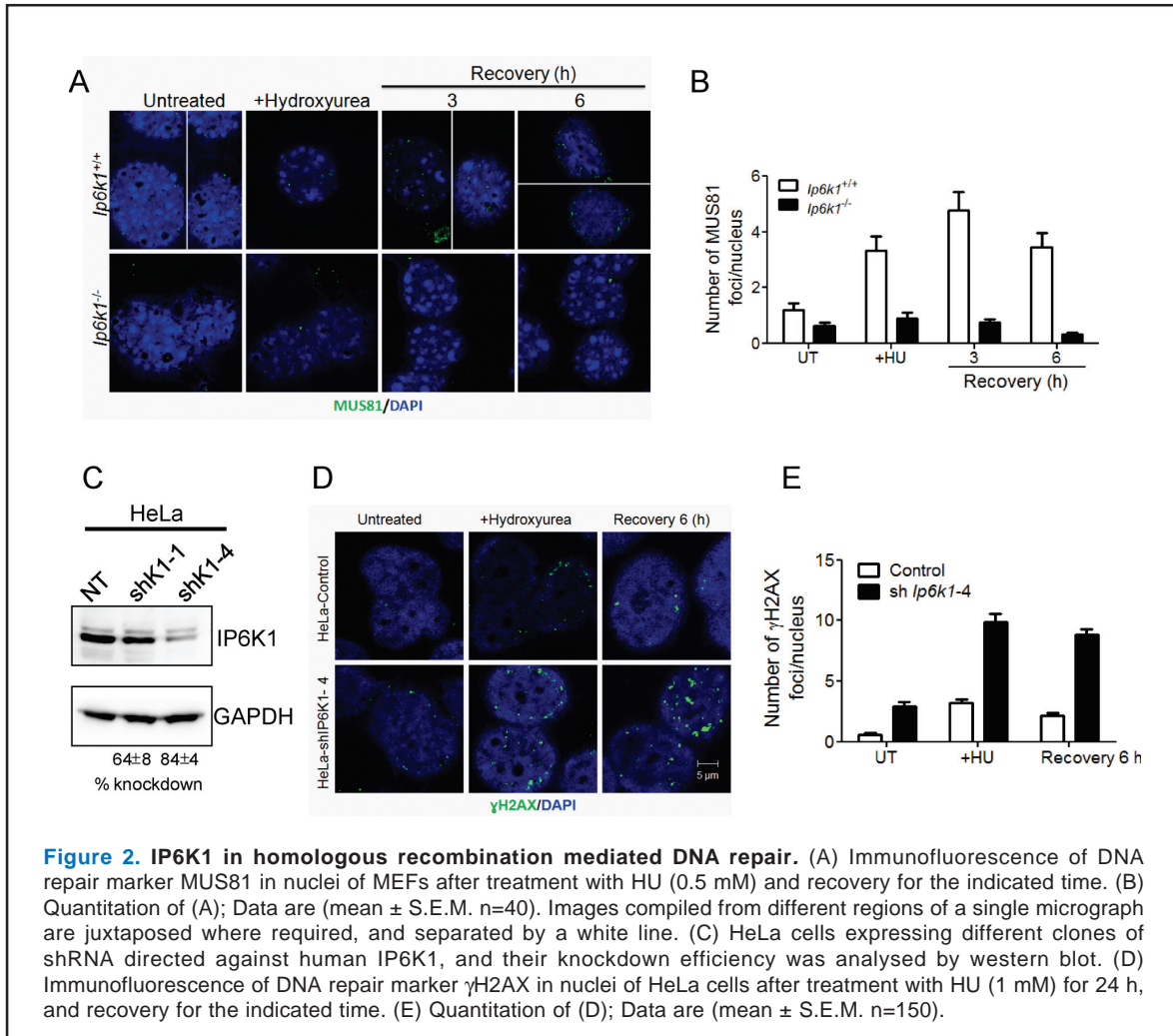
Project 2: Cellular functions of mammalian inositol hexakisphosphate kinase 1 (IP6K1): Role of inositol pyrophosphates in homologous DNA recombination.

At the molecular level, inositol pyrophosphates may influence DNA repair by binding or pyrophosphorylating one or more proteins involved in the later stages of HR, including DNA synthesis, ligation, and Holliday junction resolution. To determine the stage of HR at which *Ip6k1*^{-/-} MEFs are stalled, we utilised another marker, MUS81, a nuclease involved in resolution of Holliday junctions towards the end of HR repair. MUS81 is recruited to DNA damage foci during recovery from HU treatment in wild type, but not in *Ip6k1*^{-/-} MEFs (Fig. 2A, B), suggesting that HR repair is stalled in knockout MEFs prior to the formation of Holliday junctions. It is possible that IP₇ is involved in the removal of Rad51 from DNA damage sites, a function performed by multiple factors, including

Rad54 and the C-terminal domain of BRCA2. As we were limited by reagents available for analysing these proteins in mouse cells, we shifted to a more tractable human cell line for these analyses. We tested five shRNA constructs for knockdown of human IP6K1 in HeLa cells and found two constructs that significantly reduced IP6K1 levels (Fig. 2C). Treatment of non-targeted and sh*Ip6k1* expressing HeLa cells with HU, followed by monitoring recovery for 6 hours, revealed that the knockdown of *Ip6k1* expression in HeLa cells has the same effect on HR repair as knocking out *Ip6k1* in MEFs (Fig. 2D, E). We are currently using these cells as a model system to determine the exact step of HR and the specific proteins targeted by IP₇.

Project 3: Physiological role of IP₇ in mice: Regulation of platelet function by IP6K1.

PolyP has been shown to bind fibrinogen and is incorporated into the polymerised fibrin clot. To



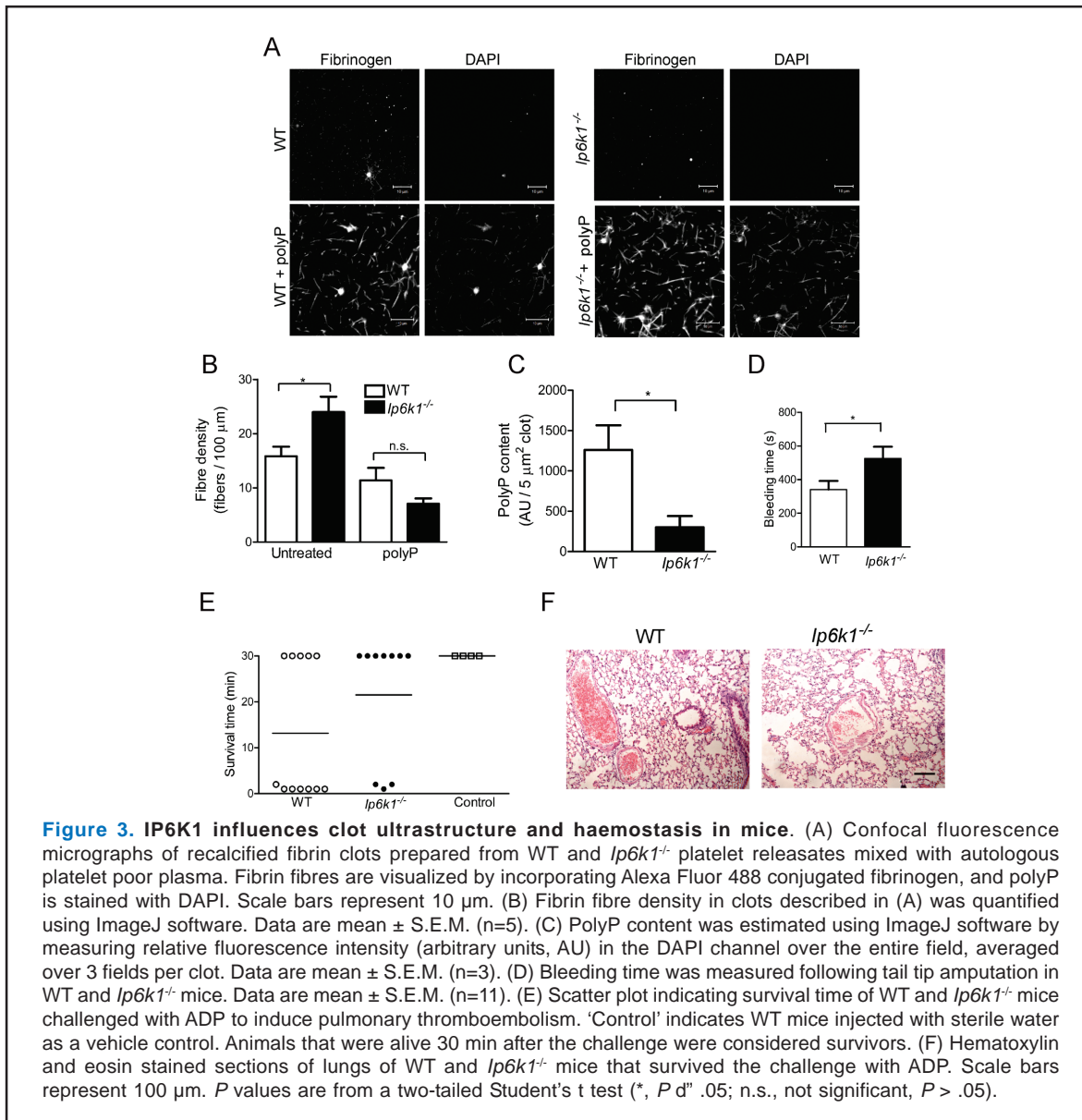
examine whether the reduction in platelet polyP levels in *Ip6k1*^{-/-} mice leads to altered clot architecture, we prepared clots by recalcifying a mixture of platelet poor plasma and clarified platelet releasate, spiked with fluorescently labelled fibrinogen, and stained these clots with DAPI to detect polyP. Clots examined by confocal microscopy revealed a homogenous web-like clot architecture in *Ip6k1*^{-/-} samples, whereas thicker fibrin fibrils, and tight fibrin aggregates interspersed with large pores were observed in WT clots (Fig. 3A). We quantified the extent of clot homogeneity by measuring the average fibre density, and observed a significant increase in the number of fibres per unit length in *Ip6k1*^{-/-} derived clots compared with WT (Fig. 3B). WT clots also stained positive for the presence of polyP, with maximal staining in the knots (Fig. 3A), whereas there was a four-fold reduction in DAPI staining of *Ip6k1*^{-/-} derived clots (Fig. 3C). Incorporation of polyP (average chain length 45) during clot formation in

WT and *Ip6k1*^{-/-} samples resulted in thickening of fibrils, an increased number of knots, and reduction in fibre density, thus eliminating the differences observed between WT and *Ip6k1*^{-/-} clots (Fig. 3A). It has been suggested that the loss of polyP contributes to bleeding diathesis observed in δ storage pool diseases. As *Ip6k1*^{-/-} mice exhibit a decrease in platelet polyP, they too are likely to display bleeding diathesis. We therefore determined the bleeding time of WT and *Ip6k1*^{-/-} mice by amputation of the tail tip. We note a significant lengthening of average bleeding time in *Ip6k1*^{-/-} compared with WT mice (Fig. 3D), reflecting the reduced aggregation of *Ip6k1*^{-/-} platelets we have observed *in vitro*, and supporting a role for polyP in primary haemostasis. To examine the effect of reduced polyP on secondary haemostasis *in vivo*, we used a pulmonary thrombosis model. WT and *Ip6k1*^{-/-} mice were challenged with a high dose of intravascular ADP to induce pulmonary thromboembolism, and animals were monitored for

respiratory pattern and survival. While fewer WT mice survived this challenge (Fig. 3E), those that lived longer than 30 minutes exhibited signs of severe respiratory distress and were immobile. In contrast, the majority of *Ip6k1*^{-/-} mice survived and were active following the challenge, displaying uniform breathing. Lung sections from WT and

Publications

1. Ghosh S, Shukla D, Suman K, Lakshmi BJ, Manorama R, Kumar S and Bhandari R (2013). Inositol hexakisphosphate kinase 1 maintains hemostasis in mice by regulating platelet polyphosphate levels. **Blood** 122: 1478-1486.



Ip6k1^{-/-} mice which survived the challenge revealed a higher degree of occlusion in large pulmonary vessels in WT compared with *Ip6k1*^{-/-} mice (Fig. 3F). Our results clearly indicate a role for IP6K1 in maintaining *in vivo* haemostasis by influencing platelet polyP levels. This project has now been completed and was published in the current reporting year.

2. Jadav RS, Chanduri MVL, Sengupta S and Bhandari R (2013). Inositol pyrophosphate synthesis by inositol hexakisphosphate kinase 1 is required for homologous recombination repair. **Journal of Biological Chemistry** 288: 3312-3321.

LABORATORY OF CHROMATIN BIOLOGY AND EPIGENETICS

Understanding the functions of Sirtuin family deacetylases in eukaryotic cell physiology

Faculty	Devyani Haldar	Staff Scientist
PhD Students	Lahiri Konada Raghavendra Vadla Amrita Sengupta Shalini Aricthota	Senior Research Fellow (Since Oct. 2013) Senior Research Fellow (Since Oct. 2013) Senior Research Fellow (Since Oct. 2013) Junior Research Fellow (Since Mar. 2014)
Other Members	Nirupama Chatterjee Ananga Ghosh	Technical Officer (Since Dec. 2013) Project-Junior Research Fellow (Since Jan. 2014)
Collaborators	Manojit Pal Shekhar C Mande	DRILS, Hyderabad NCCS, Pune

Objectives

- 1) Understanding the molecular functions of sirtuins family NAD⁺ dependent histone/protein deacetylases;
- 2) Screening to identify novel Sirtuin inhibitors and test if they act as anti-cancer agents.

Project 1: Understanding the molecular functions of sirtuin family NAD⁺ dependent histone/protein deacetylases.

Reversible acetylation/deacetylation of proteins regulates numerous important cellular processes. The Sirtuin family of protein/histone deacetylases (HDAC) are conserved enzymes that require NAD⁺ to deacetylate proteins. Sirtuins carry out a broad range of crucial cellular functions ranging from transcriptional silencing to DNA damage response, cell cycle regulation, metabolism and aging etc. Both budding yeast and fission yeast has been very effectively used as model system to understand biology of complex systems. Yeast genetics is a powerful tool which has been instrumental in discovering many novel genes and characterizing their functions in cellular signaling pathways. Since sirtuins are conserved from yeast to mammals, we use fission yeast *S. Pombe* as model systems to understand and elucidate the molecular functions sirtuins. 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 of 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 signaling pathways where Hst4 could be functioning.

To decipher novel functions of sirtuin family NAD⁺ dependent histone deacetylase Hst4 of fission yeast.

Summary of work done until the beginning of this reporting year (upto March 31, 2013)

We had previously reported that in fission yeast *S. Pombe* strains lacking sirtuin *hst4⁺*, acetylation of its substrate histone H3 lysine56 increases and S phase is prolonged. To decipher novel functions of Hst4, a slow growth and DNA damage sensitivity phenotype suppressor screen has been carried out. Among the suppressor genes identified by this screen are a few genes encoding proteins involved in DNA replication. These genetic interactions indicated that Hst4 may be involved in regulation of DNA replication. One among these is an accessory factor of DNA polymerase alpha. We have further validated and studied the interaction of *hst4* and this suppressor named Sup1. The polymerase alpha accessory factor (Sup1) has been cloned and over expressed in *S. pombe* and its ability to suppress the phenotypes of *hst4Δ* mutants has been confirmed earlier.

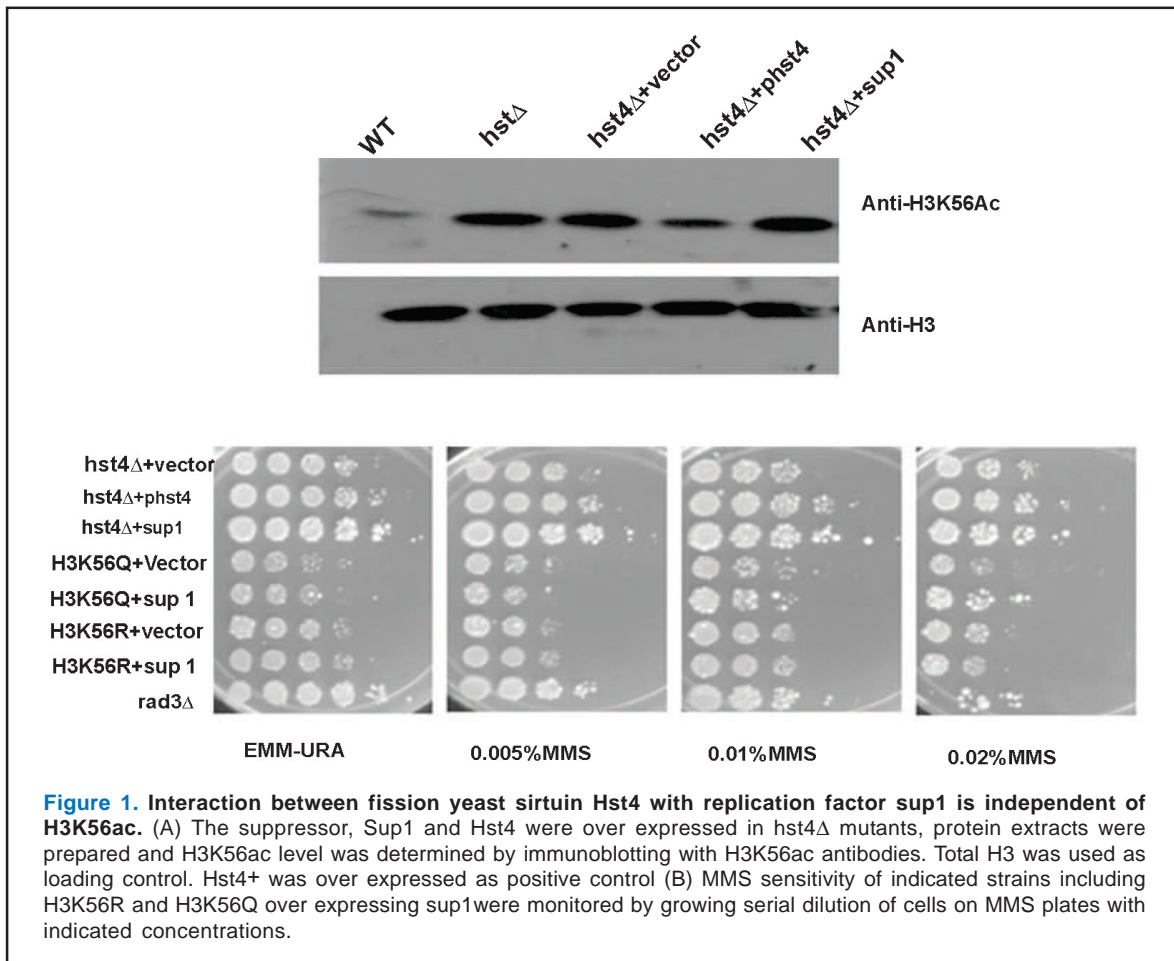
Details of progress made in the current reporting year (April 1, 2013 - March 31, 2014)

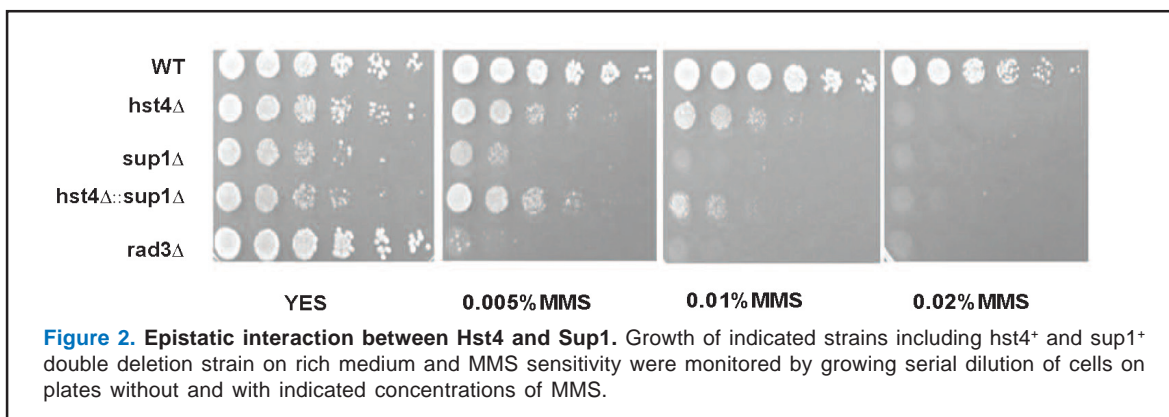
To understand the functions of Hst4 by studying mechanisms of Hst4 and suppressor interaction. There are several possible mechanisms of suppression. Therefore, to understand mechanism by which suppression happens, we are carrying out the following analysis:

i) The effects of *hst4*Δ mutants are mainly attributed to increased H3K56Ac levels. To check whether the suppressor, Sup1 is able to suppress *hst4*Δ phenotype through down regulation of H3K56Ac levels, Sup1 gene was over expressed in the *hst4*Δ mutants and level of H3K56ac was monitored by western blot using anti-H3K56ac antibodies. The H3K56ac levels remain unchanged on over expression of the suppressor gene indicating that the suppressor does not simply reduce H3K56ac levels by recruiting another deacetylase (Fig. 1A Upper Panel). We have earlier shown that the phenotypes of the H3K56R and H3K56Q mutants which mimic constitutive deacetylated and acetylated states respectively are similar to *hst4*Δ mutants. Therefore, to check if phenotypes of these mutants are recovered by the high copy suppressor Sup1, it was over expressed in H3K56R and H3K56Q strains and recovery of slow growth and MMS sensitivity phenotypes were monitored by spotting of serial dilutions of these strains on plates with and without

MMS (Fig. 1B Lower Panel). Sup1 expression could not suppress the phenotypes of these mutants. These results show that the suppressor acts through H3K56ac independent pathway.

ii) Genetic interactions between *sup1* and *hst4*: The *hst4*Δ mutants show defects in growth, elongated morphology and sensitive to DNA damaging agents similar to that of Sup1Δ mutants. To test whether Hst4 and *sup1* interact epistatically or exhibit synthetic lethality, the individual *hst4*Δ mutant and *sup1*Δ were crossed to generate a double mutant. The genetic interaction was tested by growing serial dilution of cells on normal and MMS containing medium (Fig. 2). The results shown in Figure 2 suggests that the double deletion mutants were viable and showed growth rate and MMS sensitivity similar to that of *hst4*Δ mutants. These results show that *sup1* might act in the same pathway downstream of Hst4. Since the suppressor functions in DNA replication, we are planning to investigate potential function of Hst4 in DNA replication.





iii) Co-immunoprecipitation to test if the suppressor protein and Hst4 protein interact physically: To test if the two proteins interact physically, we are currently generating yeast strains expressing epitope tagged Hst4 and *sup1* to perform Co-immunoprecipitation experiments using tag antibodies.

Project 2: A yeast based screen for discovery of novel Sirtuin inhibitors as anti-cancer agents.

Epigenetic therapeutics of cancer such as inhibitors of DNA methyltransferases and histone deacetylases (class I and class II) are already being used in combination with the standard cytotoxics with encouraging results. The Sirtuins (class III NAD-dependent deacetylases) are being considered as important targets for cancer therapeutics as they are up-regulated in many cancers. Inhibition of sirtuins allows re-expression of silenced tumor suppressor genes, leading to reduced growth of cancer cells. However, no sirtuin inhibitors have entered into the clinic yet as an anticancer agent. We would like to identify novel small molecule inhibitors of Sirtuins and characterize their potential as anti-cancer agents using budding yeast, *S. cerevisiae* as model system for compound screening.

Summary of work done until the beginning of this reporting year (upto March 31, 2013)

For screening of compounds with sirtuin inhibitory activity, we have used a yeast (*S. cerevisiae*) strains having the URA3 reporter gene integrated at the silent telomeric locus (Tel::URA3 strain). A reporter silencing assay is based on the ability of yeast Sir2 to keep the URA3 gene silent at telomeric locus and its inhibitor makes it active. The yeast strain which express URA3 will not grow in presence of 5'-fluoroorotic acid (FOA). We have

performed the assay and monitored growth of these strains in liquid medium in 96 well plates, without and with FOA. A known Sirtuin inhibitor splitomycin was used as a reference compound. Totally 361 compounds of different chemical classes were explored by following rational drug design and unbiased approaches and subsequently synthesized. These were tested for Sir2 activity inhibition using this yeast cell based URA3 reporter silencing assay. Several hit compounds were identified.

Details of progress made in the current reporting year (April 1, 2013 - March 31, 2014)

The identified hit compounds were tested for their ability to inhibit NAD-dependent HDAC activity of recombinant human sirtuins, hSIRT1 and hSIRT2 *in vitro* using HDAC fluorescent activity assay. The *SIRT Fluorescent Activity Assays* are based on the unique *Fluor de Lys-SIRT* Substrate/Developer II combination. The *Fluor de Lys-SIRT1* substrate is a unique peptide. In this assay, fluorescence signal is generated in proportion to the amount of deacetylation of the lysine, by Sirtuins and plotted percentage inhibition was calculated (Figs. 3B and 3C). One of the potent hit compound, ALN-184 was found to inhibit both hSIRT1 and hSIRT2.

The effect of treatment of ALN-184 on cell proliferation/viability was determined by MTT assay in several cell lines including HeLa, HEPG2, A549 and it was found to be cytotoxic to HEPG2 cells. Its IC₅₀ for HEPG2 cells was 15.3 μM (Fig. 3D). The Figure 3E shows that untreated cells show a triangular morphology and are well adhered and the growth of the treated cells is inhibited and rounded morphology was observed and the cells are released into media. We are currently, checking whether treatment causes apoptosis of HEPG2 cells.

Discovery of novel sirtuins inhibitor would facilitate design and development of novel anti-cancer therapeutics. In addition, deciphering molecular

mechanisms involved in eliciting the anti-cancer effect will shed substantial light on the role of sirtuins in cancer initiation and progression.

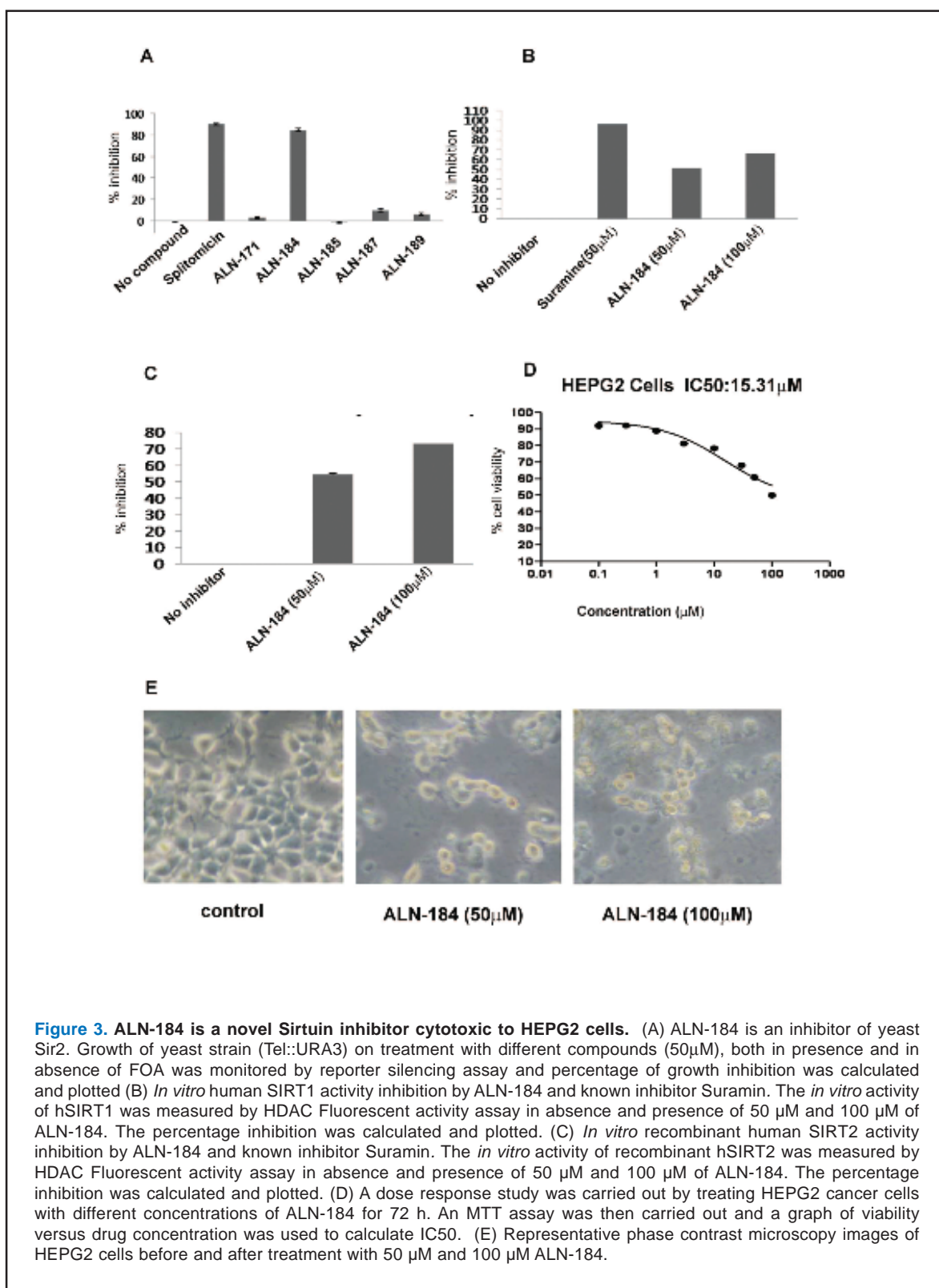


Figure 3. ALN-184 is a novel Sirtuin inhibitor cytotoxic to HEPG2 cells. (A) ALN-184 is an inhibitor of yeast Sir2. Growth of yeast strain (Tel::URA3) on treatment with different compounds (50 μ M), both in presence and in absence of FOA was monitored by reporter silencing assay and percentage of growth inhibition was calculated and plotted (B) *In vitro* human SIRT1 activity inhibition by ALN-184 and known inhibitor Suramin. The *in vitro* activity of hSIRT1 was measured by HDAC Fluorescent activity assay in absence and presence of 50 μ M and 100 μ M of ALN-184. The percentage inhibition was calculated and plotted. (C) *In vitro* recombinant human SIRT2 activity inhibition by ALN-184 and known inhibitor Suramin. The *in vitro* activity of recombinant hSIRT2 was measured by HDAC Fluorescent activity assay in absence and presence of 50 μ M and 100 μ M of ALN-184. The percentage inhibition was calculated and plotted. (D) A dose response study was carried out by treating HEPG2 cancer cells with different concentrations of ALN-184 for 72 h. An MTT assay was then carried out and a graph of viability versus drug concentration was used to calculate IC₅₀. (E) Representative phase contrast microscopy images of HEPG2 cells before and after treatment with 50 μ M and 100 μ M ALN-184.

LABORATORY OF COMPUTATIONAL BIOLOGY

Computational Studies on Protein Structure, Function and Interactions

Faculty	HA Nagarajaram	Staff Scientist
PhD Students	Anupam Sinha	Senior Research Fellow
	H Rachita	Senior Research Fellow
	Manjari K	Senior Research Fellow
	Suryanarayana Seera	Senior Research Fellow
	VA Ramesh	Junior Research Fellow
	Rakesh Trivedi	Junior Research Fellow

Objectives

Studies on protein-protein interaction networks (PPIN):

- Structural and functional characterization of central nodes in human PPIN;
- Studies on spatio-temporal dynamics of human PPIN; and
- Analysis of Human-Virus PPI (HU-Vir PPI) network

Summary of work done until the beginning of this reporting year (upto March 31, 2013)

1. Studies on hubs in human PPIN:

We examined the relation between the degree and the number of splice variants of nodes in human as well as other eukaryotic protein-protein interaction networks and found that on average, highly connected nodes (hubs) have greater number of splice variants than the non-hubs.

2. Studies on tissue-specific human PPINs:

We constructed and analyzed 70 different tissue-specific PPI interaction networks. Our studies enabled us to introduce into literature a novel dichotomy of hubs referred to as Local hubs (also referred to as tissue-specific hubs) and Global hubs (also referred to as house-keeping hubs). Each class of hubs exhibit distinct sequence, structural and functional properties.

3. Studies on human-virus PPIs:

We constructed and analyzed Bridged Hu-Vir PPI Network (BHVN) for different viruses. We were able to identify some viral proteins connecting unconnected components in human PPI network and hence act as Articulation points. These viral articulation points were found to be conserved among related viruses. Functional annotation

studies showed that viruses connect metabolic pathways to PPI network and hence seem to take over the regulation of metabolic pathways.

Details of progress made in the current reporting year (April 1, 2013-March 31, 2014)

Project 1: Structural and functional characterization of central nodes in human PPI network

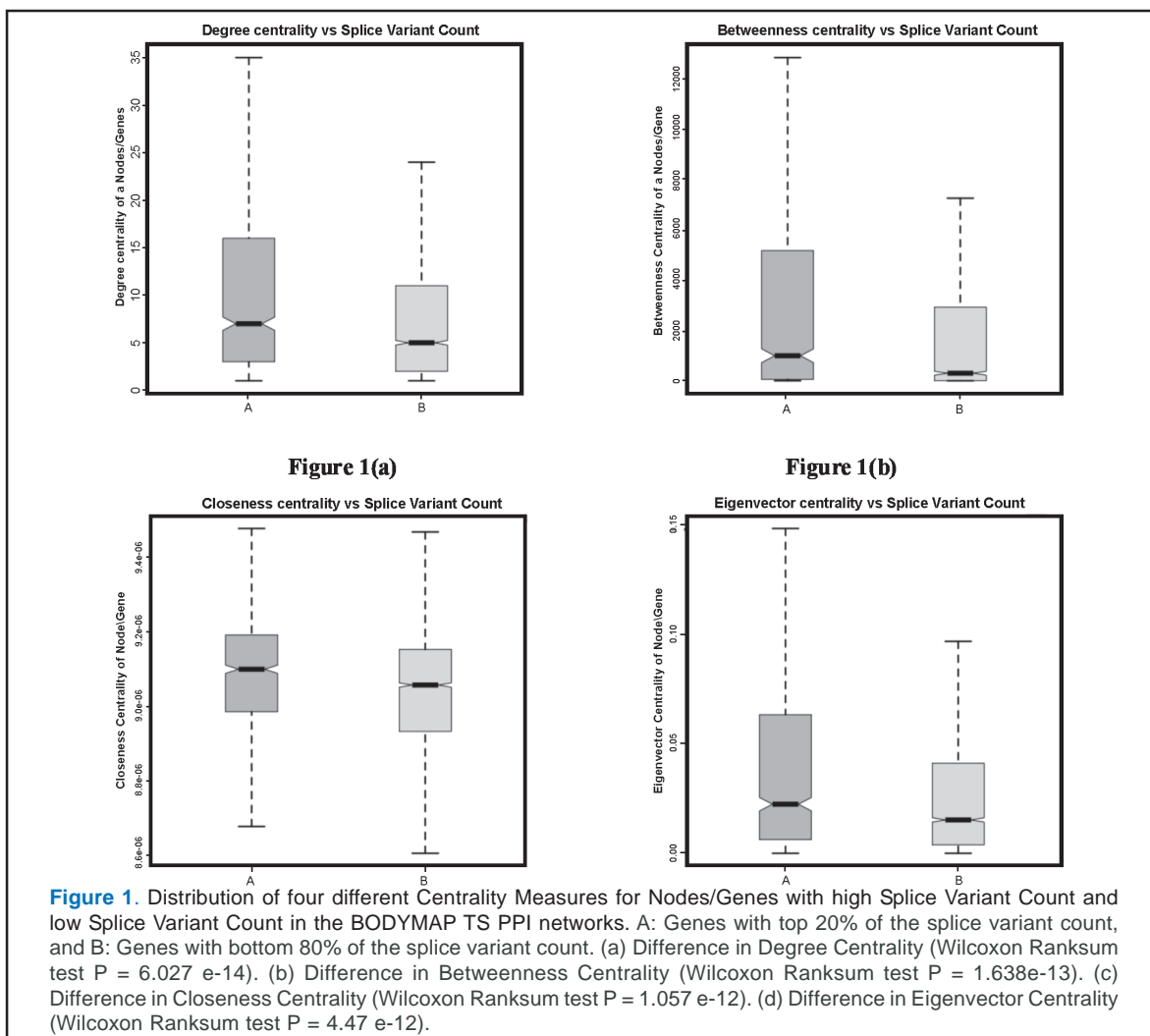
- We extended our analysis to the Tissue Specific Protein-Protein Interaction (TS PPI) networks where we considered 16 different TS PPI networks. These networks were constructed by mapping known tissue-wise RNA-sequencing data on to known PPIs.
- We investigated the distribution of the four different centrality measures viz., degree, betweenness, closeness and eigenvector in the nodes with many splice variants (genes with top 20% of the splice variant count) and in the nodes with fewer splice variants (genes with bottom 80% of the splice variant count) in the TS PPI networks and found that the genes/nodes enriched with splice variants, as compared with the genes with low number of splice variants tend to show higher values of centrality measures (Fig. 1(a)).
- We formed domain-domain interaction (DDI) by integrating relevant data from iPfam with TS PPI and calculated the propensity of interactions of each of the interacting domains. We found that the nodes with high number of splice variants have greater number of promiscuous domains (>5 interactions) (Fig. 2). We surmise that the presence of these promiscuous domains across multiple variants of a gene/node significantly enhances its centrality measures in a PPI network.

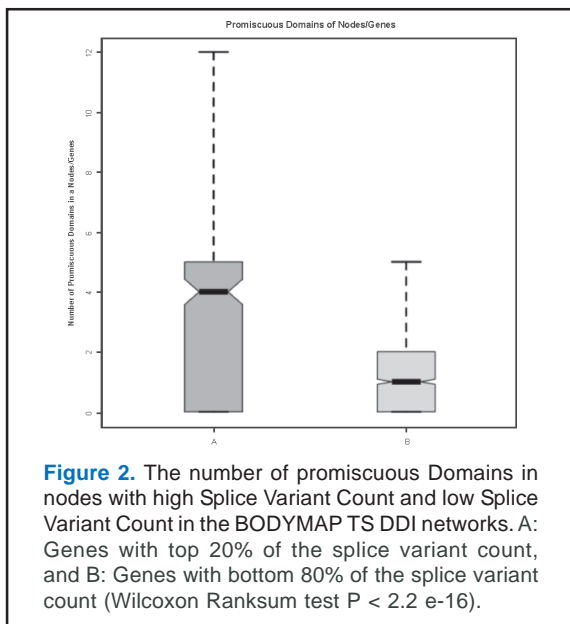
Project 2: Studies on spatio-temporal dynamics of human PPI networks

1. We continued our studies on local and global hubs in human tissue-specific PPI networks by investigating their interaction and localization diversities.
2. We calculated conservation of partners of local and global hubs across the tissues they are expressed and found that local hubs conserve their partners across all the tissues they are expressed whereas global hubs interact with diverse partners in diverse tissues.
3. We also found that the partners of global hubs occupy more diverse sub-cellular localizations than the partners of local hubs.
4. Our investigations also revealed that both local and global hubs comprise of the hubs that are intramodular in nature (akin to party hubs) and the hubs that are intermodular in nature (akin to date hubs).

Project 3: Analysis of human and virus protein-protein interaction (Hu-Vir PPI) networks

1. We investigated various properties of the human proteins (referred to as hVIPs) targeted by viral proteins.
2. We found that hVIPs are significantly enriched in disordered regions, expressed in more number of tissues and also show high centrality measures (including the new metric introduced by us called pathway centrality) than non-hVIPs. We calculated the localization diversity (LD) of hVIPs and non-hVIPs and found that the former has higher LD values than non-hVIPs suggesting that human partners of viral proteins are wide spread in cell. hVIPs were found to be evolving at slower rates than non-hVIPs. To summarize, our investigations revealed that viral proteins tend to interact with





human proteins that are essential, abundantly expressed as well as slow evolving (Fig. 3).

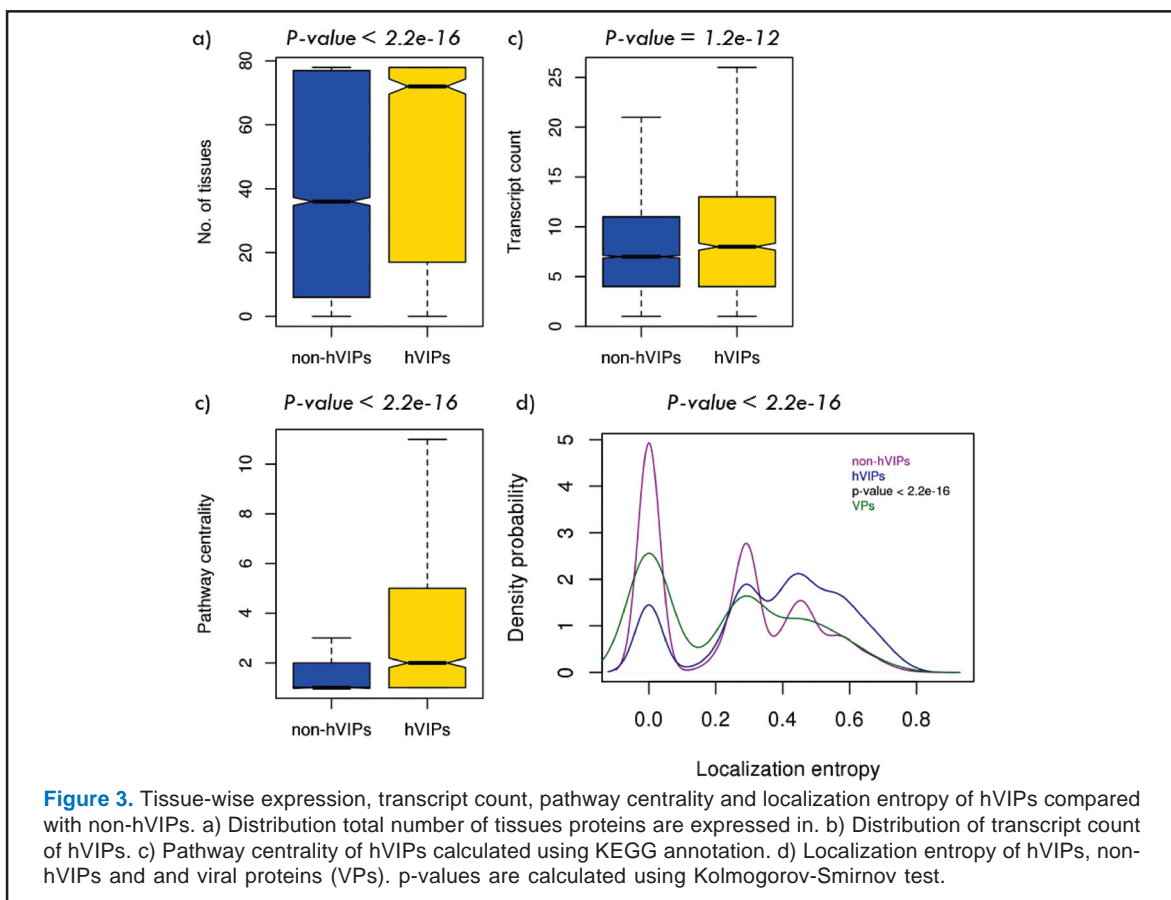
Future plans and directions

1. Integration and analysis of human nsSNP data on protein-protein interaction networks

2. Further analysis of viral-human bridge PPI network
3. Studies on tissue-wise PPI networks integrated with drug-protein interaction data
4. Further studies on structural and functional characterization of centrally important nodes in HPPIN.

Publications

1. Acharya V and Nagarajaram HA (2013). Response to: Statistical analysis of missense mutation classifiers. *Human Mutation* 34: 407.
2. Bashyam MD, Chaudhary AK, Kiran M, Reddy V, Nagarajaram HA, Dalal A, Bashyam L, Suri D, Gupta A, Gupta N, Kabra M, Puri RD, RamaDevi R, Kapoor S and Danda S (2013). Molecular analyses of novel *ASAH1* mutations causing Farber lipogranulomatosis: analyses of exonic splicing enhancer inactivating mutation. *Clinical Genetics* Nov 8. doi:10.1111/cge.12316.
3. Kiran M and Nagarajaram HA (2013). Global versus local hubs in human protein-protein interaction network. *Journal of Proteome Research* 12: 5436–5446.



4. Sinha A and Nagarajaram HA (2013). Effect of alternative splicing on the degree centrality of nodes in protein-protein interaction networks of *Homo sapiens*. ***Journal of Proteome Research*** 12: 1980–1988.
5. Bashyam MD, Chaudhary AK, Kiran M, Nagarajaram HA, Devi RR, Ranganath P, Dalal A, Bashyam L, Gupta N, Kabra M, Muranjan M, Puri RD, Verma IC Nampoothuri S and Kadamdale SS(2014). Splice, insertion-deletion and nonsense mutations that perturb the phenylalanine hydroxylase transcript cause phenylketonuria in India. ***Journal of Cellular Biochemistry*** 115: 566-574.
6. Mudunuri SB, Patnana S and Nagarajaram HA (2014). MICdb3.0: a comprehensive resource of microsatellite repeats from prokaryotic genomes ***Database*** doi:10.1093/database/bau005.
7. Anusha U, Ranganath P, Md Nurul Jain SJ, Krishna Prasad C, Sinha A, Verma IC, Phadke SR, Puri RD, Danda S, Muranjan MN, Jevalikar G, Nagarajaram HA and Dalal AB. Novel mutations of the ARSB gene in Indian patients with Mucopolysaccharidosis Type VI. ***Indian Journal of Medical Research*** (In press).

LABORATORY OF COMPUTATIONAL AND FUNCTIONAL GENOMICS

Computational and functional genomics of microbial pathogens

Faculty	Akash Ranjan	Staff Scientist
PhD Students	Rohan Misra	Senior Research Fellow
	Bhavik Sawhney	Senior Research Fellow
	Ajit Roy	Senior Research Fellow
	Suhail Yousuf	Senior Research Fellow
	Rajendra Kumar Angara	Senior Research Fellow
	Abhishek Kumar	Senior Research Fellow
	Debasish K Ghosh	Junior Research Fellow
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	Jamshaid Ali	Project-Junior Research Fellow (Till Jul. 2013)
	G Srujana	Project-Junior Research Fellow (Till May 2013)
	N Saraswathi	Project-Junior Research Fellow
Collaborators	Regine Hengge	Freie University, Germany
	Lothar H Wieler	Freie University, Germany
	M Sritharan	UoH, Hyderabad

Project 1: Genome analysis and functional characterization of regulons in microbial genomes.

Coordinating gene expression is essential to bring all the proteins together at a specific time to mediate complex physiological processes. Regulon play an important role in coordinating gene expression. We are interested in understanding how physiological processes are coordinated by regulons in microbial genomes like mycobacteria and *E. coli*.

Objectives

1. Characterization of regulons in mycobacteria

- Locating IdeR and HupB box in transcription control region of *mbtB*;
- Transcription control of *rv0494* and Rv0494 regulon; and
- Rv2989 regulon: Transcription control of *leuC* and *rv2989*

2. Characterization of regulons in *E. coli*

- HosA regulon: Structure and functional characterization of *hosA* gene and its protein product

Summary of the work done until the beginning of this reporting year (upto March 31, 2013)

Rv0494 was expressed as a recombinant protein in *E.coli* BL21. Recombinant Rv0494 was shown to exist as dimer in solution. The binding site of Rv0494 is localized near the start codon in the form of a 5 bp long palindrome and was confirmed using electrophoretic mobility shift assay. Similar sites were searched throughout the whole genome of *M. tuberculosis* and these sites were verified for their interaction with Rv0494, we have also shown that long chain fatty acyl coenzyme A molecules inhibit the protein-DNA interaction, however no effect on this interaction was observed in case of short and medium chain fatty acyl coenzyme A.

Working with *hosA*, we reported cloning of *hosA* gene in pET21b (+) vector and its expression in *E.coli* BL21. The HosA protein was purified using Ni-NTA affinity chromatography. The purity of the protein was checked by 12% SDS-PAGE and has been shown to form dimer through gel filtration chromatography using superdex 75 column.

Details of progress made in the current reporting year (April 1, 2013 - March 31, 2014)

Working with IdeR and HuB we show that both the proteins bind to upstream region of *mbtB* gene,

P_{mbtB} (Figs. 1A & B). Using DNAase I footprinting we located the IdeR and HupB box around P_{mbtB} (Figs. 2A, B & C).

Working with GntR/FadR-Rv0494, we show that rv0494 gene expression is enhanced during starvation (Fig. 3A). There are two separate promoters driving the expression of Rv0494 in a condition dependent manner. Two different sigma factors sigA and sigC were shown to interact with the upstream region of Rv0494.

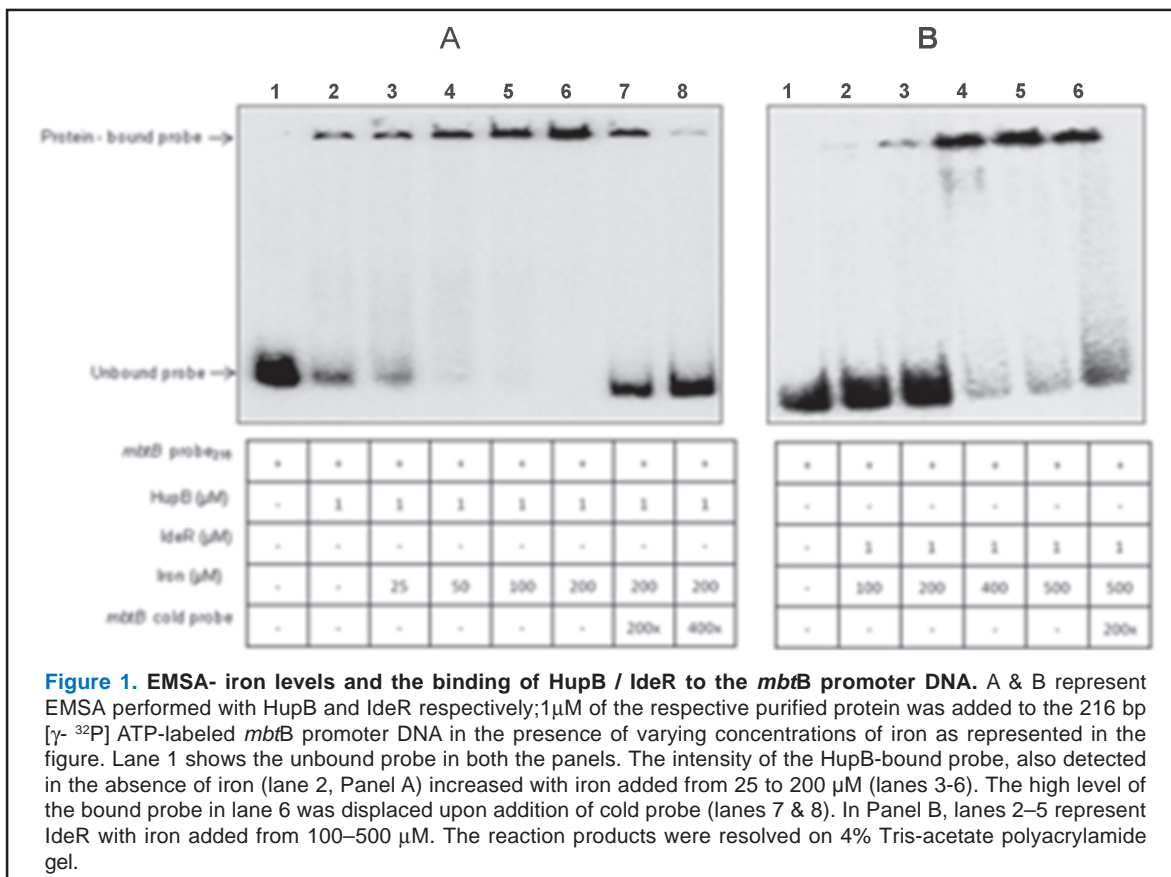
Working with Rv2989, an IclR like regulator in *M.tuberculosis*, we show that its transcription is controlled with promoters located around a 72 bp intergenic region between *leuC* and *rv2989* (Fig. 4). Using primer extension and bioinformatics analysis we identified potential promoters located around the region (Figs. 5A & B). Using a promoter-report construct we show that mutations in identified promoter leads to loss of its activity (Figs. 6A & B).

Working with HosA, we have expressed the protein as recombinant protein in *E.coli*. The recombinant protein was used to search suitable conditions for crystallization. The crystallised protein for X-ray

crystallography is shown in Figure 7A. A preliminary X-ray diffraction of Hos A crystals are shown in Figure 7B. Using the recombinant protein, HosA, and the upstream transcription control region P_{ubix} , we show that HosA specifically binds to P_{ubix} and shows mobility shift in EMSA (Fig. 8)

Project 2: Genome analysis and functional characterization of *Plasmodium falciparum*.

Acyl-CoA Binding Proteins (ACBPs) are low to medium molecular weight (MW: 10-55 kDa) proteins which are relatively well conserved in eukaryotic organisms. These proteins are known to play important role in regulation of intracellular acyl-CoA pool size, transport of acyl-CoA for beta-oxidation, vesicular trafficking, complex lipid biosynthesis, and gene regulation. Although fatty acids are essential molecules, their entry into metabolic pathways are restricted. Fatty acids are activated by thioesterification with Coenzyme-A (Co-A) to form Acyl-CoA esters. Activated fatty acids either undergo metabolic utilization, or get stored as complex with ACBPs. Among all the isoforms of ACBPs, structure of one of the isoform from *P. falciparum* is solved using X-ray crystallography



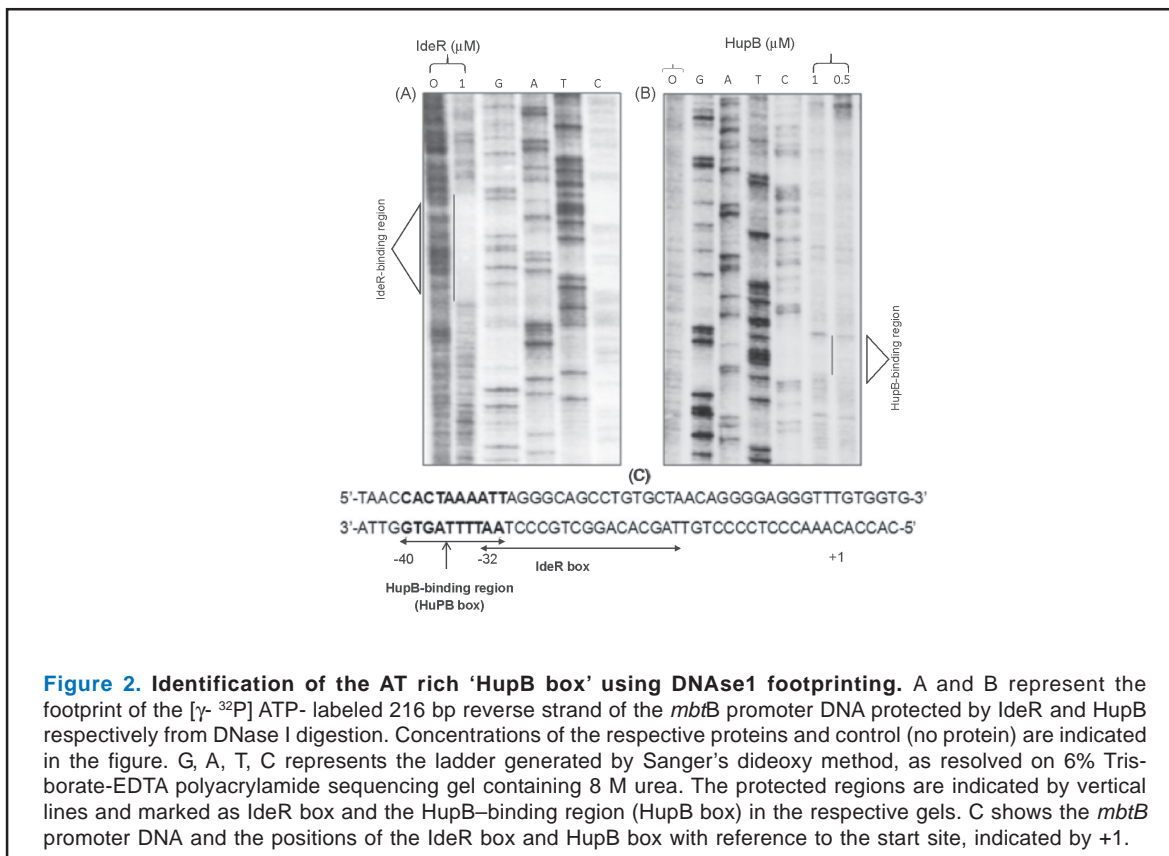


Figure 2. Identification of the AT rich 'HupB box' using DNase1 footprinting. A and B represent the footprint of the [γ - 32 P] ATP- labeled 216 bp reverse strand of the *mbtB* promoter DNA protected by IdeR and HupB respectively from DNase I digestion. Concentrations of the respective proteins and control (no protein) are indicated in the figure. G, A, T, C represents the ladder generated by Sanger's dideoxy method, as resolved on 6% Tris-borate-EDTA polyacrylamide sequencing gel containing 8 M urea. The protected regions are indicated by vertical lines and marked as IdeR box and the HupB-binding region (HupB box) in the respective gels. C shows the *mbtB* promoter DNA and the positions of the IdeR box and HupB box with reference to the start site, indicated by +1.

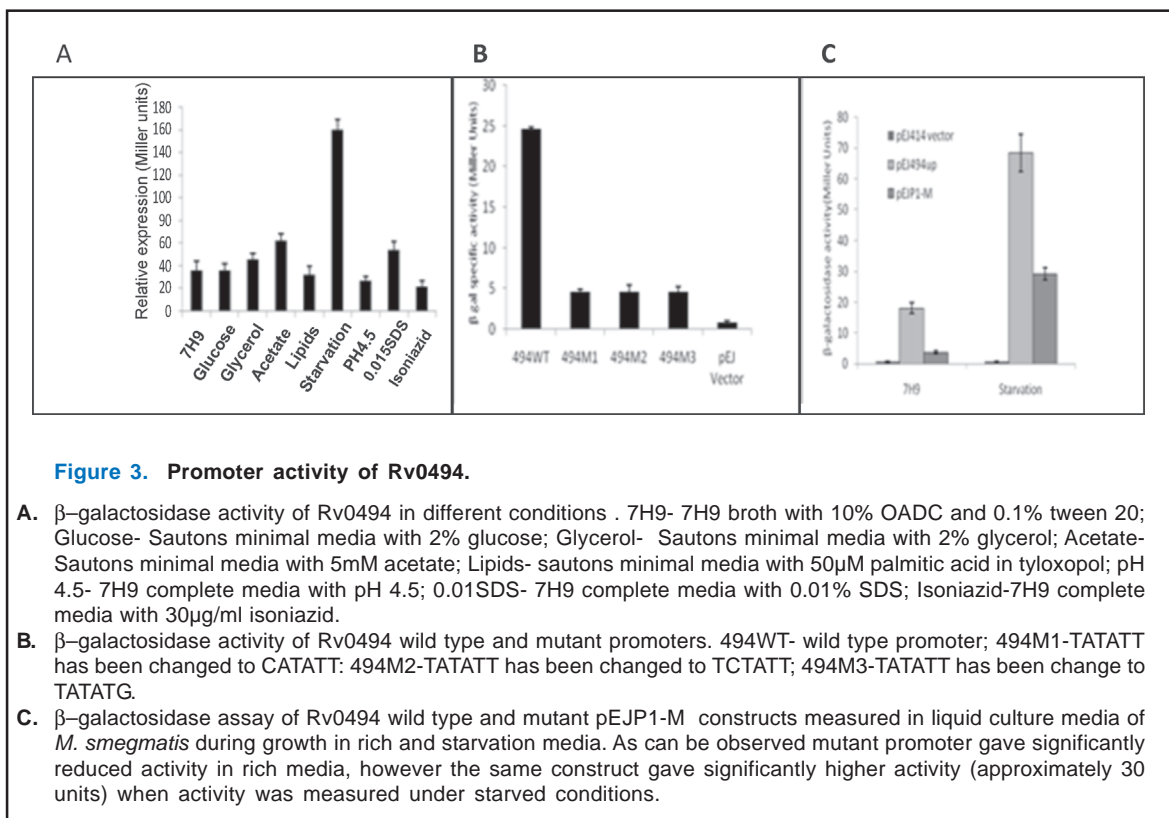


Figure 3. Promoter activity of Rv0494.

- β -galactosidase activity of Rv0494 in different conditions . 7H9- 7H9 broth with 10% OADC and 0.1% tween 20; Glucose- Sautons minimal media with 2% glucose; Glycerol- Sautons minimal media with 2% glycerol; Acetate- Sautons minimal media with 5mM acetate; Lipids- sautons minimal media with 50 μ M palmitic acid in tyloxopol; pH 4.5- 7H9 complete media with pH 4.5; 0.015SDS- 7H9 complete media with 0.01% SDS; Isoniazid-7H9 complete media with 30 μ g/ml isoniazid.
- β -galactosidase activity of Rv0494 wild type and mutant promoters. 494WT- wild type promoter; 494M1-TATATT has been changed to CATATT; 494M2-TATATT has been changed to TCTATT; 494M3-TATATT has been change to TATATG.
- β -galactosidase assay of Rv0494 wild type and mutant pEJ1-M constructs measured in liquid culture media of *M. smegmatis* during growth in rich and starvation media. As can be observed mutant promoter gave significantly reduced activity in rich media, however the same construct gave significantly higher activity (approximately 30 units) when activity was measured under starved conditions.

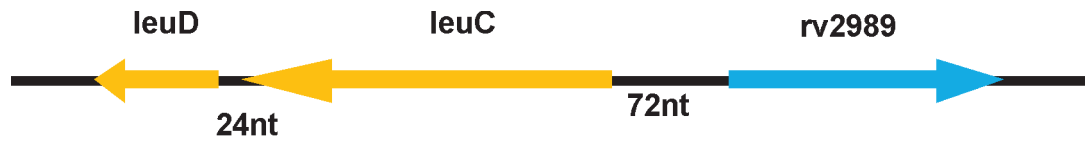


Figure 4. Genetic organization of *leuCD* operon and *rv2989*

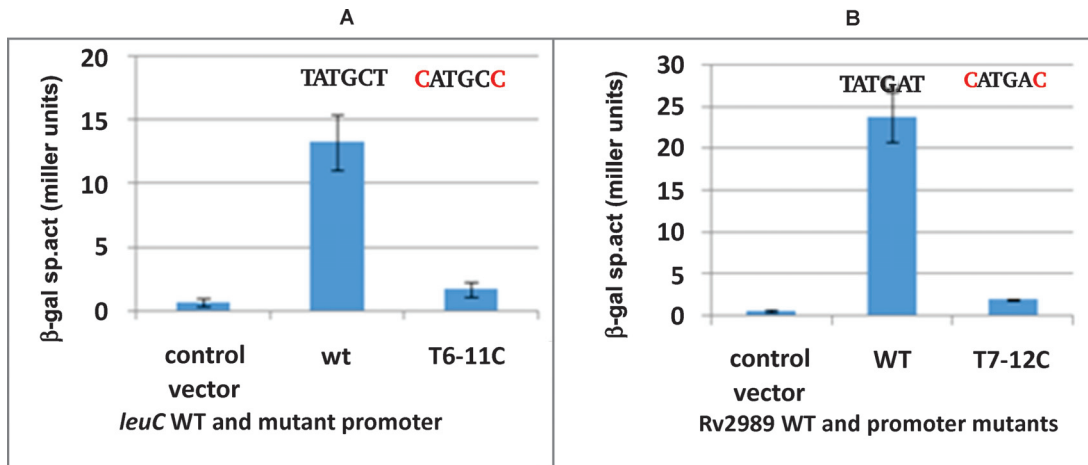


Figure 5. Promoter activity at *leuC-rv2989* locus. A. Activity of P_{leuC} . B. Activity of P_{rv2989} .

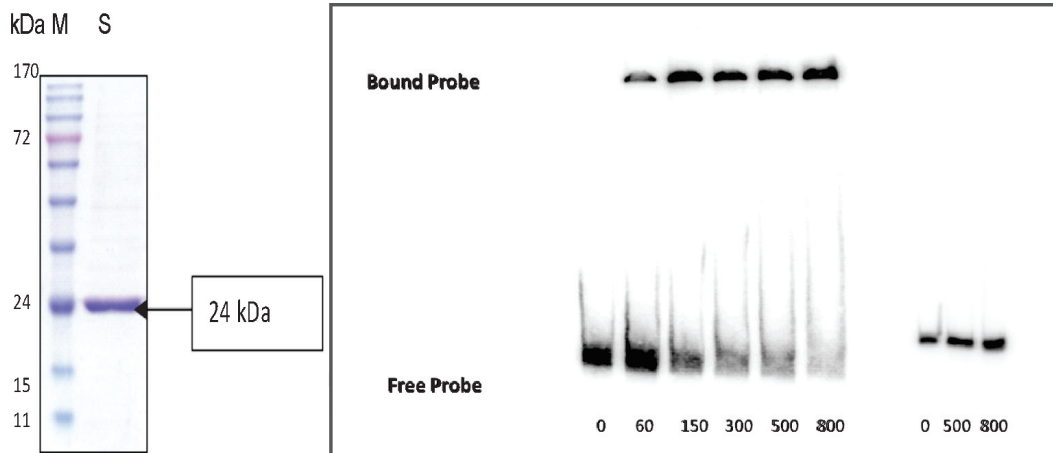


Figure 6. Rv2989 binds to the transcription control region *leuC-rv2989* locus. A. SDS PAGE analysis of purified recombinant Rv2989 protein. B. Electrophoretic mobility shift assay (EMSA) of *leuC-rv2989* locus and Rv2989 protein

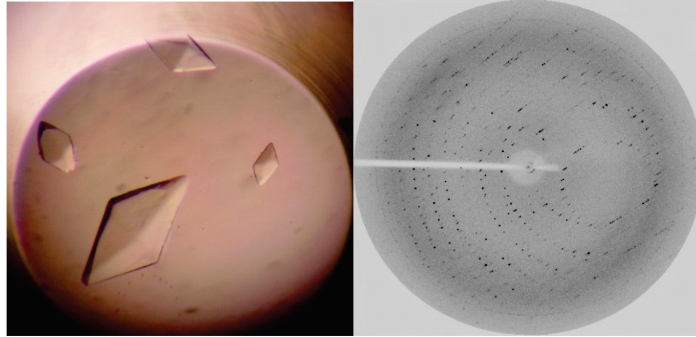


Figure 7. Crystallisation and preliminary X-ray diffraction of HosA. Protein crystals of HosA. B. Preliminary X-ray diffraction of HosA Protein crystals.

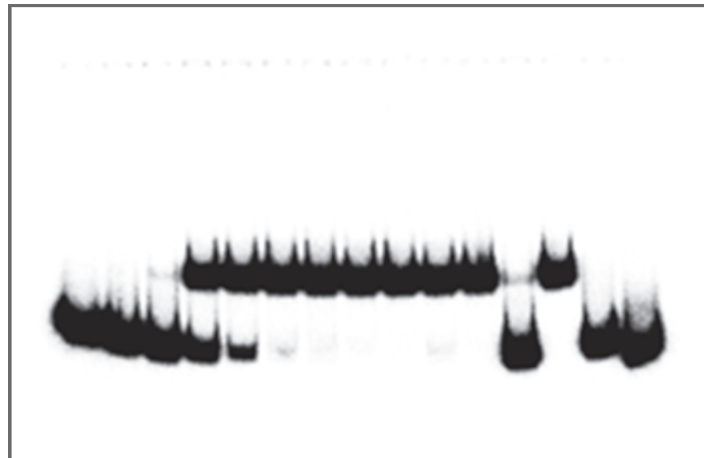


Figure 8. HosA binds to P_{ubiX} transcription control region. Electrophoretic mobility shift assay (EMSA) of P_{ubiX} with HosA protein (Lane1: Free probe, Lane 2: 10nM of HosA, Lane3: 20nM of HosA, Lane4: 30nM of HosA, Lane5: 40nM of HosA, Lane6: 50nM of HosA, Lane7: 60nM of HosA, Lane8: 70nM of HosA, Lane 9: 80nM of HosA, Lane10: 90nM of HosA, Lane 11: 100nM of HosA, Lane 12: 100nM of HosA with 50 fold cold P_{hosA} probe, Lane 13:100nM of HosA with 50 fold nonspecific cold probe, Lane14: 100nM of nonspecific protein (MarR homologue EmrR), Lane 15: Radiolabelled nonspecific probe (i.e. P_{slyA} promoter) with 100nM of HosA.

but we are yet to understand complete functional relevance of ACBPs in lower eukaryotic organisms like plasmodia. ACBP have been shown to be essential proteins for *Trypanosoma brucei* in its host, it would be interesting to study its role in biology of *P. falciparum*.

Objectives

- Comparative analysis and functional studies of *P. falciparum* ACBPs

Summary of the work done until the beginning of this reporting year (upto March 31, 2013)

We have done comparative analysis of Pf ACBPs and human ACBPs. We have expressed all the four ACBPs as histidine-tagged recombinant proteins. The maximum expression of ACBPs occurs at merozoite in which these lipid changes are observed. In order to know whether expression of PfACBP has anything to do with lipid changes in merozoite stage or not, we started *Plasmodium falciparum* culture. Further, a hydrophathy index plot for the protein ACBP 15 (PF3D7_1001100.1) using

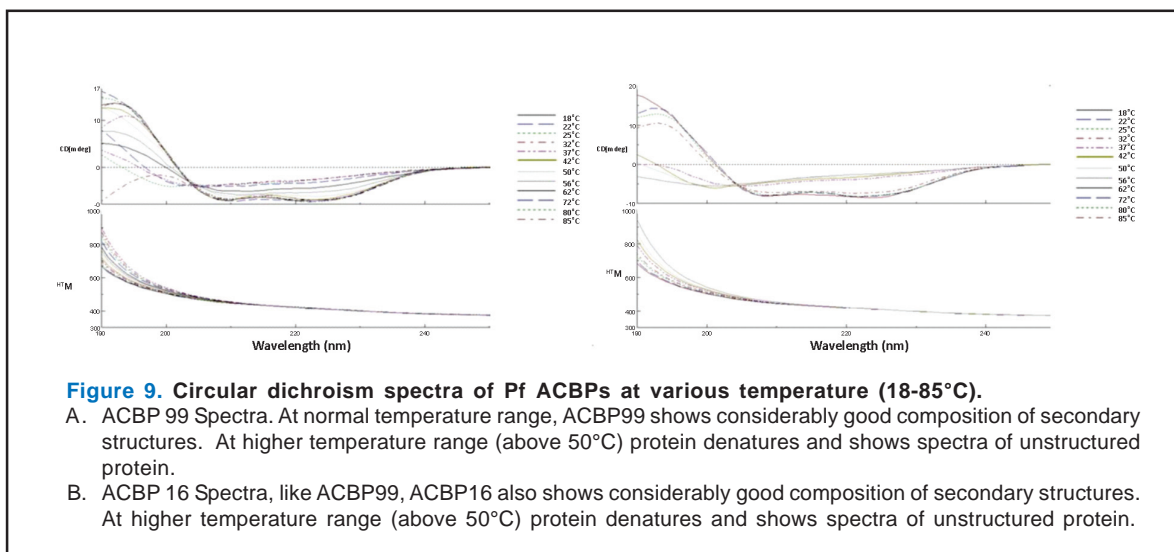
Prot Scale analysis showed that ACBP15 (PF3D7_1001100.1), is significantly differs from others.

Details of progress made in the current reporting year (April 1, 2013 - March 31, 2014)

We have expressed two Pf ACBPs -ACBP 99 (PF3D7_0810000) and ACBP 16 (PF3D7_1001200) as recombinant protein to compare them using Circular dichroism at a range of temperature (18-85°C). Figure 9 shows the CD spectra of the two proteins.

Publications

1. Muley VY and Ranjan A (2013). Evaluation of physical and functional protein-protein interaction prediction methods for detecting biological pathways. *PLoS One* 8: e54325.
2. Pandey SD, Choudhury M, Yousuf S, Wheeler PR, Gordon SV, Ranjan A and Sritharan M. Iron-regulated protein HupB of *Mycobacterium tuberculosis* positively regulates siderophore biosynthesis and is essential for growth in macrophages. *Journal of Bacteriology* (In press).



LABORATORY OF DROSOPHILA NEURAL DEVELOPMENT

Understanding patterning and development of Central Nervous System using
Drosophila melanogaster

Faculty	Rohit Joshi	WT-DBT India Alliance Intermediate Fellow
PhD Students	Risha Khandelwal Neha Ghosh Ravi Ranjan Rashmi Sipani Raza Alikhan	Senior Research Fellow Senior Research Fellow Junior Research Fellow Junior Research Fellow Technical Officer (Since Jul. 2012)
Other Members	P Kalyani Karnika Tripathi C Maheshvari Sromana Mukherjee	Technical Officer Project Assistant (Till Feb. 2014) Project Assistant (Since Jan. 2014) Project Assistant (Since Jan. 2014)

Objectives

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 (as represented in the Fig. 1). Hox family of transcription factors are known to play an important role in giving the positional identity to the cells and generation of a variety of cell types along the AP axis of the CNS during development. The molecular basis of this phenomenon is not well investigated. We are interested in understanding the molecular basis of Hox function in patterning CNS using *Drosophila melanogaster* as our model organism, focusing mainly on early embryonic and late 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 a less number of neurons compared to its thoracic counterpart. 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 precise molecular details of how *Abd-A* cause Nb apoptosis are unknown. Genetic evidence suggests a role for a helix-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. Moreover since Grh

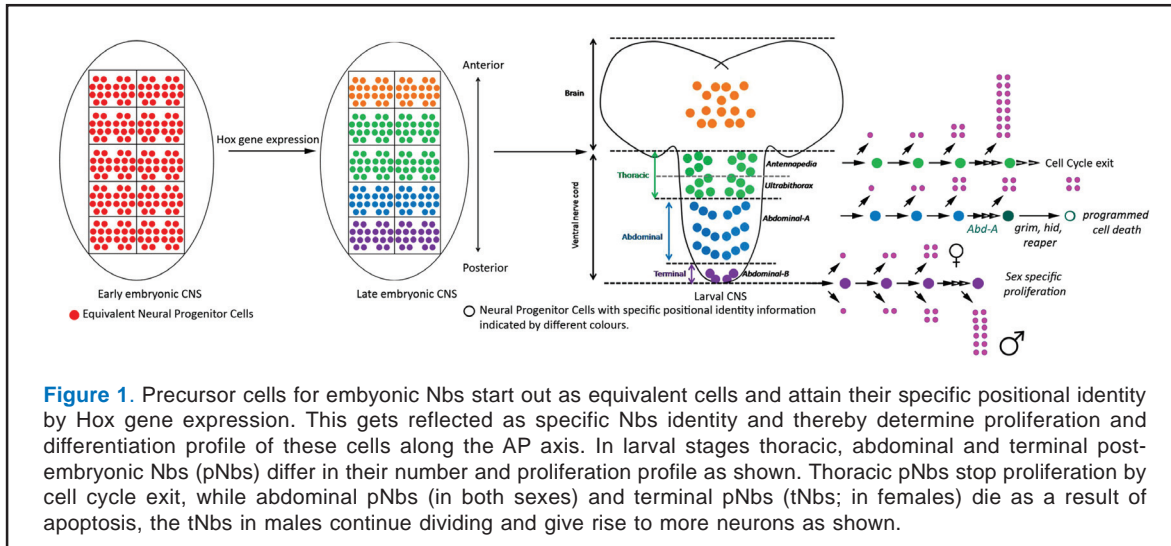
is involved in Nb apoptosis and is not expressed in neuronal progeny refractory to this apoptosis, it is of interest to define *grh* regulation in these cells which keeps *grh* "on" in the pNbs and "off" in the neuronal progeny of pNbs.

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

Hox genes express in CNS (in neural progenitor cells) in embryonic stages of development (as represented in Fig. 1) but how does their expression patterns the embryonic nervous system is not well understood. *Deformed (Dfd)* is known to express in the cells of subesophageal ganglion of embryonic CNS, this project focuses on understanding auto-regulation of *Dfd* in this region and find out how this helps in giving cells their specific positional identity. This is being done by using a 3.2kb auto-regulatory CNS specific enhancer for *Dfd* which recapitulates the expression of *Dfd* gene in developing embryonic CNS. A smaller region of 630bp of NAE has also been reported to recapitulate the expression of the entire 3.2kb enhancer and this region is also being analysed.

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

The set of Nbs in the terminal region of CNS show sex specific proliferation and survival. 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 terminal Nbs (**tNBs**) in the larval VNC. Double-sex (**Dsx**) is the most downstream transcription factor of the sex-specification hierarchy. I intend to test the interaction between Abd-B and Dsx in gender specific proliferation of these cells.

Summary of work done until the beginning of this reporting year (upto 31 March, 2013)

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

It is known that *grim* gene play primary role in this apoptosis and relevant enhancer for the *grim* gene in Nbs lies in 23kb genomic region referred to as *NBRR-Neuroblast Regulatory Region*. A systematic screening of the 23kb NBRR is ongoing to identify Nb specific *grim* enhancer responsible for *grim* activation and pNb apoptosis. The 23kb region was divided into 4 overlapping genomic fragments (of 8-10kb) which are being screened for their ability to drive pNb specific expression of lacZ reporter in late third instar larval (**LL3**) brain. These 4 fragments have been amplified by PCR using region specific primers from genomic DNA and all the four fragments have been cloned into *pCasPer-lacZ* shuttle vectors to make transgenic lines. The transgenic line for two of the fragments have already been made and one of them have been analyzed which has helped us to narrow down the search to 8kb for the relevant *grim* enhancer. Simultaneously a 4kb enhancer of *grainyhead* which is responsible for its expression in CNS was sub-fragmented into three parts and transgenic lines for the three sub-fragments were generated and analysed. This analysis helped us 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 subesophageal ganglia.

The 630bp *Dfd* autoregulatory element was scanned for Hox-Exd binding sites and was two putative compound binding sites were identified for these two transcription factors. In vitro binding studies were done on these binding sites using EMSA and both of the binding sites showed binding to Dfd-Exd heterodimer. In order to investigate the *in vivo* relevance of these binding sites, these sites were mutagenized in 630bp DNA element and these various mutagenized forms of the enhancers have been sub-cloned into the *pCasPer-*nls-lacZ** shuttle vector and the transgenic lines are being made for the same. These transgenic lines will be tested for their capacity to activate the reporter β -galactosidase to test the relevance of the binding site and direct role for these transcription factors in auto-regulation of *Dfd* gene.

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

There are 12 Nbs in this region of CNS of which 8 stop dividing in both males and females at mid L3 stage of development. The remaining 4 Nbs which we refer to as tNBs have been known divide differentially in males and females. The hypothesis

for this part of work is that Abd-B and Dsx (Double-Sex being the most downstream member of sex specification hierarchy) play a role in sex specific proliferation of these tNbs.

The standardizations for co-staining with BrDU and other antibodies for the larval nervous system was done, this is important to monitor the dividing cells (Nbs and ganglion mother cells) in LL3 CNS. The co-staining procedure for BrDU and other epitopes will be used to monitor the tNbs division to test the role of Abd-B in this proliferation by making Abd-B null clones in larval CNS. This will be attempted in two genetic backgrounds, first wherein the dividing Nbs will be randomly marked by GFP using MARCM technique and in second case where GFP will be specifically driven in tNbs.

Summary of work done until the beginning of this reporting year (1 April, 2013-31 March, 2014)

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

The 3 out of 4 *enhancer-lacZ* lines of *NBRR* have been generated and analyzed. In case of wild type larvae by late L3 (LL3) stage of development all the pNbs in abdominal region normally undergo apoptosis. The expression pattern of *enhancer-lacZ* lines suggest that relevant enhancer for activation of *grim* gene in abdominal Nbs lies in the overlapping region of two 8kb sub-fragments *NBRRF3* and *NBRRF4*.

Genetic isolation of *grim* enhancer

A transposon insertion in *NBRR* was mobilized to generate smaller deletions and screening has resulted in isolation of a deletion line (*NBRR-22*). This deletion in transheterozygotic combination with already existing deletion of *NBRR* gives ectopic Nbs in the abdominal region of CNS at LL3 stage when all the Nbs in this region have normally undergone apoptosis. This deletion was mapped by PCR mapping and has narrowed the search region for the enhancer from 23kb to 7kb. Expectedly the 7kb region deleted in *NBRR-22* deletes a part of the overlapping region of *NBRRF3* & *F4* fragments.

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

The costaining of Dfd and Dpn (a neural progenitor specific marker) established that Dfd is expressed in neural progenitor cells (neuroblasts-Nbs).

Subsequently using the *NAE3.2-lacZ* transgenic line, it was established that expression of Dfd is auto regulated in Nbs since Dpn positive cells in maxillary region were LacZ positive as well.

In order to investigate the *in vivo* relevance of Hox-Exd binding sites tested earlier, these sites have been mutagenized in 630bp DNA element and the transgenic lines are being made for the mutagenized and wild type 600bp enhancer. These transgenic lines will be tested for their capacity to activate the reporter β -galactosidase in Nbs to test the relevance of the binding site and direct role for these transcription factors in auto-regulation of *Dfd* gene. Reagents are also being generated to test the activity of the *NAE3.2-lacZ* in genetic background mutant for known Hox cofactors like Exd and Hth.

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

A recent report characterized the Nb lineage in terminal. Report elucidated that female specific isoform of Dsx (Dsx^F) is responsible for the apoptosis of sex-specific tNbs in females while these cells continue dividing in males. The report overlaps with some of our existing results, but (A) it doesn't elucidate the molecular mechanism behind the phenomenon of apoptosis of sex-specific tNbs in females and (B) doesn't give any insight into how Dsx play a role in tNb proliferation and how sex specific tNbs are different from other 8 Nbs in the same region which stop dividing at mid L3 stage of development.

We tested the expression of Abd-B and Dsx in tNbs in CNS. We find that both these proteins are expressed in tNbs, to conclusively test the role of Abd-B and Dsx in tNb proliferation we intend to genetically test mutants for *Abd-B* and *dsx* for tNb apoptosis by making mutant clones for these gene in larval CNS using the MARCM technique. The reagents for these experiment are being generated. Since Grh is already known to play a role in pNb apoptosis, its role in tNbs apoptosis was tested as well. We find that Grh was expressed in tNbs of male larvae at mid L3 stage. Currently we are checking the role of Grh and different apoptotic genes (like *reaper* and *grim*) in tNbs apoptosis.

Simultaneously *Drosophila Cyclin E* gene is being tested to identify the mechanism behind continued sex specific proliferation of tNbs in male larval CNS. *cycE* is known to play a central role in cell cycle by promoting G1-S transition in dividing cells during

cell cycle and a detailed enhancer analysis has identified a 1.9kb enhancer element which controls the expression of the gene in Nbs. This enhancer is known to have binding site for Hox gene Abd-A and Abd-B and our analysis identify potential Dsx binding sites in the enhancer. A BrDU, lacZ and Dpn staining of *cycE-1.9kb-lacZ* transgenic flies show that lacZ line marks dividing Nbs in terminal

regions of CNS. The experiments are ongoing to characterize 1.9kb enhancer to understand how *cycE* integrates spatial temporal and sex specific information in tNbs. One of the central experiments to test the validity of this gene is to check the expression pattern of the enhancer in Abd-B and Dsx mutant MARCM clones, reagents are being generated for these experiments.

LABORATORY OF FUNGAL PATHOGENESIS

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

Faculty	Rupinder Kaur	Staff Scientist
PhD Students	Gaurav Bairwa	Senior Research Fellow (Till Jul. 2013)
	Maruti Nandan Rai	Senior Research Fellow
	Sapan Borah	Senior Research Fellow
	Vivek Kumar Srivastava	Senior Research Fellow
	Vandana	Senior Research Fellow
	Mubashshir Rasheed	Senior Research Fellow
	Priyanka Bhakt	Junior Research Fellow (Since Feb. 2014)
Other Members	Suneetha KJ	Technical Officer
	Gaurav Bairwa	Project-Senior Research Fellow (Since Aug. 2013)
	Shivarathri Raju	Project Assistant
	Ritu Taigwal	Project-Junior Research Fellow (Till Jul. 2013)
	Gujjula Rahul	Project-Junior Research Fellow
Collaborators	Rajendra Prasad	JNU, New Delhi
	Krishnaveni Mishra	UoH, Hyderabad
	Suman Thakur	CCMB, Hyderabad

Candida spp. are the leading cause of disseminated fungal infections and rank fourth among the most common nosocomial pathogens. Prevalence of *Candida glabrata*, the second most common cause of invasive candidiasis, is on the rise and accounts for 12-20% of total *Candida* blood stream infections. Despite being a common resident of healthy human microflora, *C. glabrata* causes life-threatening, systemic infections in the immunocompromised host. *C. glabrata* is a haploid budding yeast and exists in the blastoconidial form in both commensal and pathogenic states. Research in our laboratory is aimed at a better understanding of interaction of *C. glabrata* cells with host immune cells, antifungal drug resistance mechanisms and iron homeostasis mechanisms operational in *C. glabrata*.

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

Objectives

1. Analysis of intra-cellular behavior;
2. Screening of a *C. glabrata* mutant library for altered survival profiles; and
3. 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 (upto March 31, 2013)

Using an *in vitro* system comprised of human monocytic cell line THP-1, we demonstrated that wild-type *C. glabrata* cells possessed the ability to impede phagolysosome acidification, counteract/survive the reactive oxygen species generated and proliferate in THP-1 macrophages. We further showed that response of *C. glabrata* cells to THP-1 macrophage internal milieu is composed of three distinct phases: an Early-, a Mid- and a Late-phase. Activated DNA damage repair signaling, shut-down of translational machinery and remodeled carbon metabolism, and heterochromatinization of the *C. glabrata* genome mark the Early- (0-2 h) and the Mid-phase (3-12 h), respectively. In contrast, the Late-phase represents the proliferation stage with active yeast transcriptional machinery. Additionally, we identified, by screening a library of 18,350 *C. glabrata* Tn7 insertion mutants for altered intracellular survival profiles, a set of 56 genes required to survive and/or replicate in the intracellular milieu of THP-1 macrophages. These genes were implicated in diverse biological processes including chromatin and cell wall organization, signal transduction and Golgi vesicle transport.

Details of the progress made in the current reporting year (April 1, 2013 - March 31, 2014)

In the current reporting period, we characterized further a subset of 56 mutants for effects associated with survival of the macrophage antimicrobial barrage. Among ten mutants disrupted for vesicle-mediated transport, *Cgvps15::Tn7* and *Cgpan1::Tn7* mutants displayed less than 10% survival in THP-1 macrophages after 24 h co-culturing (Fig. 1A). Owing to highly attenuated growth of the *Cgpan1::Tn7* mutant at 37°C, we decided to focus on the *Cgvps15::Tn7* mutant. *CgVPS15* gene codes for a putative membrane-associated serine/threonine protein kinase and constitutes the regulatory subunit of the class III PI3K (Phosphoinositide 3-kinase) complex which catalyzes production of the lipid signaling molecule, phosphatidylinositol-3-phosphate (PI3P). Vps15 in *S. cerevisiae* phosphorylates and recruits the catalytic subunit of the PI3K complex Vps34 to the Golgi membrane and stimulates the PI3K activity of Vps34. Vps15 and Vps34 are also required for Gpa1 (GTP-binding α -subunit of the heterotrimeric G protein)-mediated pheromone signaling at the endosome in *S. cerevisiae*. Hence, to investigate the role of PI3K and GPCR (G-protein-coupled receptor) signaling in survival of *C. glabrata* cells in THP-1 macrophages, we deleted the ORFs encoding CgVps15, CgVps34 and CgGpa1 completely from the *C. glabrata* genome, using a homologous recombination-based strategy and examined their survival in THP-1 macrophages. While *Cggpa1* Δ cells underwent 5-fold replication, similar to *wt* cells, only 5-9% of *Cgvps15* Δ and *Cgvps34* Δ cells remained viable during 24 h co-incubation with THP-1 macrophages (Fig. 1B) indicating an essential role for CgVps15 and CgVps34 in survival in host macrophages. Importantly, ectopic expression of *CgVPS15* and *CgVPS34* could rescue the viability loss of *Cgvps15* Δ and *Cgvps34* Δ mutants in THP-1 macrophages, respectively (Fig. 1B).

Phenotypic characterization of mutants in medium supplemented with oxidative stress-causing agents (hydrogen peroxide and menadione), genotoxin (hydroxyurea), osmotic stressor (sodium chloride), cell wall stressor (caffeine) and membrane stress-causing agent (SDS) and at high temperature (37°C and 42°C), revealed sensitivity of *Cgvps15* Δ and *Cgvps34* Δ mutants to thermal, salt, oxidative, genotoxic, cell wall and cell membrane stresses (Fig. 1C). Notably, growth of

the *Cggpa1* Δ mutant remained unaffected under all tested conditions (Fig. 1C). Further, labelling of vacuolar membranes with the lipophilic styryl fluorescent dye FM4-64 revealed an enlarged vacuole, occupying most of the cell volume, in 58 and 64% of log-phase *Cgvps15* Δ and *Cgvps34* Δ cells, respectively, compared to 21% of *wt* cells carrying the large vacuole (Fig. 1D). Altogether, these data point to a pivotal role for CgVps15 and CgVps34 in survival of diverse stresses and maintenance of vacuolar morphology in *C. glabrata*.

Next, to examine if CgVps15 and CgVps34 are constituents of a functional PI3K in *C. glabrata*, we measured the PI3K activity in cell lysates of *wt*, *Cgvps15* Δ and *Cgvps34* Δ mutants in an *in vitro* enzymatic assay containing γ -P³² ATP and phosphatidylinositol. Compared to *wt* cells, only negligible amount of radiolabelled PI3P was observed in the *Cgvps34* Δ mutant (Fig. 1E). Contrarily, the PI3K appears to be functional in the *Cgvps15* Δ mutant as it could appreciably synthesize PI3P from ATP and phosphatidylinositol (Fig. 1E). Treatment of cell lysates with the PI3K inhibitor wortmannin abolished the PI3K activity in *wt* cell extracts (Fig. 1E). Collectively, these results indicate that the *CgVPS34* gene codes for the PI3K in *C. glabrata*, and CgVps34 possesses high basal levels of lipid kinase activity and may not be dependent upon CgVps15 for further stimulation under regular growth conditions.

To assess the role of PI3K signaling in virulence of *C. glabrata*, we measured organ fungal load in BALB/c model of systemic candidiasis after 7 days post intravenous injection with *wt*, *Cgvps15* Δ and *Cgvps34* Δ cells. As shown in Figure 1F, *Cgvps15* Δ and *Cgvps34* Δ - infected mice exhibited 10⁴-, 10³-, 10³- and 10⁵-fold lower yeast CFUs in kidneys, liver, spleen and brain, respectively, compared to corresponding organs of the *wt*-infected mice. Ectopic expression of *CgVPS15* and *CgVPS34* led to *wt*-like organ fungal load in *Cgvps15* Δ - and *Cgvps34* Δ - infected mice indicating that clearance of *Cgvps15* Δ and *Cgvps34* Δ mutants in mice was owing to the lack of *CgVPS15* and *CgVPS34* genes, respectively. Contrarily, significant differences in organ fungal burden between *wt*- and *Cggpa1* Δ - infected mice were not observed (Fig. 1F), thus, precluding a role for GPCR signaling in *C. glabrata* pathogenesis.

Altogether, our data suggest that CgVps15 and CgVps34, putative subunits of the PI3K complex, are essential for intracellular survival and virulence in *C. glabrata*. Notably, PI3K in *S. cerevisiae* is

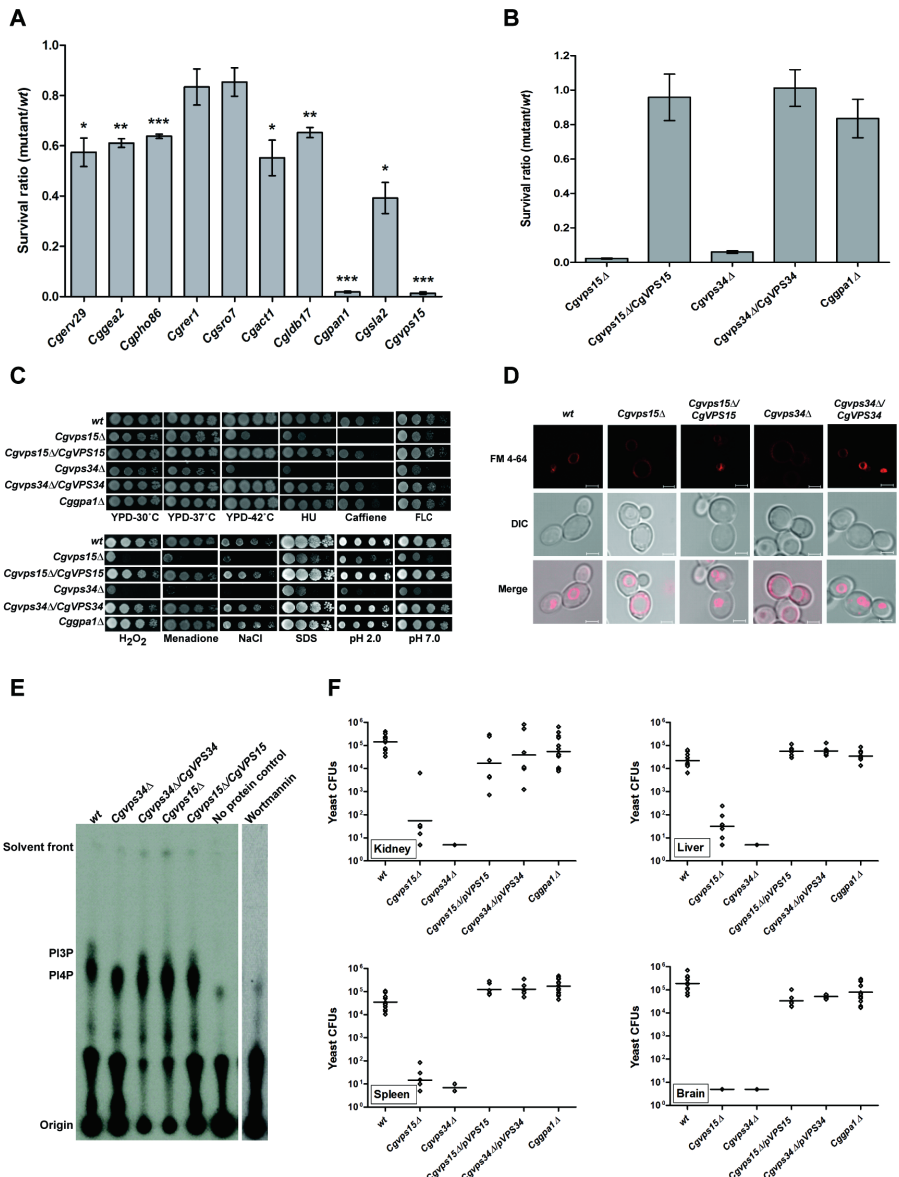


Figure 1. An essential role for phosphatidylinositol 3-kinase in intracellular survival and virulence in *C. glabrata*. (A & B) Intracellular survival and/or replication of *C. glabrata* mutants. After 24 h infection with indicated *C. glabrata* strains, THP-1 macrophages were lysed and appropriate lysate dilutions were plated on the YPD medium to determine yeast CFUs. Increase in CFUs for each strain was calculated by dividing the CFUs obtained at 24 h with those for 2 h. Survival ratio indicates the ratio of fold replication of mutant to that of *wt* cells 24 h post infection. (C) Serial dilution-spotting assay. Overnight grown cultures of *wt* and indicated mutants were 10-fold serially diluted in PBS from an initial OD₆₀₀ of 1 and spotted on YPD, YPD medium containing hydroxyurea (HU; 50 mM), caffeine (7.5 mM), hydrogen peroxide (H₂O₂; 20 mM), menadione (100 mM), sodium chloride (NaCl; 1 M) and sodium dodecyl sulphate (SDS; 0.05%), YNB, YNB medium buffered to pH 2.0 and pH 7.0. Plates were incubated at 30°C unless indicated otherwise and pictures were taken after 2-4 days (D) Confocal fluorescence micrographs of FM 4-64-stained log-phase *C. glabrata* cells. Scale bar = 2 μm. (E) Representative autoradiograph of thin-layer chromatography (TLC)-based separation of an *in vitro* PI3K assay products from three independent experiments. Log-phase cell extracts were incubated with γ -P³² ATP and phosphatidylinositol for 30 min and lipids were extracted. P³²-labelled-enzymatic products of PI3K were resolved by TLC using silica gel 60 and visualized by autoradiography. Wortmannin (30 M) was used to inhibit the PI3K activity. (F) BALB/c mice were infected with 4X10⁷ *C. glabrata* cells intravenously and sacrificed 7 days after infection. Appropriate dilutions of organ homogenates were spread-plated on YPD medium and fungal burden in liver, kidneys, spleen and brain was determined by CFU analysis. Diamonds and bars represent CFUs recovered from the target organs for individual mice and the geometric mean (n=6-14) of the CFUs per organ, respectively. Notably, of 14 mice infected with each mutant, no fungal CFUs were recovered from four *Cgyps15Δ*- and nine *Cgyps34Δ*-infected mice.

pivotal to endosomal trafficking processes. Consistently, our preliminary analysis revealed impaired processing and/or missorting of vacuolar hydrolase carboxypeptidase Y and cell wall proteins in *Cgvps15Δ* and *Cgvps34Δ* mutants. Hence, it is plausible that elevated stress susceptibility and mis-expression of fungal molecules due to aberrant anterograde and retrograde vesicular transport jointly contribute to impaired survival of *Cgvps15Δ* and *Cgvps34Δ* mutants in macrophages. Experiments are currently underway to address this hypothesis.

Project 2: Innate resistance of *C. glabrata* to fluconazole.

Objectives

1. Understanding the molecular basis of low inherent susceptibility of *C. glabrata* towards fluconazole; and
2. Identification of targets for combinatorial therapy with azole antifungals

Summary of the work done until the beginning of this reporting year (upto March 31, 2013)

We have previously reported three players of Rho GTPase-mediated signaling cascade (CgBem2, CgSlt2 and CgBnr1) and two components of RNA polymerase II mediator complex (CgMed2 and CgPgd1) to be essential for survival of stress imposed by the azole antifungal, fluconazole, which targets an essential enzyme, lanosterol 14 α -demethylase (CgErg11), of the ergosterol biosynthesis pathway. The *CgMED2* gene codes for a fungal-specific, tail-subunit of the multiprotein Mediator complex which interacts with the carboxy-terminal domain of the largest subunit of RNA polymerase II and acts as a bridge between upstream gene-specific regulatory proteins and core RNA polymerase II complex to activate target gene transcription. Among the three Mediator subunits, Head, Middle and Tail, the tail domain is the largest and structurally least conserved module and serves as a target for gene-specific activators. We could further show that inability to transcriptionally activate genes encoding a zinc-finger transcriptional factor, CgPdr1, and multidrug efflux pump, CgCdr1, largely accounts for elevated susceptibility of the *Cgmed2Δ* mutant towards azole antifungals.

Details of the progress made in the current reporting year (April 1, 2013 - March 31, 2014)

An apparent requirement for CgMed2 in transcriptional activation of the multidrug efflux

pumps upon fluconazole exposure prompted us to investigate whether high-level constitutive expression of *CgCDR1* and *CgPDR1*, often observed in fluconazole-resistant *C. glabrata* clinical isolates carrying gain-of-function (GOF) *CgPDR1* alleles, is abolished in the *Cgmed2Δ* mutant. Notably, the Zn₂-Cys₆ transcription factor, CgPdr1, is a master regulator of *CgCDR* gene expression in *C. glabrata*. Hence, we expressed the hyperactive/GOF allele of *CgPDR1*, which contains phenylalanine in place of leucine at 280aa position in the putative inhibitory domain (L280F), in both *wt* and *Cgmed2Δ* mutant. As shown in Figure 2A, *wt* cells expressing the *CgPDR1*-GOF allele exhibited robust growth even in the presence of 64 μ g/ml fluconazole while growth of *wt* cells carrying empty vector was attenuated in medium containing 16 μ g/ml fluconazole. Importantly, no discernible differences in growth profiles were recorded between *Cgmed2Δ* cells carrying either vector or hyperactive *CgPDR1* allele on fluconazole-containing medium (Fig. 2A). Consistently, although two- and four-fold upregulation of *CgPDR1* and *CgCDR1* gene expression, respectively, was observed in *wt* cells expressing the *CgPDR1*-GOF allele, no such induction was seen in *Cgmed2Δ* cells carrying *CgPDR1* allele containing the L280F substitution (Fig. 2B). These data indicate an essential role for CgMed2 in the *CgPDR1* GOF allele-mediated transcriptional activation of *CgCDR1* and *CgPDR1* genes.

In our previous screens for mutants with altered fluconazole susceptibility profiles, we have identified Tn7 insertions in genes coding for RNA polymerase II coactivators, CgSrb8, CgRgr1, CgNut1 and CgPgd1, which rendered *C. glabrata* cells sensitive to fluconazole. Of these, *CgPGD1* (*CgMED3*) and *CgRGR1* (*CgMED14*) code for putative components of tail module while *CgSRB8* (*CgMED12*) and *CgNUT1* (*CgMED5*) encode putative components of CDK/cyclin and middle module of the RNA polymerase II mediator complex, respectively. To examine whether these RNA polymerase II coactivators are also required for high levels of azole resistance conferred by the *CgPDR1*-GOF allele, we replaced the endogenous *CgPDR1* locus with the GOF *CgPDR1* allele in the genome of *Cgsrb8::Tn7*, *Cgrgr1::Tn7*, *Cgnut1::Tn7* and *Cgpgd1::Tn7* mutants. As a control, this exchange of *CgPDR1* allele was also performed in the *wt* and *Cgmed2::Tn7* mutant. Compared to *wt* cells, all mutants exhibited increased sensitivity to fluconazole (Fig. 2C). Intriguingly, expression of the hyperactive allele of *CgPDR1* led to elevated

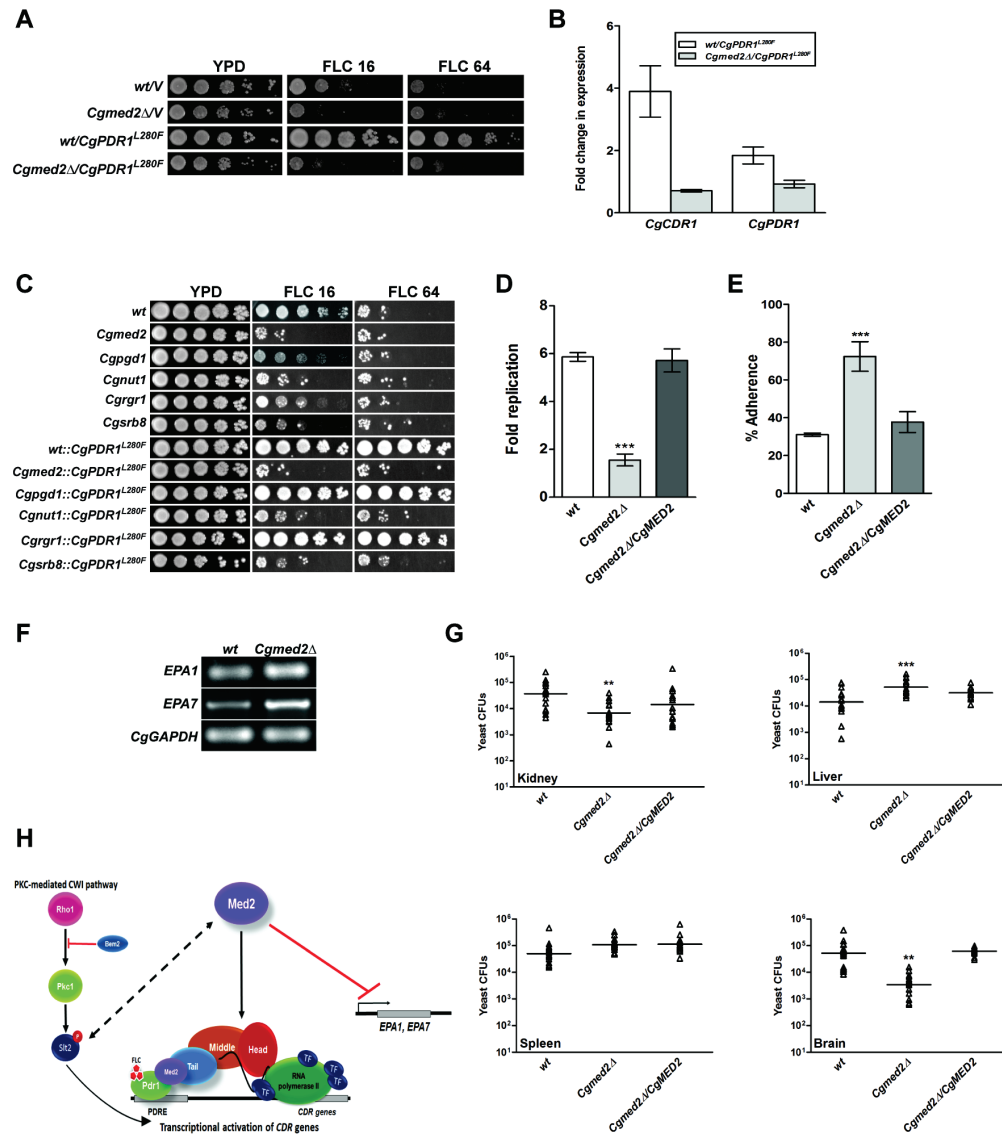


Figure 2. CgMed2 is required for virulence of *C. glabrata*. (A) Serial dilution spot assays of indicated *C. glabrata* strains on YPD medium lacking or containing 16 $\mu\text{g/ml}$ (FLC 16) and 64 $\mu\text{g/ml}$ (FLC 64) fluconazole. (B) qPCR-based quantification of *CgCDR1* and *CgPDR1* mRNA levels in log-phase *C. glabrata* strains. Data (mean \pm S.E.M. of 3-4 independent experiments) represent fold change in expression in wt and *Cgmed2* Δ cells expressing the GOF *CgPDR1* allele compared to respective strains carrying empty vector. (C) Serial dilution spot assay of indicated *C. glabrata* strains. (D) CFU assay-based intracellular replication measurement. After 24 h infection with indicated *C. glabrata* strains, THP-1 macrophages were lysed and appropriate lysate dilutions were plated on YPD medium to determine yeast CFUs. (E) Adherence enumeration of S³⁵-labelled *C. glabrata* cells to Lec2 ovary epithelial cells. Indicated *C. glabrata* strains were grown for 16-20 h in the CAA medium containing S³⁵(Met:Cys-65:25)-labeling mix followed by incubation with p-formaldehyde-fixed Lec-2 ovary epithelial cells for 30 min. Percentage adherence for each strain was determined by dividing the radioactive counts of Lec2 lysates to those of *C. glabrata* cell suspensions used for the adherence assay. (F) Reverse-transcription semi-quantitative PCR analysis of *EPA1*, *EPA7* and *CgGAPDH* gene expression in indicated *C. glabrata* strains. (G) Assessment of virulence potential of the *Cgmed2* Δ mutant. Groups of mice (N=12-20) were infected by tail vein injection with 4×10^7 *C. glabrata* cells and indicated organs were harvested 7 days post infection. Triangles represent the CFUs recovered from kidneys, liver, spleen and brain for individual mice. Bars represent the geometric mean of CFUs per organ. (H) Schematic representation summarizing the multiple roles played by CgMed2. Upon fluconazole exposure, activated PKC-mediated cell wall integrity pathway results in phosphorylation of the terminal MAPK, CgSit2, and CgMed2 upregulates the expression of *CDR* genes probably through association of the tail module of the Mediator complex with CgPdr1. In addition, CgMed2 is involved in the transcriptional silencing of the adhesin-encoding genes at subtelomeric regions thereby regulating adherence to epithelial cells and survival in the mammalian host.

resistance to azole antifungals in *Cgrgr1::Tn7* and *Cgpgd1::Tn7* mutants. Further, no growth advantage was conferred by the hyperactive *CgPDR1* allele to *Cgsrb8::Tn7*, *Cgnut1::Tn7* and *Cgmed2::Tn7* mutants on fluconazole-supplemented medium (Fig. 2C). These results indicate differential roles for tail subunits of the RNA polymerase II mediator complex in interaction with the CgPdr1 zinc finger transcription factor and suggest a prerequisite for functional CgMed2, CgNut1 and CgSrb8 proteins for azole resistance acquired *via* mutations in the *CgPDR1* gene during azole antifungal therapy.

Further, during our phenotypic analyses, we noticed that *Cgmed2Δ* cells always formed a loose pellet after centrifugation which could reflect altered cell-cell interactions and may impact pathogenesis. Hence, we next investigated the consequences of *CgMED2* deletion on virulence-associated traits including survival in macrophages and adherence to host tissues. Despite the similar uptake by THP-1 macrophages, no appreciable increase in colony forming units (CFUs) was observed for the *Cgmed2Δ* mutant while *wild-type* (*wt*) cells exhibited 6-fold replication in macrophages (Fig. 2D). This attenuated intracellular proliferation of the *Cgmed2Δ* mutant was not due to diminished growth under tissue culture conditions as similar number of CFUs were obtained when *wt* and *Cgmed2Δ* cells were grown in RPMI medium at 37°C and 5% CO₂, indicating a specific role for *CgMED2* in survival of the macrophage internal milieu. Next, we examined whether adherence of *C. glabrata* cells to host epithelial cells is affected by *CgMED2* deletion and found *Cgmed2Δ* cells to exhibit ~ 2.5-fold higher adherence to Lec2 ovary epithelial cells compared to *wt* cells (Fig. 2E). Importantly, ectopic expression of *CgMED2* in the *Cgmed2Δ* mutant resulted in *wt*-like adherence (Fig. 2E). Consistent with the hyperadherence phenotype, basal transcript levels of the adhesin-encoding genes, *EPA1* and *EPA7*, were found to be higher in *Cgmed2Δ* mutant compared to *wt* cells (Fig. 2F). It is worth noting that the *EPA7* gene is located in subtelomeric region and expressed at very low levels under *in vitro* conditions due to subtelomeric silencing. Hence, it is likely that lack of CgMed2 may result in derepression of *EPA1* and *EPA7* transcription.

Lastly, we assessed the fungal burden in kidneys, liver, spleen and brain of BALB/c mice infected intravenously with *Cgmed2Δ* cells. We recovered

5- and 15-fold lower yeast CFUs from kidneys and brain, respectively, of the *Cgmed2Δ* - infected mice compared to those of the *wt*-infected mice (Fig. 2G). No statistically significant CFU differences were observed in spleen harvested from mice infected with *wt* and *Cgmed2* mutant (Fig. 2G). Quite surprisingly, hepatic fungal load of the *Cgmed2Δ* - infected mice was 4-fold higher than that of the *wt*-infected mice (Fig. 2G). The precise relevance of this observation remains to be elucidated.

Taken together, our data implicate CgMed2 in the *GOF CgPDR1* allele-mediated transcriptional activation of multidrug efflux pumps, derepression of subtelomerically located adhesin-encoding genes, intracellular survival and virulence in a murine model of systemic candidiasis.

Publications

1. Bairwa G, Balusu S and Kaur R (2013). Aspartyl proteases in human pathogenic fungi: roles in physiology and virulence in the book entitled "The Fungal Cell Wall", Ed. Héctor M. Mora-Montes. **Nova Science Publishers** 159-198.
2. Rai MN, Borah S, Bairwa G, Balusu S, Goritala N and Kaur R (2013). Establishment of an *in vitro* system to study intracellular behavior of *Candida glabrata* in human THP-1 macrophages. **Journal of Visualized Experiments** 82: e50625.
3. Bairwa G, Rasheed M, Taigwal R, Sahoo R and Kaur R (2014). GPI (glycosylphosphatidylinositol)-linked aspartyl proteases regulate vacuole homeostasis in *Candida glabrata*. **Biochemical Journal** 458: 323-334.
4. Shah AH, Singh A, Dhamgaye S, Chauhan N, Vandeputte P, Suneetha KJ, Kaur R, Mukherjee PK, Chandra J, Ghannoum MA, Sanglard D, Goswami SK and Prasad R. Novel role of a family of major facilitator transporters in biofilm development and virulence of *Candida albicans*. **Biochemical Journal** (In press).

Other Publications

1. Kaur R (2013). Review of: Annual review of microbiology, 2012. Edited by Susan Gottesman, Caroline S. Harwood and Olaf Schneewind. **Current Science** 105: 390-391.

LABORATORY OF GENOMICS AND PROFILING APPLICATIONS

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	Soumya Rao	Senior Research Fellow
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	Vineesha Oddi	Project-Junior Research Fellow (Since Oct. 2013)

Objectives

1. To study the human genetic diversity among various population groups in India; and
2. Plant-fungal interaction studies in the chilli-*Colletotrichum* pathosystem

Project 1: To study human genetic diversity in various population groups in India.

Summary of work done until the beginning of this reporting year (upto March 31, 2013)

Towards our efforts to build a SNP-based panel for forensic human identification (HID) in Indian populations, we had previously reported testing of a panel comprising of 92 single nucleotide polymorphisms (SNPs) proposed by Dr. Kenneth Kidd for HID in various Indian populations employing the Illumina *GoldenGate*[®] Genotyping Assay system (96-plex). Based on the distribution and abundance of the alleles of the studied SNPs, it was observed that only about one-third of the Kidd panel could meet the criteria set for HID purposes in the Indian populations. In order to incorporate additional SNPs for forensic HID in India, a bioinformatics approach was adopted to screen SNPs from publicly available databases based on numerous parameters described in the previous report. A panel of ~270 SNPs was shortlisted for testing in the Indian populations in order to select the best performing SNPs for identity-testing. Further, we had reported about the initiative to unravel the genetic diversity among different human populations in India by studying the microbiome variation.

Details of progress made in the current reporting year (April 1, 2013 - March 31, 2014)

a) SNP for HID purposes

In the reporting year, in addition to the identity-testing SNPs shortlisted previously, phenotype

informative SNPs have been included. SNPs known to be associated with externally visible characteristics (EVCs) like skin pigmentation and height, were shortlisted based on literature and those that are reported to be strongly associated and/or having a pronounced effect on the target phenotype were included in the panel to be tested in Indian populations. A total of 43, 6 and 17 SNPs were selected for skin pigmentation, BMI and height, respectively. Finally, a total of 384 SNPs (which include identity-testing, phenotype-informative SNPs described previously) were shortlisted for testing in Indian populations, which would be genotyped employing the Illumina BeadXpress genotyping platform in the coming days.

b) Studies on uni-parental markers for HID purposes

As part of genetic variation studies and its application in forensic HID for Indian populations, studies involving uni-parental markers have been undertaken. Both mitochondrial genome and Y-chromosome markers carry information relevant to maternal and paternal lineages, respectively and hence are useful for evolutionary studies and in forensic cases like kinship analysis, mass disaster and missing person identification. Also, because of high copy-number and increased tolerance to DNA degradation as compared to the nuclear genome, the mitochondrial genome is of great help especially in mass disaster cases wherein the DNA can be substantially degraded. Even though PCR amplification followed by sequencing is the regular procedure to analyze the mitochondrial genomes, however, in certain cases, the sequencing results might not be conclusive owing to gaps and uncalled bases. In such cases, it is desirable to sequence the PCR products with various overlapping sequencing primers to obtain conclusive results

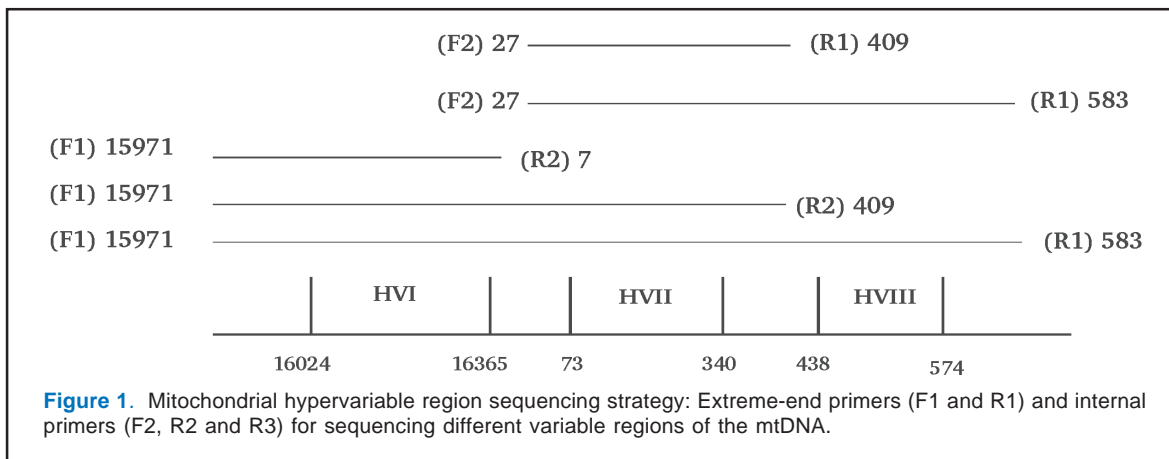


Figure 1. Mitochondrial hypervariable region sequencing strategy: Extreme-end primers (F1 and R1) and internal primers (F2, R2 and R3) for sequencing different variable regions of the mtDNA.

from mitochondrial analyses. In addition to the routinely used hypervariable (HV) regions I and II, the sequencing of HV-III region would help to build a reliable consensus sequence of the hypervariable regions of the mtDNA (Fig.1).

The complete HVI, II and III sequences from various representative Indian populations would be generated to study the human diversity patterns and compared with the corresponding Y-STR data to understand the phenomena of migration like patrilocality and matrilocality. The mtDNA sequence of HV regions would be helpful in determining the haplogroups of the people who are the contributors of a biological sample in forensic cases.

c) Studies on human salivary microbiome in Indian populations

The primary objective of the project is to study the human salivary microbiome to identify the various bacterial taxa in saliva that may be able to provide insights into human population structure and migrations. It was planned to study the microbiome diversity in saliva samples sourced from various geographical locations (states) in India. In the previous report, a brief introduction and objectives were mentioned.

Here, two approaches were adopted

1. Capillary electrophoresis - based sequencing

In the pilot study, DNA from the saliva samples of eight individuals (4 each from Jammu & Kashmir and West Bengal) was used as template to amplify the variable (V1 and V2) regions of the 16S rRNA gene. The amplicons were cloned into TOPO cloning vector and ~120 individual clones from each of the saliva sample were sequenced by capillary

electrophoresis (CE) to assess the microbial diversity in these samples. The 16S rRNA sequences were subjected to BLAST analysis and uploaded into Ribosomal Database Project (RDP) to identify the organism. A minimum threshold of 90% match in BLAST was adopted to assign a sequence to a particular bacterial genera.

2. Massively parallel sequencing approach

A library suitable for sequencing on the Illumina platform was prepared from the 16S rRNA amplicons, using published protocols from saliva samples belonging to 94 unrelated individuals (10-12 individuals from 8 different geographical regions in India) and the library was sequenced on the Illumina MiSeq next generation sequencing (NGS) platform (2X250 cycles) at the Max Planck Institute for Evolutionary Anthropology, Germany. The data obtained (~10 GB) were processed and classified into operational taxonomic units (OTUs) and the corresponding bacterial genera were identified using the Bayesian classifier on the Ribosomal Database Project (RDP II).

Six samples common to CE and NGS approaches were compared for the microbiome composition and informativeness and the data showed that the NGS approach provided much more detailed information of the microbial phyla and genera present in the studied saliva samples. The number of phyla and genera discovered increased tremendously upon sequencing at a greater depth by NGS. The TM7 microbial sequences could not be observed in the saliva samples from WB population when sequenced by CE-based method, however NGS approach showed that they constitute ~1.1% of the total microbiome in this population. *Streptococcus* was the most abundant

genera (~40%) observed in the Indian populations, which was slightly higher as compared to other world populations reported in previous studies. The distribution of the major phyla across the major geographical locations is shown in Fig.2. Although, only a few bacterial phyla contributed to a major proportion (> 90%) in all the populations, considerable variation was observed in the abundance of the bacterial phyla among the populations, which warrants further analysis to substantiate and examine whether the microbiome is geographically structured in the Indian populations.

Further, rarefaction study was carried out to ascertain whether the number of genera discovered by both the sequencing approaches was enough to decipher the total microbiome richness in the saliva of the selected samples and the data suggested that the NGS approach is an efficient strategy to uncover the richness in the targeted saliva samples. Further analysis is under progress to understand the microbiome variation in the various Indian populations and to inquire whether the microbiome variation is structured according to geographical regions of India.

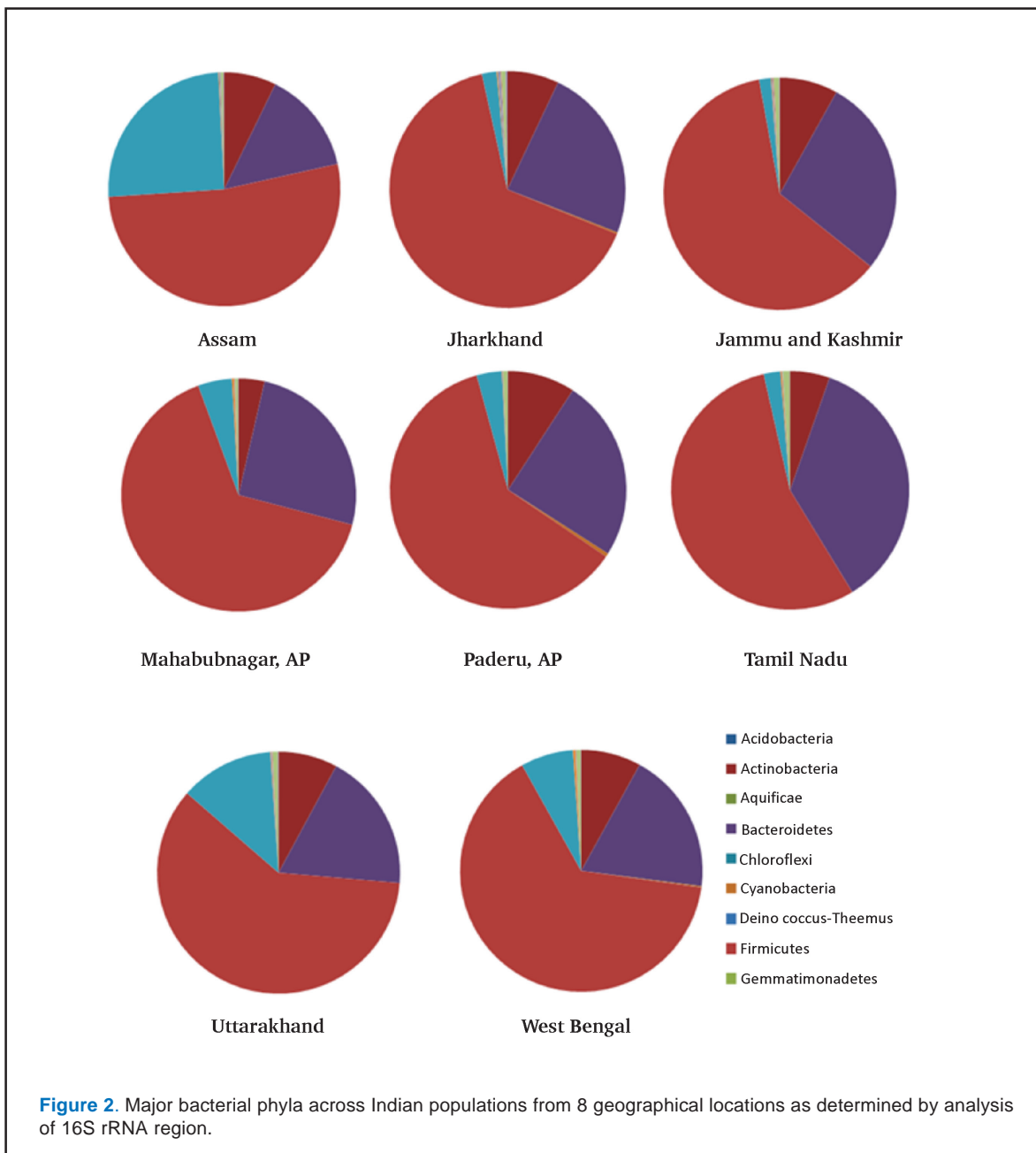


Figure 2. Major bacterial phyla across Indian populations from 8 geographical locations as determined by analysis of 16S rRNA region.

Project 2: Plant-Fungal Interaction studies in the Chilli - *Colletotrichum* Pathosystem.

Summary of work done until the beginning of this reporting year (upto March 31, 2013)

This is a new activity.

Chilli, a native of Central and South America, is an indispensable spice in everyday cuisine all over the world due to the characteristic piquancy of capsaicin, and is also a rich source of vitamin C. *Capsicum annuum* L., family Solanaceae, is the most economically important cultivated species of chilli and is the most widely grown spice in the world. India has the largest area under cultivation for chilli and is the world leader in its production, consumption and export, with Andhra Pradesh and Telangana contributing to 49% of total production. One of the most devastating diseases and major economic constraint to chilli production, especially in tropical and subtropical regions of the world, including India is anthracnose. It mostly affects the ripe fruits turning red. It is caused by the fungus *Colletotrichum*, a large genus belonging to the Ascomycetes that is one of the most widespread and important genera of plant-pathogenic fungi. The disease is characterized by dark, sunken necrotic lesions with concentric rings of acervuli containing curved conidiospores, reducing the quality and marketability of chilli fruits. Traditional control measures like use of fungicides are not sustainable and there are no resistant cultivars of *C. annuum* successfully developed so far. The natural resistance to anthracnose has been observed in two species: *C. chinense* and *C. baccatum*, with later showing the broad spectrum of resistance to many fungi. *Colletotrichum truncatum* (formerly called as *C. capsici*) is the most predominant species in India causing chilli anthracnose. It follows a subcuticular/intramural colonization strategy in chillies. Some recent studies suggest that this pathogen has asymptomatic endophytic phase after initial infection and prior to necrotrophic development. However, both infection strategies contrast with the intracellular hemibiotrophic infection of most other *Colletotrichum* species, where the pathogen first establishes intracellular biotrophic hyphae inside living host cells before switching to destructive necrotrophy.

With the whole genome sequence available for chilli and four *Colletotrichum* species, the chilli - *C. truncatum* pathosystem provides an excellent model for studies of the infection process and molecular interactions between the host and

pathogen. Elucidation of the mechanism of resistance in *Capsicum* spp., and virulence of *C. truncatum* would lead to the development of resistant genotypes. This 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 using random insertional mutagenesis and whole genome sequencing of the *C. truncatum* genome.

Details of progress made in the current reporting year (April 1, 2013- March 31, 2014)

The plant materials (fruits and seedlings) of *C. baccatum* var. PBC80 and *C. annuum* var. CAR1 were procured from J.K. AgriGenetics, Hyderabad, while the six *C. truncatum* cultures were procured from Microbial Type Culture Collection and Gene Bank (MTCC), Institute of Microbial Technology, Chandigarh (MTCC numbers 2071, 3414, 8473, 9691, 10147 and 10327). All the cultures showed different colony morphology and sporulation capacity. Conserved universal fungal barcode primer pairs for internal transcribed spacer region (ITS), ITS 5 and ITS 4, encoded in the 28S ribosomal subunit, and large subunit of ribosome (LSU) gene fragments were used to confirm the identity of the fungal species. The ITS and LSU regions were amplified from the genomic DNA of all the cultures resulting in ~550 bp and ~950 bp fragments, respectively. ITS amplicons were sequenced using specific forward and reverse primers. BLAST analysis showed 100% homology with *C. truncatum* confirming the authenticity of the MTCC cultures. Pathogenicity of these fungi was established by inoculating the conidial suspension from sporulating cultures on chilli by wound-drop method and recovering the fungus by transferring the lesion developed from chilli onto PDA, proving Koch's postulates (Fig.3). The highly virulent fungal strain (MTCC No. 3414) among the collection was selected for the subsequent experiments.

For *de novo* whole genome sequencing, genomic DNA was extracted from *C. truncatum* (MTCC No. 3414) using the DNeasy Plant Minikit (Qiagen, Germany) and was quantified by Qubit assay (Life Technologies, USA). Two Short Insert Paired End (300 bp and 500 bp), and two Long Insert Mate Pair (3000 bp and 5000 bp) barcoded genomic DNA libraries were constructed and the sequencing was performed at the Cofactor Genomics (St. Louis, MO, USA) on the Illumina HiSeq platform (2x100 bp reads). The raw data from each of the libraries consisted of 255 million reads in total. Base calls were generated using Casava 1.8.2 (Illumina), and

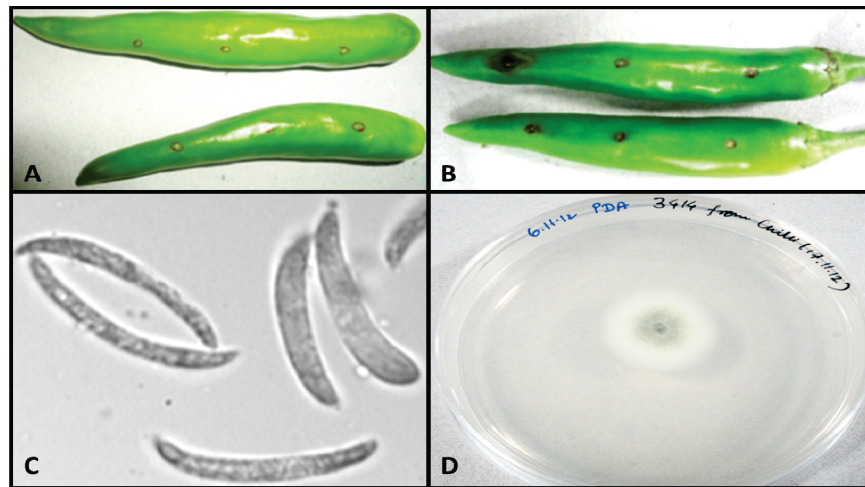


Figure 3. Pathogenicity assay; A-B: *C. annuum* fruits inoculated with distilled water as control (A) and *C. truncatum* (MTCC no. 3414) conidia (B), respectively; C: light microscopy of conidia observed under 60x, D: recovery of fungus from infected chilli on PDA.

the resulting demultiplexed sequence reads were filtered for low quality. An assembly on the sequence data was performed using SOAPdenovo 1.05 (Beijing Genomics Institute, Beijing, China). The assembled sequence consisted of 81 scaffolds with a total length of 55.3 Mb, equivalent to 460X coverage, and N50 of 1.6 Mb (i.e. 50% of all bases are contained in scaffolds of at least 1.6 Mb). For preliminary annotation of the assembly, scaffolds were aligned to the predicted gene set of well annotated *C. higginsianum* genome using BLASTX with threshold expect value of $1e^{-3}$ identifying 6,511 unique genes. In future, further analysis of the genomic data obtained, phylogenetic analysis with other *Colletotrichum* species, gene annotation by *ab initio* gene prediction methods and functional characterization of pathogenicity genes would be carried out.

In order to identify pathogenicity genes in *C. truncatum* through forward genetics approach, random insertional mutagenesis of *C. truncatum* conidia by *Agrobacterium tumefaciens* mediated transformation (ATMT) was taken up using *A. tumefaciens* strain C58C1 harboring binary vector pBIN-GFP-hph (kind gift from Dr. Richard O'Connell, INRA-BIOGER, France), which carries both the hygromycin B phosphotransferase (hph) as

selection marker for transformants and green fluorescent protein (gfp) genes as reporter gene for tracking the fungus *in planta*. Aliquots of *A. tumefaciens* culture mixed with conidial suspension of *C. truncatum* in glycerol induction broth supplemented with acetosyringone were spread on cellophane membrane supported on solid medium. After co-cultivation, fungal transformants were selected on PDA containing hygromycin, cefotaxime and spectinomycin. Around 700 transformants have been obtained so far, some of which were confirmed by polymerase chain reaction (PCR) with hph primers. Mitotic stability of the transformants was confirmed by repeated subculturing on PDA without hygromycin and then on PDA with hygromycin. The transformants are currently being screened for partial or complete loss of pathogenicity on chilli. The characterization of pathogenicity mutants obtained in the screen would be carried out in future.

Publications

1. Sharma V and Nandineni MR (2014). Assessment of genetic diversity among Indian potato (*Solanum tuberosum* L.) collection using microsatellite and retrotransposon based marker systems. **Molecular Phylogenetics and Evolution** 73: 10-17.

LABORATORY OF IMMUNOLOGY

Role of advanced glycation end products (AGE) in inducing obesity and its regulation

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	S Adeel Husain Zaidi	Senior Research Fellow
	Raveendra Babu M	Senior Research Fellow
	Neeharika Verma	Senior Research Fellow
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	T Navaneetha	Technical Assistant
	Binay Kumar Sahoo	Project-Junior Research Fellow
Collaborators	Biswadev Bishayi	Calcutta University, Kolkata
	Tushar Basu Baul	NEHU, Shillong

Objectives

1. Understanding and regulation of inflammatory and tumorigenic responses;
2. Understanding and regulation of advanced glycation endproducts (AGE)-mediated lipogenesis; and
3. Understanding the molecular mechanism of autophagy.

Summary of work done until the beginning of this reporting year (upto March 31, 2013)

Advanced glycation end products (AGE) that accumulate, due to high amounts of 3- or 4-carbon sugars derived from glucose, cause multiple consequences in diabetic patients and aged persons. The transcription factor, peroxisome proliferator-activated receptor gamma (PPAR γ), is often downregulated in the diabetic condition. Drugs targeting PPAR γ for diabetes therapy were developed. We found that AGE inhibited PPAR γ activity induced by PPAR γ activators, like troglitazone, rosiglitazone, oleamide, and anandamide. AGE induced translocation of PPAR γ from nucleus to cytoplasm, which was increased on activation of ERK in cells. Antioxidants that inhibit AGE-induced NF- κ B activation via ROI generation were unable to protect AGE-mediated decrease in PPAR γ activity. Only mangiferin, a β -D-glucoside, prevented AGE-mediated decrease in PPAR γ activity. Mangiferin interacts with PPAR γ and enhanced its DNA binding activity as predicted by *in silico* and shown by *in vitro* DNA-binding

activity. Overall, our data suggest that (i) mangiferin inhibited AGE-induced ERK activation thereby inhibited PPAR γ phosphorylation and cytoplasmic translocation; (ii) mangiferin interacts with PPAR γ and enhances its DNA-binding ability. With these dual effects, mangiferin can be a potential candidate for developing therapeutic drug against diabetes.

Cardiac glycosides are potent inducers of cell death, but very toxic to cells. Use of these molecules as therapeutics after reducing toxicity would be viable strategy. In this report we provide evidences that oleandrin alone induced cell death, but pulse treatment of it did not show any induction of cell death. Pulse exposure of oleandrin, but not by azadirachtin, resveratrol, thiadiazolidine, or benzofuran enhanced IL-8-, but not TNF-, IL-1-, EGF-, or LPS-mediated induction of NF- κ B. This enhancement of NF- κ B activation is not restricted in specific cell types. Increase in IL-8-mediated biological responses further proved in the oleandrin-pulsed cells upon overexpression of TRAF6. Oleandrin-pulsed cells did not show increase in NF- κ B activation mediated by other ligands for G-protein-coupled receptors, except IL-8. Oleandrin-pulse increased expression of IL-8Rs (CXCR1 and CXCR2) thereby increased IL-8-induced biological responses like chemotaxis, proteolytic enzymes release and activation of NF- κ B and AP-1. Oleandrin pulse treatment decreased cell surface IL-8Rs by changing the microviscosity and further culturing compensated IL-8Rs by degranulation and expression of NF-AT-dependent transcription. Overall, first time we are providing data that the

pulse exposure of toxic cardiac glycoside enhances biological activity in a typical manner by activating IL-8-mediated biologic responses. This study might be helpful to design oleandrin for therapy against those diseases where cell migration is required to improve the conditions of patients.

Details of progress made in the current reporting year (April 1, 2013 - March 31, 2014)

1) Advanced glycation end products (AGE) induce lipogenesis: regulation by natural Xanthone through inhibition of ERK and NF-kappaB.

Advanced glycation end products (AGE) accumulate in diabetic patients and aged persons

due to high amounts of 3- or 4-carbon derivatives of glucose. Understanding the mechanism of AGE-mediated signaling leading to these consequences, like oxidative stress, inflammation, apoptosis, etc. and its regulation would be a viable strategy to control diabetic complication and age-related diseases. We have detected the probable mechanism by which AGE increases lipogenesis, the cause of fatty liver in diabetic patients. AGE increased lipid accumulation not only in liver cells (Fig.1A), but also in other cell types as shown by Oil Red O staining. AGE-mediated upregulation of several transcription factors, like NF- κ B, AP-1, NRF, SREBP, etc. was observed as determined by gel shift assay (Fig.1B). AGE-mediated several of these activities had been occurred upon interaction with its receptor, RAGE as shown by

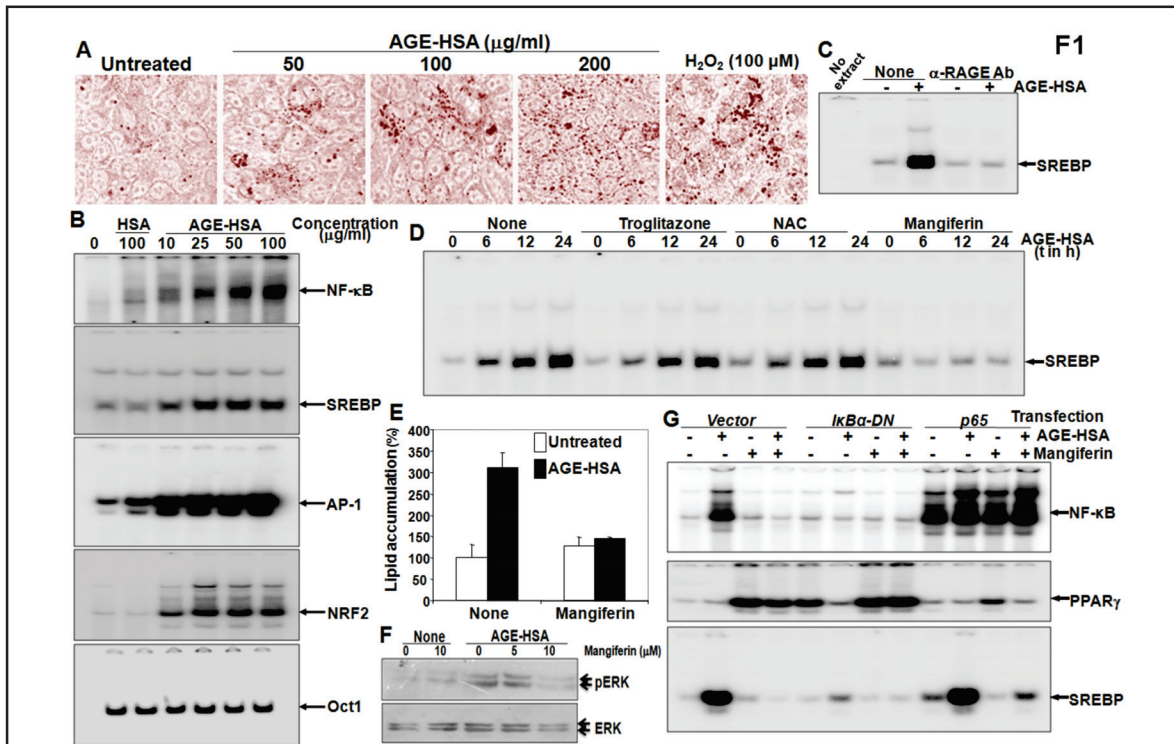


Figure 1. Advanced glycation endproducts induce lipogenesis: regulation by natural xanthone through inhibition of ERK and NF-kappa B. HepG2 cells were treated with different concentrations of AGE-HSA or 100 μ M of H_2O_2 for 24 h. Cells were stained for 15 min in freshly diluted Oil Red O solution and visualized under the microscope (A). HepG2 cells were stimulated with 100 μ g/ml HSA or different concentrations of AGE-HSA for 24 h. Nuclear extracts (NE) were prepared and measured for NF- κ B-, SREBP-, AP-1-, NRF2-, and Oct1-DNA binding (B). HepG2 cells, preincubated with anti-RAGE Ab for 2 h were stimulated with AGE-HSA for 24 h and SREBP-DNA binding was assayed by EMSA from NE (C). HepG2 cells, treated with troglitazone (10 μ M), NAC (5 mM) or mangiferin (10 μ M) for 6 h were stimulated with 100 μ g/ml of AGE-HSA for different times till 24 h. NE were prepared and assayed for SREBP-DNA binding (D). Cells were stained with Oil Red O and the absorbance of Oil Red O stain at 500 nm was taken from the supernatant of cells, treated with mangiferin (10 μ M) for 2 h followed by stimulated with AGE-HSA (100 μ g/ml for 24 h) and represented in percentage (E). HepG2 cells, treated with different concentrations of mangiferin for 2 h were stimulated with AGE-HSA for 12 h. WCE were prepared and 50 μ g WCE was used to measure the amount of phospho-ERK, followed by total ERK by Western blot (F). Cells were transiently transfected with p65, $\kappa B\alpha$ -DN and GFP constructs. After 12 h of culture, cells, treated with mangiferin (10 μ M) for 2 h were stimulated with AGE-HSA for 12 h. NF- κ B-, PPAR γ - and SREBP-DNA binding were assayed from NE (G).

inhibition of SREBP DNA binding upon incubation of anti-RAGE antibody on cells (Fig.1C). Antioxidant like NAC or known activator troglitazone, an anti-diabetic agent, except mangiferin were unable to protect AGE-induced activation of SREBP (Fig.1D) and subsequent lipid accumulation (Fig.1E). AGE increased the phosphorylation of ERK, and IKK and also DNA binding ability of SREBP, thereby its dependent

gene transcription. AGE induces NF- κ B which might suppress PPAR γ activity, in turn reducing lipid breakdown and mobilization. Mangiferin not only inhibits AGE-mediated ROI generation that requires NF- κ B activation, but also inhibits ERK (Fig.1F) and IKK activity, thereby suppression of SREBP activity and lipogenesis. NF- κ B suppressed PPAR γ activity, but increased SREBP-DNA binding and that mangiferin inhibited both

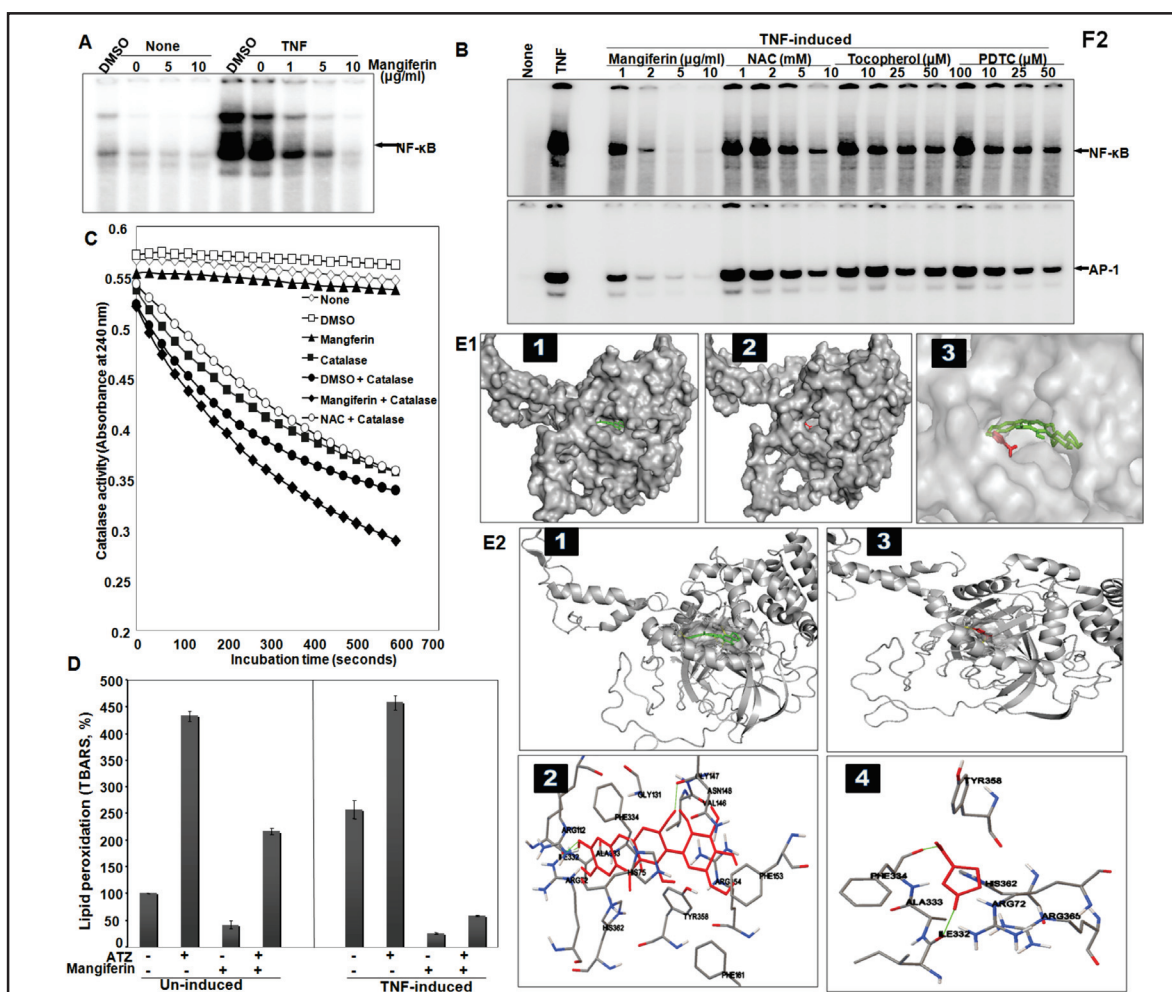


Figure 2. A natural xanthone glucoside increases catalase activity thereby redox sensitivity and suppresses NF-kappa B and activator protein 1. U-937 cells (2×10^6 /ml) were preincubated at 37°C for 4 h with different concentrations of mangiferin, followed by 30-min incubation with 100 pM of TNF. After these treatments, nuclear extracts were prepared and then assayed for NF- κ B DNA binding by gel shift assay (A). Cells, pretreated with varying concentrations of different antioxidants for 3 h were stimulated with TNF (100 pM) for 2 h. Nuclear extract were assayed for NF- κ B, and AP-1 DNA binding activity (B). Equal amount of catalase protein were pre-incubated with different concentrations of mangiferin *in vitro*. Catalase activity was assayed subsequently by spectrometric analysis at 240 nm (C). Cells were pretreated either with mangiferin (10 $\mu\text{g}/\text{ml}$) for 4 h or ATZ (2 mM) for 2 h and then treated with TNF(100 pM) and were cultured for 12 h. WCE extract were prepared and 70 μg of protein was used for the assay of amount of MDA as a measure of lipid peroxidation (D). The results were expressed as percentage increase in the amount of MDA formed. The results shown are the mean (\pm SEM) absorbance of independent, duplicate assays. Docking interaction of catalase with mangiferin (E1, 1), ATZ (E1, 2), and in combination (E1, 3). Details interaction involving amino acids of catalase was shown with mangiferin (E2, 1 and 3) and ATZ (E1, 2 and 4).

SREBP and NF- κ B and thereby inhibited AGE-mediated lipogenesis as detected from cells having NF- κ B upregulated (*p65*-overexpressed) or downregulated (*I κ B α -DN*-transfected) cells (Fig.1G). Mangiferin has shown a double-edged sword effect to suppress AGE-mediated ailments by reducing ROI-mediated responses as antioxidant and inhibiting SREBP activation thereby lipogenesis, suggesting its potential efficacy against diabetes and obesity-related diseases.

2) Mangiferin increases catalase activity thereby redox sensitivity and suppresses NF-kappa B and activator protein 1.

Mangiferin, a c-glycosyl xanthone glucoside and a known polyphenol, has shown anti-inflammatory, anti-oxidant, and anti-tumorigenic activities. In the present study, we have investigated the molecular mechanism for the antioxidant property of mangiferin. As, nuclear transcription factor kappaB (NF- κ B) and activator protein 1 (AP-1) are the major transcription factors involved in the propagation of chronic inflammation and tumor, we hypothesized that modulating the activity of NF- κ B and AP-1 will be a valuable and major therapeutic target in regulating the redox-sensitive ailments. We compared different antioxidants for their role in inhibiting TNF-induced pro-oxidant and pro-inflammatory signaling. Our results show that mangiferin blocks TNF-induced NF- κ B (Fig. 2A) and AP-1 activation in a dose dependent manner. Mangiferin, like known anti-oxidants, N-acetyl cysteine (NAC), tocopherol and pyrrolidone dithiocarbamate (PDTC), inhibits TNF-induced reactive oxygen intermediates (ROI) generation, but was most potent in inhibiting NF- κ B and AP-1 activation induced by TNF (Fig. 2B) as well as other

inflammatory agents like phorbol myristate acetate (PMA), endotoxin, oleamide and H₂O₂. Mangiferin was found to increase the catalase activity *in vitro* (Fig. 2C) and thereby reduced lipid peroxidation more potently than known inhibitor of catalase, aminotriazole (ATZ) (Fig. 2D). Mangiferin and ATZ interact with the catalytic site of catalase, but in separate amino acid residues (Fig. 2E1) and the predicted amino acids were detected (Fig. 2E2). The affinity of catalase is more with mangiferin than ATZ as detected from the free energy binding data. Hence mangiferin with its ability to inhibit NF- κ B and to increase the catalase activity may prove to be a potent drug for anti-inflammatory and anti-oxidant therapy.

Publications

1. Mulakayala C, Babajan B, Madhusudana P, Anuradha CM, Rao RM, Nune RP, Manna SK, Mulakayala N and Kumar CS (2013). Synthesis and evaluation of resveratrol derivatives as new chemical entities for cancer. ***Journal of Molecular Graphics and Modelling*** 41: 43-54.
2. Basu Baul TS, Kundu S, Linden A, Raviprakash N, Manna SK, Guedes da Silva MF. (2014) Synthesis and characterization of some water soluble Zn(II) complexes with (E)-N-(pyridin-2-ylmethylene)arylamines that regulate tumour cell death by interacting with DNA. ***Dalton Transactions*** 43: 1191-1202.
3. Raviprakash N and Manna SK. Pulse exposure of cardiac glycoside enhances IL-8-mediated biological responses by increasing cell surface IL-8 receptors. ***British Journal of Pharmacology*** (In press).

LABORATORY OF MAMMALIAN GENETICS

Epigenetic mechanisms underlying developmental pathways

Faculty	Sanjeev Khosla	Staff Scientist
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	Thushara Thamban	Senior Research Fellow
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	S Mahalingam	IIT, Madras, Chennai

Project 1: *DNMT3L*: Role in Development

Summary of work done until the beginning of this reporting year (upto March 31, 2013)

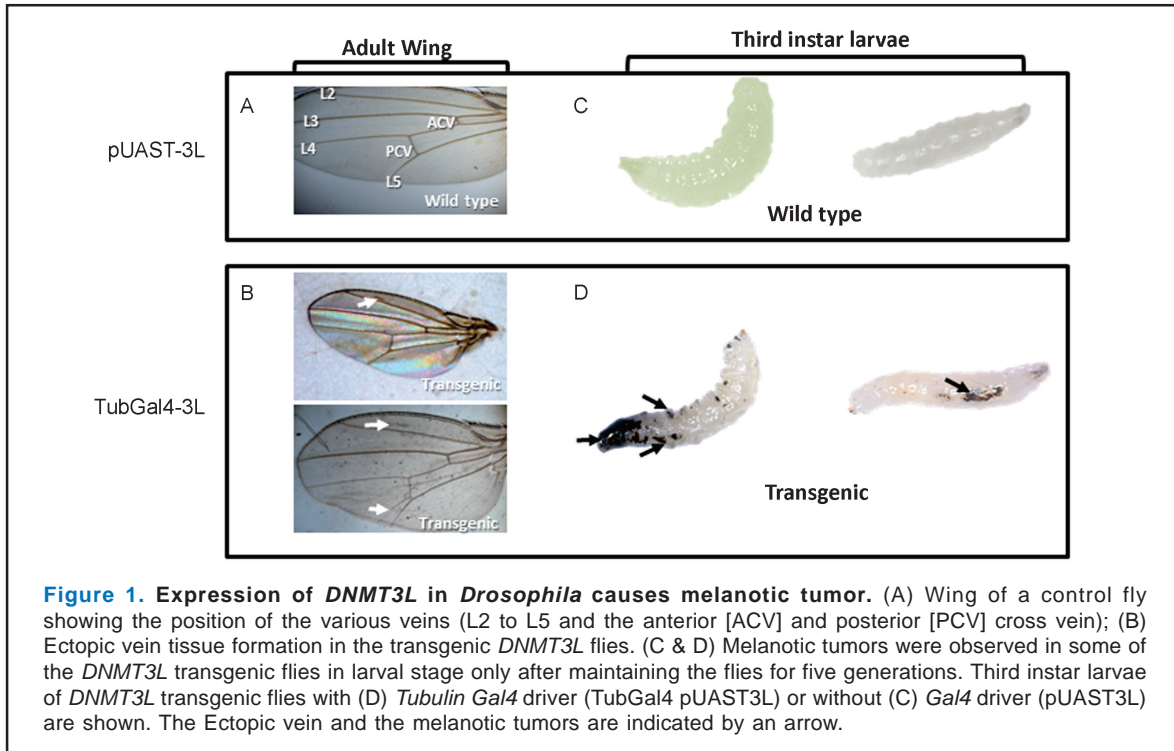
We had shown by transgene reporter assay in *Drosophila* and by mammalian transient transfection reporter gene assay that the CpG island spanning the *DNMT3L* Promoter-Exon 1 behaves as a transcriptional inhibitor. The inhibitory nature of this CpG island was found to be due to its interaction with Polycomb proteins that are known to inhibit transcription. Concordant with the observation of its interaction with Polycomb proteins, our results also showed that this region adopts an inactive chromatin conformation.

Details of progress made in the current reporting year (April 1, 2013- March 31, 2014)

DNMT3L has been shown to influence DNA methylation by stimulating the activity of DNMT3A and DNMT3B through protein-protein interaction. Reports also suggest that *DNMT3L* can interact with Histone H3 at Lysine 4 only when it is unmethylated. Thus *DNMT3L* has been suggested to be a reprogramming molecule that can regulate *de novo* DNA methylation as well as respond to the status of histone modifications. It has been our endeavor to understand the regulatory influence of *DNMT3L*'s interaction with DNMT3A/DNMT3B on one hand and with Histone H3 at lysine 4 on the other.

In *Drosophila*, epigenetic circuitry consists of histone modifications but DNA methylation is absent or present at very low levels and its effector molecules including DNMT1, DNMT3A AND DNMT3B are missing from the genome. Since *DNMT3L* can directly bind histone tails to read histone modifications we wanted to examine through the *Drosophila* system whether epigenetic changes brought by DNMT3L through its interactions with histone tails alone could influence the epigenetic circuitry.

To examine this, transgenic *Drosophila* expressing *DNMT3L* were generated. 7 independent *DNMT3L*-transgenic lines were obtained. Each of these transgenic *Drosophila* lines was crossed with *Tubulin Gal4* driver flies to obtain flies expressing *DNMT3L* either ubiquitously or in specific tissues. The *DNMT3L*-transgenic *Drosophila* showed wing phenotypes in six of the lines. In the 7th line we found that expression of *DNMT3L* caused lethality (Fig. 1B). To our surprise, we found that when the *DNMT3L* expressing transgenic *Drosophila* lines were maintained over several generations, some of the flies showed melanotic tumors (Fig. 1D). This delayed effect of *DNMT3L* expression in *Drosophila* was similar to what we had previously observed. HeLa cells overexpressing *DNMT3L* were found to have undergone nuclear reprogramming gradually and showed morphological changes only in the 20th generation post transfection of *DNMT3L* construct (Gokul *et al.*, 2009; Epigenetics 4: 322-329).



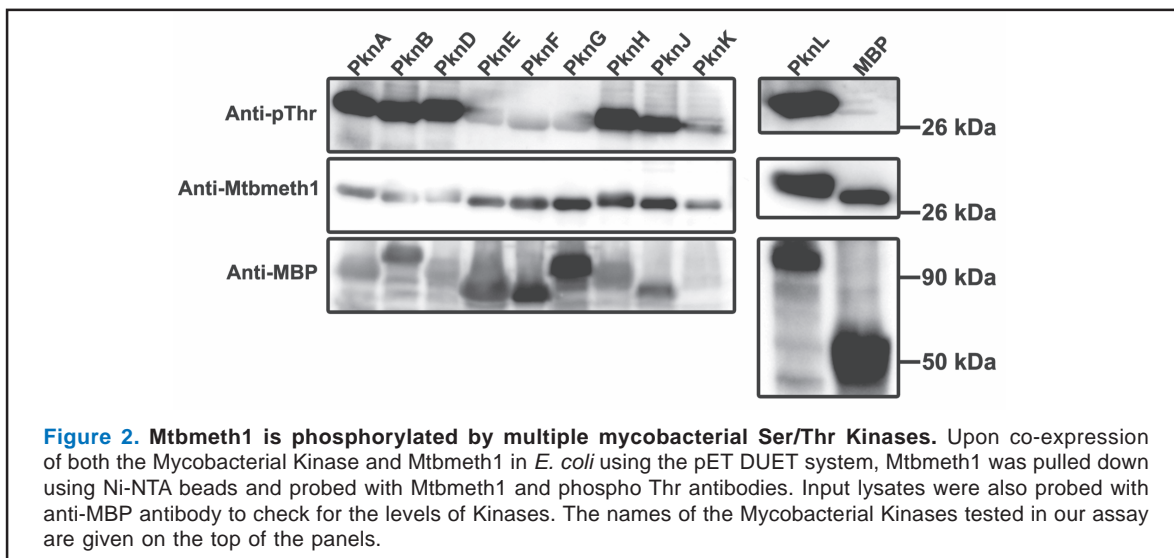
Project 2: Host epigenetic response to infection

Summary of work done until the beginning of this reporting year (up to March 31, 2013)

We have previously identified putative DNA methyltransferases (DNMTs) in the *Mycobacterium tuberculosis* based on a combination of bioinformatics analysis and DNA methylation assay. We had also showed that one of the DNMTs, which we had termed as Mtbmeth1, is secreted out of BCG and our transient transfection assay showed that it can localize to the THP1 nucleus.

Details of progress made in the current reporting year (April 1, 2013- March 31, 2014)

The endogenous Mtbmeth1 protein from *M. tuberculosis* H37Rv and *M. bovis* BCG showed anomalous migration on a SDS-PAGE. Instead of the expected molecular weight of approximately 19.8 kDa, the endogenous protein was detected as a 34 kDa. Since post translational modifications are known to influence protein activity as well as its mobility on a SDS-PAGE, we investigated whether Mtbmeth1 indeed was modified. Protein



phosphorylation of a protein by Ser/Thr kinases (STKs) is one of the most common modifications implicated in anomalous migration of proteins. Therefore, we tested whether MtbMeth1 gets phosphorylated at any of the 16 Threonine present in the protein. His-tagged MtbMeth1 gene was cloned into pET-DUET vector that also contained MBP-tagged mycobacterium kinases (there are 11 eukaryotic-like Ser/Thr kinases in the mycobacterial genome; 21). His-MtbMeth1 was affinity purified using Ni-NTA column from the *E.coli* protein lysate and probed for Threonine phosphorylation. MtbMeth1 was found to be a substrate for multiple kinases, namely, PknA, PknB, PknD, PknH and PknL (Fig. 2). This was further confirmed by mass spectrometry analysis of MtbMeth1 incubated with PknB protein that prefers T over S as the phospho-acceptor. Interestingly, the same peptide also contained the catalytic cysteine (C156) residue. Furthermore, phosphorylation of Mtbmeth1 was found to modulate its DNA binding and methylation activity.

Details of progress made in the current reporting year (April 1, 2013- March 31, 2014)

Gene expression analysis of mammalian cell lines upon *Dnmt2* overexpression in our previous study had shown misregulation of several genes involved in host response to viral infection. To examine whether viral infection has an effect on the expression and localization of *Dnmt2*, CEMX174 cells were infected with HIV1 and the localization of endogenous *Dnmt2* was observed post infection. As can be seen in Figure 3, Dnmt2 protein re-localizes to the cytoplasm from the nucleus post infection. This observation was similar to what was previously observed for localization of *Dnmt2* in cells under stress (Thiagarajan *et al.*, 2011; Epigenetics 6:103-113).

Publications

1. Basu A, Dasari V, Mishra RK, Khosla S. The CpG island encompassing the promoter and first exon of human DNMT3L gene is a PcG/TrX response element (PRE). *PLoS One* (In press)

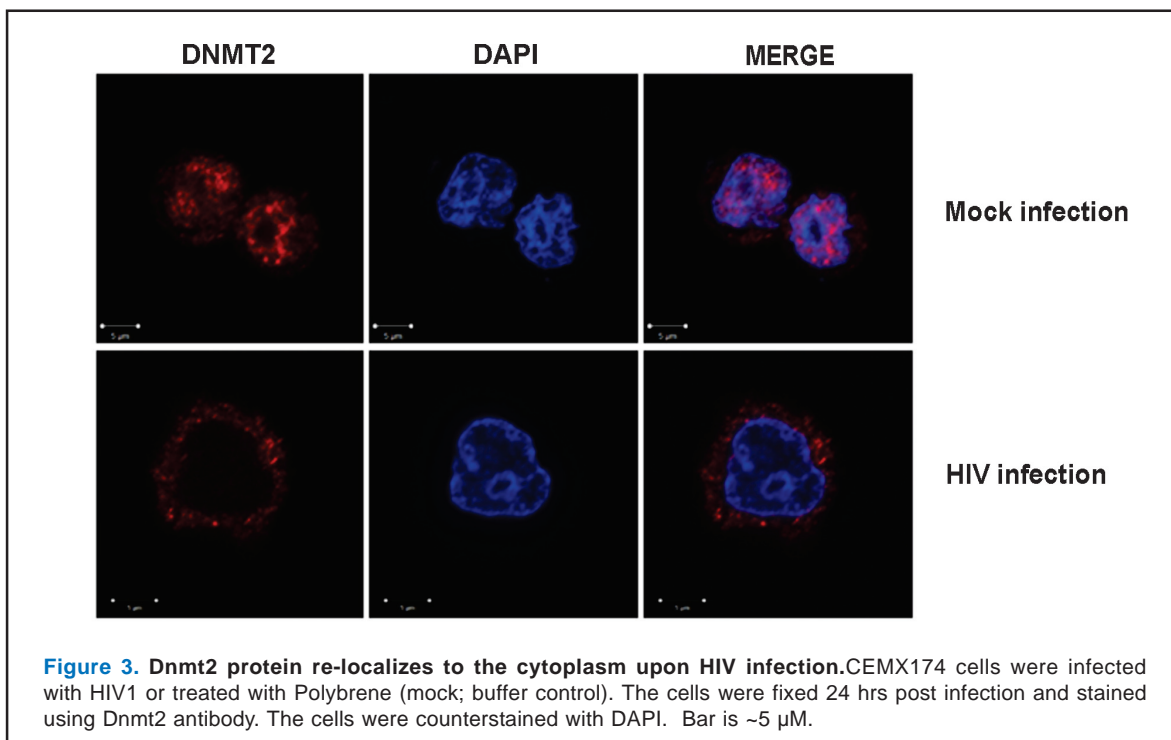


Figure 3. Dnmt2 protein re-localizes to the cytoplasm upon HIV infection. CEMX174 cells were infected with HIV1 or treated with Polybrene (mock; buffer control). The cells were fixed 24 hrs post infection and stained using Dnmt2 antibody. The cells were counterstained with DAPI. Bar is ~5 µM.

Project 3: Role of *Dnmt2* in RNA processing

Summary of work done until the beginning of this reporting year (up to March 31, 2013)

Previous studies from our laboratory has shown that the DNA methyltransferase *Dnmt2* is involved in RNA processing during cellular stress.

LABORATORY OF MOLECULAR CELL BIOLOGY

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

Faculty	Sangita Mukhopadhyay	Staff Scientist
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	Gourango Pradhan	Senior Research Fellow
	Parul Singh	Senior Research Fellow
	Vishwanath Jha	Senior Research Fellow
	D Komal Chandresh Kumar	Junior Research Fellow
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	Niteen R Pathak	Senior Technical Officer
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	Asma Ahmed	DBT Research Associate
	Khalid Hussain Bhat	Research Associate
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	Anand Kondapi	UoH, Hyderabad

Objectives

1. Signal transduction pathways in macrophages regulating its innate-effector immune responses; and
2. Studying how various candidate proteins of *Mycobacterium tuberculosis* interfere with macrophage signaling cascades to modulate host's protective responses against the bacilli.

Project 1: Effect of reactive oxygen species on macrophage signalosome: impact on antigen presentation functions and T cell priming responses.

Summary of work done until the beginning of this reporting year (upto March 31, 2013)

Earlier studies by us reveal that the reactive oxygen species (ROS) downregulate interleukin (IL)-12 production in activated macrophage by inhibiting nuclear translocation of c-rel transcription factor involving the Calmodulin (CaM) protein (*Khan et al.[2006]Blood, 107:1513*). Since T cell proliferation is influenced to a great extent by the peptide-major histocompatibility complex (MHC)-driven stimulus

and the costimulatory signal of macrophages, it is possible that ROS can also affect T cell proliferation by modulating these stimuli in addition to interfering with IL-12 production. Since macrophages represent one of the major APCs that regulate both the magnitude and the effector phenotypes of T cell responses by controlling three signals, viz., the cognate peptide-MHC-driven stimulus, the non-cognate costimulatory stimulus, and the production of cytokine/effector molecules, it was proposed that ROS could affect the T cell responses by modulating these signalings emanating from macrophages.

Details of progress made in the current reporting year (April 1, 2013 - March 31, 2014)

a. Exogenously added H₂O₂ inhibit MHC class II presentation of exogenous OVA antigen:

Antigen presentation assay was carried out *in vitro* using ovalbumin (OVA)-specific I-A^b-restricted T cell line 13.8 (a kind gift from Drs. Satyajit Rath and Vineeta Bal, National Institute of Immunology, India) and thioglycolate elicited peritoneal macrophages (*Parveen et al.[2013]J Biol.*

Chem.,288:24956) from C57Bl/6 mice (H-2^b) as antigen presenting cells (APCs). The macrophages were pre-treated with various concentrations of H₂O₂ (25, 50 and 100 μM) for 1 h and pulsed exogenously with titrating concentrations of soluble OVA antigen (Ag) for a fixed time of 3 h in the presence of H₂O₂. Cells were washed and fixed with paraformaldehyde and cultured (1-2 X 10⁵) in the presence of 13.8 T cells (1 X 10⁵/well) in 200 μl of Dulbecco Minimal Essential Medium (DMEM) containing 10% fetal calf serum and antibiotics (DMEM-10) in 96-well tissue culture plate. After 24 h, IL-2 levels secreted in the culture supernatants were examined by enzyme immunoassay (EIA) as a direct measurement for T cell priming and APC function of macrophages. Comparisons with proliferation induced by OVA-pulsed APCs indicate that although H₂O₂ at 100 μM did not affect viability of macrophages (Fig. 1A), MHC class II-restricted antigen presentation was inhibited by H₂O₂ in dose dependent manner (Fig. 1B) when compared with untreated macrophages.

b. H₂O₂ inhibits antigen processing:

Next we incubated the untreated and H₂O₂-treated macrophages with a fixed concentration of OVA Ag (300 μg/ml) for a fixed time period of 30 min, washed and allowed the OVA antigen to process for different time points in the presence of 100 μM H₂O₂ and observed whether antigen processing is affected by H₂O₂ which in turn can interfere with peptide loading on class II molecules and presentation to 13.8 CD4 T cells. It was observed that antigen presentation was inhibited by H₂O₂ when added at 100 μM concentration (Fig. 1C) indicating that probably H₂O₂ affects antigen processing.

c. H₂O₂/ROS at physiological concentration also inhibits MHC class II-restricted antigen presentation and macrophage APC functions:

We were next interested to check whether the physiological level of H₂O₂/ROS produced by activated macrophages during respiratory burst can also show a direct inhibitory effect on antigen presentation. The peritoneal macrophages from C57Bl/6 mice were therefore stimulated with LPS to activate H₂O₂/ROS production in macrophages. In one group, 100 μM NAC was used to scavenge endogenous H₂O₂/ROS (*Khan et al.[2006]Blood, 107:1513*). After 6 h, macrophages were washed and treated for 30 min with OVA antigen and allowed it to process for 3 h. After fixing with paraformal-

dehyde, the cells were cultured for 24 h along with 13.8 T cells. It could be observed that LPS treatment causes inhibition of OVA antigen presentation as measured by IL-2 level produced by the 13.8 T cells and scavenging the H₂O₂/ROS by NAC improved antigen presentation function of macrophages (Fig. 1D). This results further confirm the fact that reactive oxygen species particularly the H₂O₂ is involved in decreasing antigen presentation function of macrophages.

d. Exogenous H₂O₂ does not alter surface expression of the co-stimulatory molecules in peritoneal macrophages:

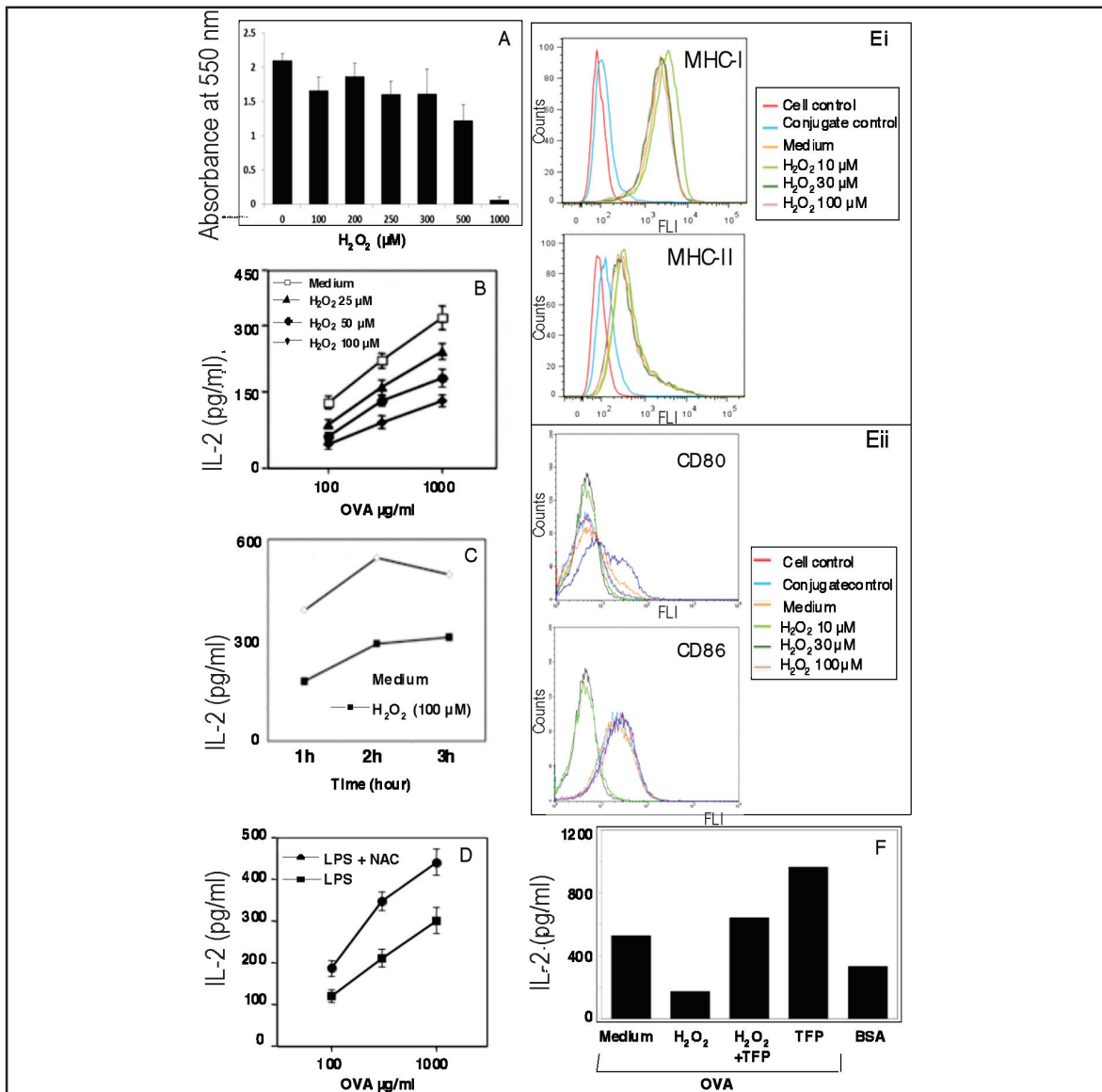
Since, MHC as well as co-stimulatory molecules of macrophages strongly influences T cell priming responses, we next checked whether H₂O₂ downregulates levels of these molecules to affect T cell proliferation. Therefore, thioglycolate elicited peritoneal macrophages from C57Bl/6 mice were either left untreated or pre-treated with different concentrations of H₂O₂ for 1 h. After 24 h, cells were stained with antibody to MHC-I, MHC-II, CD80 and CD86 followed by incubation with FITC-labeled appropriate secondary conjugate. Cells were washed and fluorescence was measured using flow cytometry. It was observed that H₂O₂ did not alter the surface expression of MHC-I, MHC-II, CD80 and CD86 co-stimulatory molecules (Figs. 1Ei and 1Eii) indicating that exogenous H₂O₂/ROS does not significantly affect the co-stimulatory signaling to inhibit antigen presentation. The surface expression of CD40 and ICAM-1 was also not modulated by H₂O₂.

e. Role of calmodulin (CaM) in H₂O₂ mediated inhibition of antigen presentation:

It was demonstrated earlier that inhibition of IL-12 p40 induction by H₂O₂ involves role of calmodulin (CaM). H₂O₂ increases CaM expression which then binds and sequesters c-rel transcription factor in the cytoplasm inhibiting its translocation to nucleus and binding to specific promoters (*Khan et al.[2006]Blood, 107:1513*). We observed an important contribution of the c-rel transcription factor in H₂O₂-mediated downregulation of antigen presentation. Since nuclear c-rel translocation in the H₂O₂-treated macrophages was found to be under the control of CaM protein, a role of CaM in the H₂O₂-mediated downregulation of antigen presentation was speculated. Therefore, in the next experiment, we used trifluoperazine (TFP), a known agonist of CaM. Peritoneal macrophages from C57Bl/6 mice were either left untreated or pre-

treated with TFP for 30 min followed by incubation with H₂O₂ (100 μM). The macrophages were then pulsed exogenously with OVA (1 mg/ml) or BSA (as control) for 30 min, followed by chasing for 3 h. The cells were then fixed, washed and incubated with 13.8 T cells for 24 h and IL-2 levels were

measured by EIA. The results indicate that TFP treatment increased antigen presentation of OVA by H₂O₂-treated macrophages (Fig. 1F) indicating a role of CaM in downregulation of antigen presentation by H₂O₂/ROS.



Future Plans:

We have highlighted the role of ROS/H₂O₂ to inhibit antigen presentation of exogenously delivered OVA antigen. We aim to study whether H₂O₂ also modulate cytosolic OVA presentation and CD8 T cell priming responses and whether the *M. tuberculosis* protein(s) modulates macrophage antigen presentation function by targeting the CaM-c-rel signaling cascades.

Project 2: Signaling pathways involved in downregulation of proinflammatory responses by PPE18 protein of *Mycobacterium tuberculosis*: Implication of PPE18 as therapeutic for microbial sepsis.

Summary of work done until the beginning of this reporting year (upto March 31, 2013)

We observed earlier that the PPE18 protein of *M. tuberculosis* targets the p38 MAPK-SOCS3 signaling to downregulate nuclear translocation of NF- κ B/rel transcription factors resulting in suppression of proinflammatory cytokines like IL-12 and TNF- α (Nair et al.[2011]J. Immunol., 186:5413). We observed that PPE18 binds to TLR2 and increased expression and at the same time, induced tyrosine phosphorylation of SOCS3 (suppressor of cytokine signaling 3) which then physically interacts with I κ B α -NF- κ B/rel complex, inhibiting phosphorylation of I κ B α at the serine 32/36 residues by I κ B kinase (IKK)- α/β , and thereby prevents nuclear translocation of the NF- κ B/rel subunits in lipopolysaccharide (LPS)-activated macrophages suppressing activation of IL-12/TNF- α cytokines. Further, we demonstrated that SOCS3-mediated regulation of I κ B α phosphorylation is not dependent on its direct effect on the I κ B α kinases in PPE18-treated macrophages.

Details of progress made in the current reporting year (April 1, 2013 - March 31, 2014)

PPE18 is shown to upregulate IL-10 and has the ability to skew T cell responses towards the T-helper 2 type (Nair et al.[2009]J. Immunol., 183:6269). Also, PPE18 downregulates LPS induced TNF- α and IL-12 (Nair et al., 2011). During *M. tuberculosis* infection, the PPE18 which selectively reduce levels of IL-12/TNF- α and the T-helper (Th) 1 response on the whole may help in survival of the bacilli. However, the same properties of PPE18 can be exploited to dampen effects of extreme inflammation. A good example of extreme inflammation is sepsis that occurs either due to uncontrolled microbial infection or exposure to microbial products such as LPS. It is a major cause

of mortality in hospitals. The excessive production of inflammatory mediators, the primary being TNF- α is responsible for the pathophysiology of sepsis which includes cardiac dysfunction, hypotension and multiple organ failure. Therefore, blockage of TNF- α is crucial for effective therapy in cases of sepsis. With this rationale, we decided to test the ability of PPE18 to reduce TNF- α and IL-1 β in a mouse model of septic shock.

PPE18 reduces levels of TNF- α and IL-1 β cytokines in a mouse model of *E coli*-induced septicemia:

The commonly used laboratory strain of *E. coli*, BL21 when injected at high doses causes peritonitis and septic shock with increased serum TNF- α and IL-1 β levels as early as 3 h. Since, 2.5 X 10⁸ cfu did not result in death of mice till 24 h, this was the dose selected for future experiments. Mice were administered with PBS or 100 μ g of recombinant PPE18 (rPPE18) 1 h prior to being infected with 2.5 X 10⁸ *E. coli*. A significant reduction in TNF- α (Figs. 2Ai and 2Aii) and IL-1 β (Figs. 2Bi and 2Bii) levels in rPPE18 pre-treated mice was observed in sera (Figs. 2Ai and 2Bi) and peritoneal lavages (Figs. 2Aii and 2Bii) at 3 h and 24 h (data not shown) after induction of peritonitis when compared with the control that received only PBS.

One of the organs that is majorly affected during sepsis is liver. Liver damage results in rise of serum alanine aminotransferase (ALT) levels. Therefore, serum ALT levels will be measured with the help of a commercial kit to assess liver damage resulting from sepsis. The results indicate that pre-treatment of mice with 100 μ g rPPE18 protected liver damage in mice injected with 2.5 X 10⁸ *E. coli* as ALT level was significantly reduced in the PPE18-treated mice when compared with the control that received only PBS (Fig. 2C).

PPE18 protein treated animals appear clinically healthier after induction of peritonitis:

Mice suffering from peritonitis exhibit clinical features which are reflective of the septic shock that they are experiencing. These clinical features can be graded to reflect the severity of septic shock. Mice which received PBS as opposed to those which received rPPE18 prior to induction of peritonitis (injected with 2.5 X 10⁸ *E. coli*) were found to be lethargic and less alert, had open eyes with serious discharge and had rough hair coat. The rPPE18 treated mice were relatively active and more alert, had normal eyes, relatively shiner and

groomed hair coat suggesting that sepsis was less severe in them.

rPPE18 Experiments carried out so far showed that PPE18 could reduce severity of *E. coli* induced sepsis. Whether this translated to a survival advantage in mice which received PPE18 remained to be investigated. Therefore, survival of Balb/c mice injected with 2.5×10^8 of *E. coli* strain was monitored. The results indicate that mice received rPPE18 survived for longer period compared to the control mice which received only PBS before induction of septicemia (Fig. 2D). This suggests

that administration of rPPE18 may confer survival advantage during septicemia.

Future plans:

We aim to study whether PPE18 can induce alternate activation of macrophages which perhaps provide protection during peritonitis/shock. Also we will be testing the therapeutic effect of PPE18 in the mouse model of septic shock as well as if PPE18-encapsulated nanoparticles can be used to reduce symptoms of endotoxemia as nanoparticles are delivery systems that enhance stability.

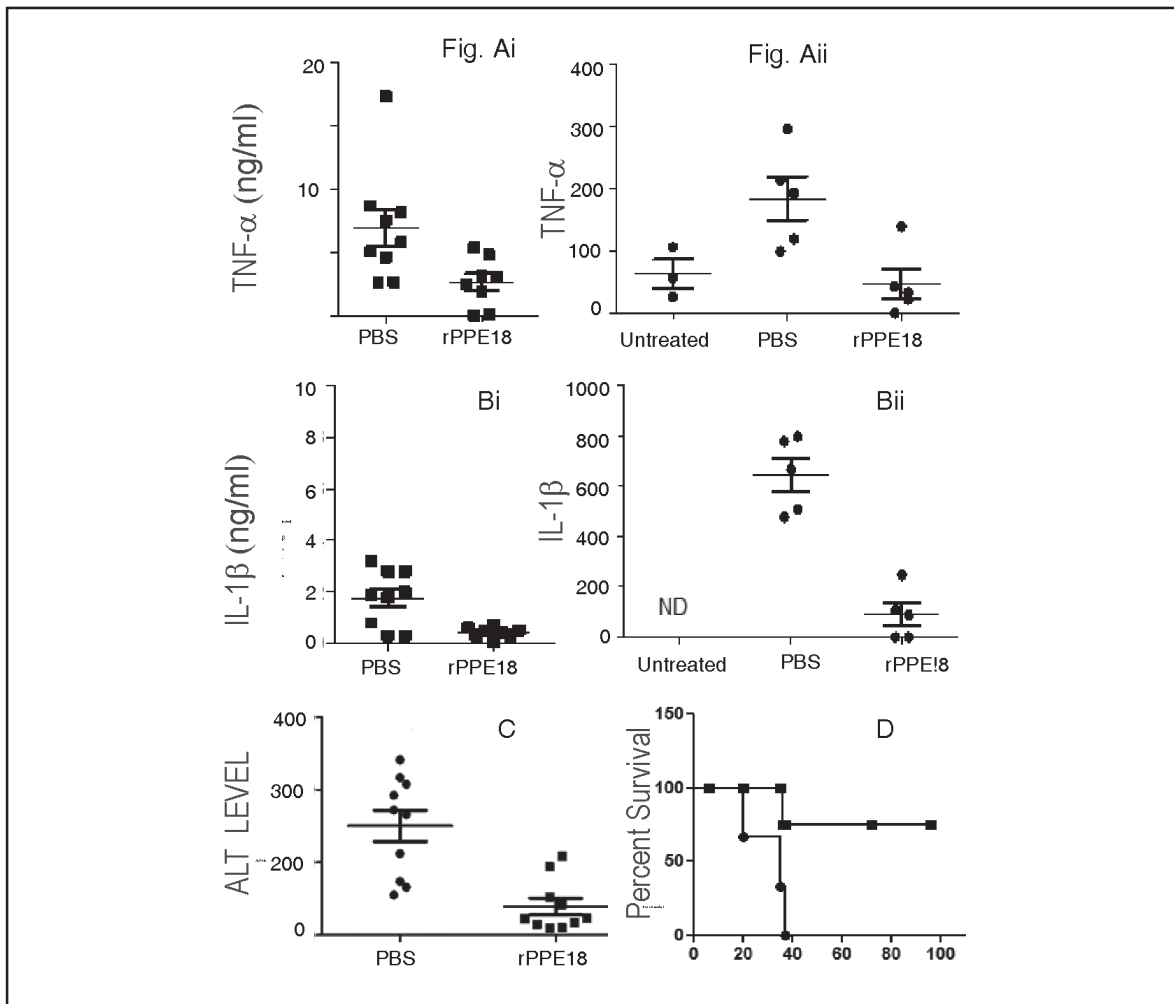


Figure 2. PPE18 reduces levels of inflammatory cytokines (TNF- α and IL-1 β) and increases survival time in mice subjected to *E. coli* induced septic shock in mice. Balb/c mice were given either PBS or 100 μ g of recombinant PPE18 (rPPE18) intraperitoneally prior to infection with 2.5×10^8 *E. coli* BL21 cfu. Mice were bled retro-orbitally 3 h post induction of peritonitis. TNF- α (Ai) and IL-1 β (Bi) levels were measured in the sera by ELISA. At 24 h mice were sacrificed by CO₂ inhalation and peritoneal lavages were collected and TNF- α (Aii) and IL-1 β (Bii) levels in the lavage were measured by ELISA. Serum ALT levels were compared between the rPPE18-treated and PBS-treated mice after induction of peritonitis with 2.5×10^8 *E. coli* (C). Survival of Balb/c mice injected with 2.5×10^8 of *E. coli* strain was monitored for about 100 days post injection (D). ND indicates 'Not detected by ELISA'. Student's t test was used to determine P values.

Publications

1. Bhat KH, Das A, Srikantam A and Mukhopadhyay S (2013). PPE2 protein of *Mycobacterium tuberculosis* may inhibit nitric oxide in activated macrophages. ***Annals of the New York Academy of Sciences*** 1283: 97-101.
2. Parveen N, Varman R, Nair S, Das G, Ghosh S and Mukhopadhyay S (2013). Endocytosis of *Mycobacterium tuberculosis* heat shock protein 60 is required to induce interleukin-10 production in macrophages. ***Journal of Biological Chemistry*** 288: 24956-24971.
3. Abraham PR, Latha GS, Valluri VL and Mukhopadhyay S (2014). *Mycobacterium tuberculosis* PPE protein Rv0256c induces strong B cell response in tuberculosis patients. ***Infection Genetics and Evolution*** 22: 244-249.

Patents

1. Mukhopadhyay S, Bhat KH and Khan N (2013). A novel protein as potential candidate for development anti-tuberculosis therapeutics.
US Patent Application No.: US-12/551,115
Invention ID: IN-000044-02-US-REG
Patent No.: US-8603739B2 Date of grant: December 10, 2013

LABORATORY OF MOLECULAR GENETICS

(Explanatory note: The Laboratory of Molecular Genetics, which also includes the Centre of Excellence (CoE) in Silkworm 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).

A. Report of Dr KP Arun Kumar's group

Faculty	KP Arun Kumar	Scientist
PhD Students	Asha Minz	Senior Research Fellow
	Chandra Pal Singh	Senior Research Fellow (Till Nov. 2013)
	S Suresh Kumar	Senior Research Fellow
	G Gopinath	Senior Research Fellow
Other Members	Varsha	Staff Scientist (Till Dec. 2013)
	A Sobhan Babu	Technical Officer (Till Nov. 2013)
	M Muthulakshmi	Technical Officer (Till Feb. 2014)
	S Annapurna Bhavani	Technical Officer
	Archana Tomar	Bioinformatician (Till Nov. 2013)
	R Lakshmi Vaishna	Technical Assistant
	MJ Reddy	Technical Assistant (Till Nov. 2013)
	CVE Rajendra	Research Associate (Since May 2013)
	Adarsh Gupta	Project Assistant
	Kushal Ravindra Kekan	Project Assistant (Since May 2013)
	Saikat Chakraborty	Project-Junior Research Fellow
K Srikeerthana	Research Associate (Since Feb. 2014)	

Objectives

1. Functional characterization of sex-determination genes in *B. mori*: comparative studies on gonad specific genes;
2. Characterization of antiviral and antibacterial activity of *Bombyx mori* seroin proteins; and
3. Studies on host pathogen interactions as mediated by microRNAs (miRNAs).

Summary of the work done until the beginning of this reporting year (upto March 31, 2013)

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

Details of progress made in the current reporting year (April 1, 2013 - March 31, 2014)

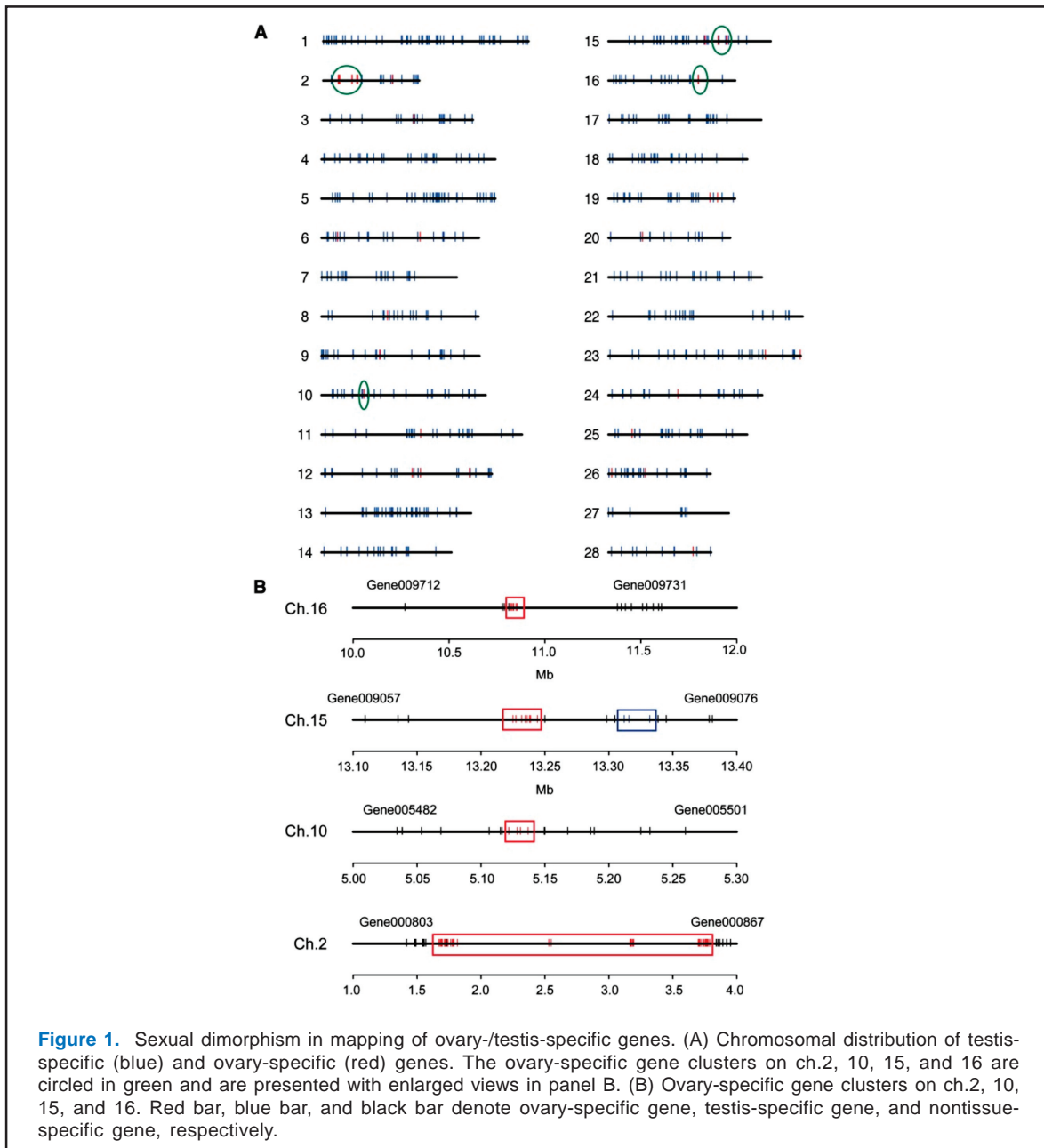
Objective 1: Functional characterization of sex-determination genes in *B. mori*: comparative studies on gonad specific genes.

The sex chromosomes, apart from their primary role in sex determination, are also involved in

sexual dimorphism, and their genetic landscape has undergone distinctive changes in the course of evolution compared to autosomes. Interesting findings on the organization of genes with sex-limited expression have emerged from the analysis and mapping of 11,104 FL-cDNAs in the silkworm. We found that the Z chromosome is conspicuous by the absence of any female-enhanced or female-biased genes; in other words, it is heavily defeminized (Fig. 1). In contrast, it is enriched in male-specific and male-biased genes as reported previously by our group. Recent studies suggest that similarities and dissimilarities exist within and between male and female heterogametic systems. For example, the chicken Z chromosome is known to harbor a massive tandem array of testis-specific genes, and clustering of testis-specific genes is also observed on human and mouse X chromosomes. However, although the present analysis revealed clusters of ovary-specific genes on autosomes, no clusters of tandemly duplicated testis-specific genes were found on the *B. mori* Z chromosome or autosomes.

Female-biased genes located on the Z chromosome are underrepresented among those expressed in the germline of the chicken. This may result from the inactivation of sex chromosomes during female meiosis, referred to as female MSCI, which was first reported in this species. Although the presence of MSCI has not been reported in silkworm, we cannot rule out the existence of such a phenomenon. We therefore speculate that the observed depletion of ovary-specific genes on the silkworm Z chromosome may result in part because of silencing of sex chromosomes during female meiosis due to MSCI.

Unlike many animal species where male heterogamety prevails (e.g., mammals and dipterans), *B. mori* has a female heterogametic sex chromosome system. Silkworm also lacks sex chromosome dosage compensation. This makes the Z chromosome a favorable place for male advantageous genes, as genes on Z are expressed in a double dose in males, which also carry 2/3 of the Z chromosomes present in a population. Therefore, it is possible that the observed depletion of female-enhanced genes on the Z may be preferred in part because of disadvantages

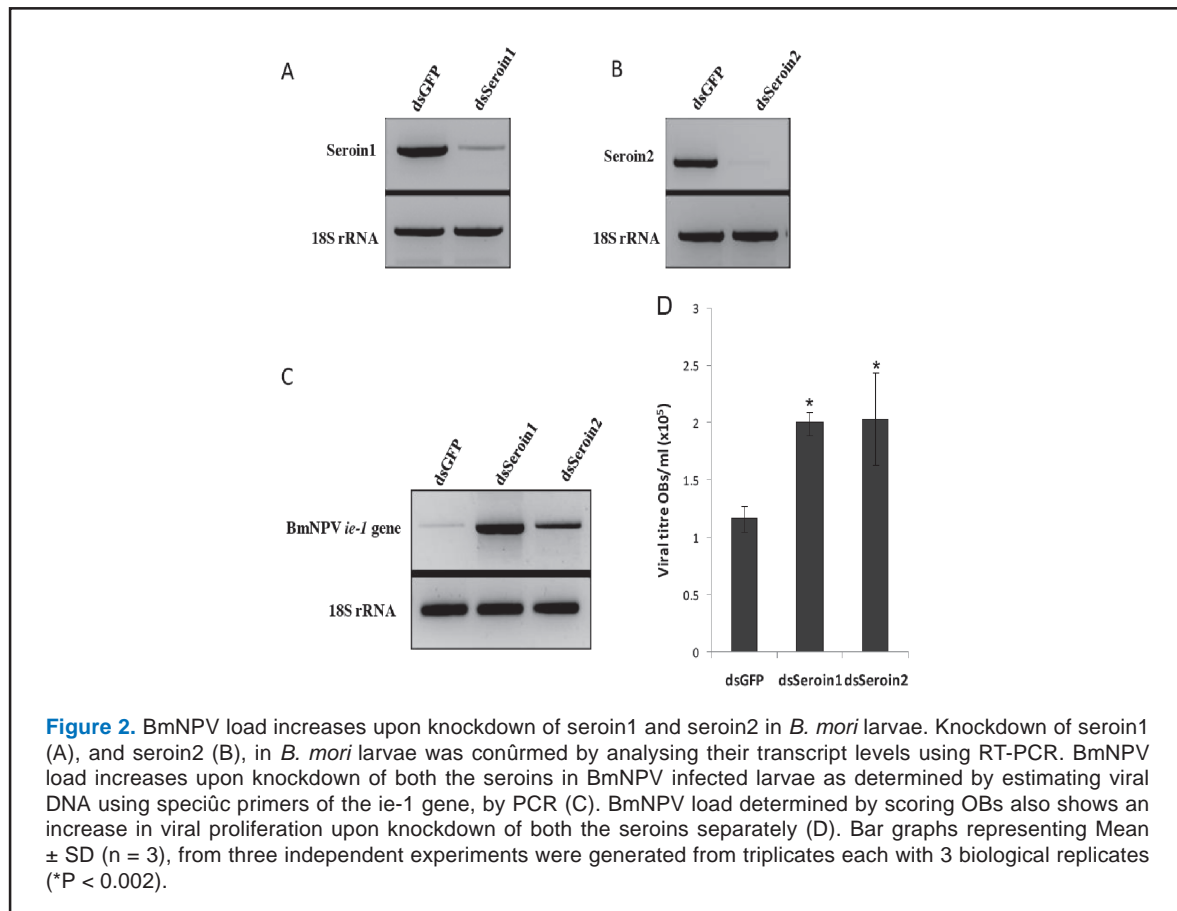


experienced in females from their hemizygous condition and an absence of dosage compensation.

Objective 2: Characterization of antiviral and antibacterial activity of *Bombyx mori* seroin proteins.

Insects possess very potent innate immune system to prevent various pathogenic infections but unlike vertebrate they lack memory based adaptive immune system. The innate immune system of insects basically comprises cellular and humoral defense mechanisms against microbes. Pathogen recognition receptors of insects can directly bind microbes and kill them by encapsulation followed by phagocytosis. Alternatively, upon binding they can induce downstream signaling factors, which in turn provoke production of antimicrobial proteins, nodule formation, and melanization in specific tissues such as the fat body and hemocytes. In lepidopteran insects, not much is known about the defence mechanisms against viral pathogens, such as baculoviruses. Here we show that small silk proteins of the domesticated silkworm, *Bombyx mori* called seroins, act as antiviral agents against

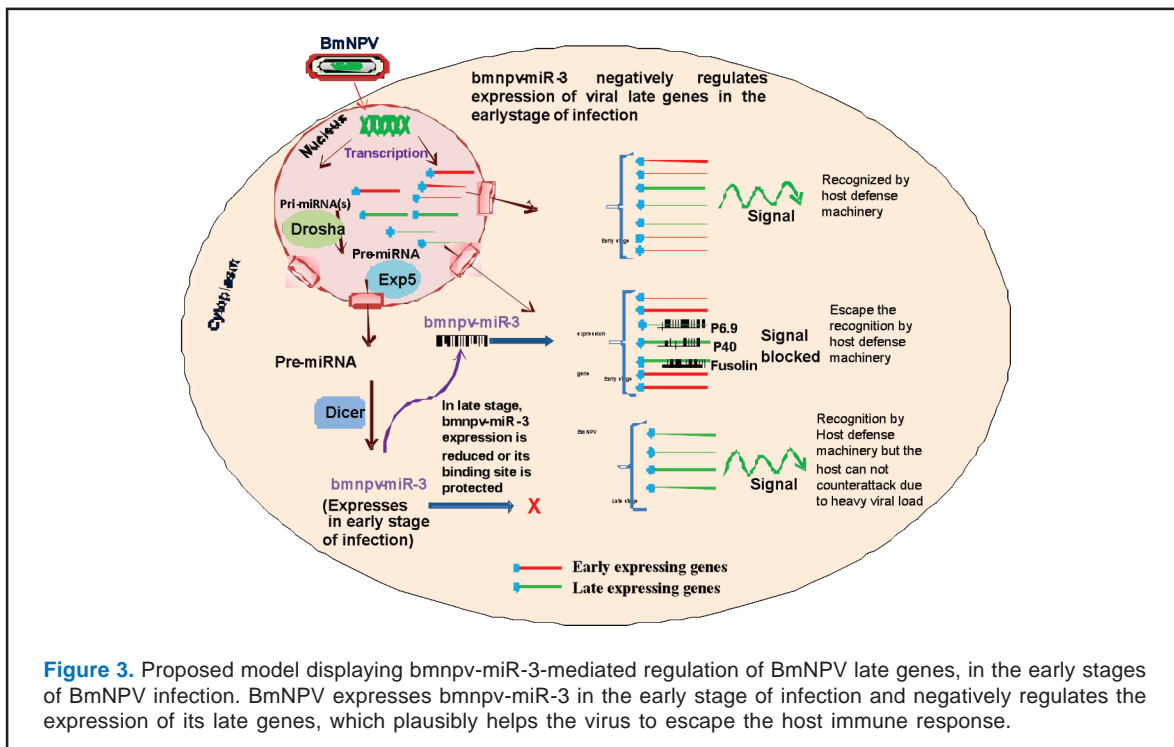
a baculovirus pathogen, *Bombyx mori* nucleopolyhedrosis Virus (BmNPV). We also show that seroins are also potentially capable of eradicating bacterial infections. Involvement of these proteins in inhibition of baculovirus infection was revealed by estimating virus load upon their dsRNA-mediated knockdown (Fig. 2). Additionally, we found that Seroins are potent inhibitors of bacterial growth, as shown by antimicrobial assays. Binding competition followed by antimicrobial assays showed that seroins bind cell wall components of bacteria to prevent their proliferation. Broad-spectrum antimicrobial nature of seroins prompted us to investigate the pathway(s) which Seroins may follow. We found Toll receptors of *B. mori* are also involved in antiviral response, and Seroins are likely to be downstream components of Toll pathway, demonstrated by knockdown of Toll receptors followed by measuring viral titre and seroins expression. Our results provide strong evidences to describe Seroins as novel antimicrobial proteins, which are involved in defense against virus as well as bacteria. Hence, seroins can be used as potent inhibitor of microbes.



Objective 3: Studies on host pathogen interactions as mediated by microRNAs (miRNAs)

During the last decade, microRNAs (miRNAs) have emerged as fine tuners of gene expression in various biological processes including host-pathogen interactions. Apart from the role of host encoded miRNAs in host-virus interactions, recent studies have also indicated the key role of virus-encoded miRNAs in the regulation of host defense responses. In the present study, we show that *bmnpv-miR-3*, a *Bombyx mori* nucleopolyhedrovirus (BmNPV) encoded miRNA, regulates the expression of DNA binding protein (P6.9) and other late genes, vital for the late stage of viral infection in the host, *Bombyx mori*. We have performed both cell culture and *in vivo* experiments to establish the role of *bmnpv-miR-3* in the infection cycle of BmNPV. Our findings showed that *bmnpv-miR-3*

expresses during early stage of infection, and negatively regulates the expression of P6.9. There was an upregulation in P6.9 expression upon blocking of *bmnpv-miR-3* by Locked Nucleic Acid (LNA), whereas overexpression of *bmnpv-miR-3* resulted in a decreased expression of P6.9. Besides, a remarkable enhancement and reduction in the viral loads were observed upon blocking and overexpression of *bmnpv-miR-3*, respectively. Furthermore, we have also assessed the host immune response using one of the Lepidoptera-specific antimicrobial proteins, Gloverin-1 upon blocking and overexpression of *bmnpv-miR-3*, which correlated viral load with the host immune response. All these results together; clearly imply that *bmnpv-miR-3*-mediated controlled regulation of BmNPV late genes in the early stage of infection helps BmNPV to escape the early immune response from the host (Fig. 3).



B. Report of Dr VV Satyavathi's group

Members	VV Satyavathi	Technical Officer
	RM Pavani	Project-Junior Research Fellow (Since Oct. 2013)
	S Babu	CDFD-IKP Fellow (Till Dec. 2013)
	HK Basavaraja	Breeder Consultant CoE
Collaborators	PJ Raju	APSSRDI, Hindupur
	BB Bindroo	CSR&TI, Mysore
	S Nirmal Kumar	CSR&TI, Berhampore
	KA Sahaf	CSR&TI, Pampore
	KI Basha	APSSRDI, Hindupur
	SV Seshagiri	APSSRDI, Hindupur

Objectives

1. Introduction of anti-baculoviral property from transgenic silkworms to commercial silkworm strains followed by limited multilocational field trials;
2. Characterization of *Bombyx mori* nucleopolyhedrosis virus (BmNPV) resistant transgenic silkworm strains;
3. Identification and functional characterization of novel genes involved in immune response pathways of silkmoths; and
4. Development of baculovirus resistant silkworm strains using marker assisted selection.

Summary of the work done until the beginning of this reporting year (upto March 31, 2013)

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

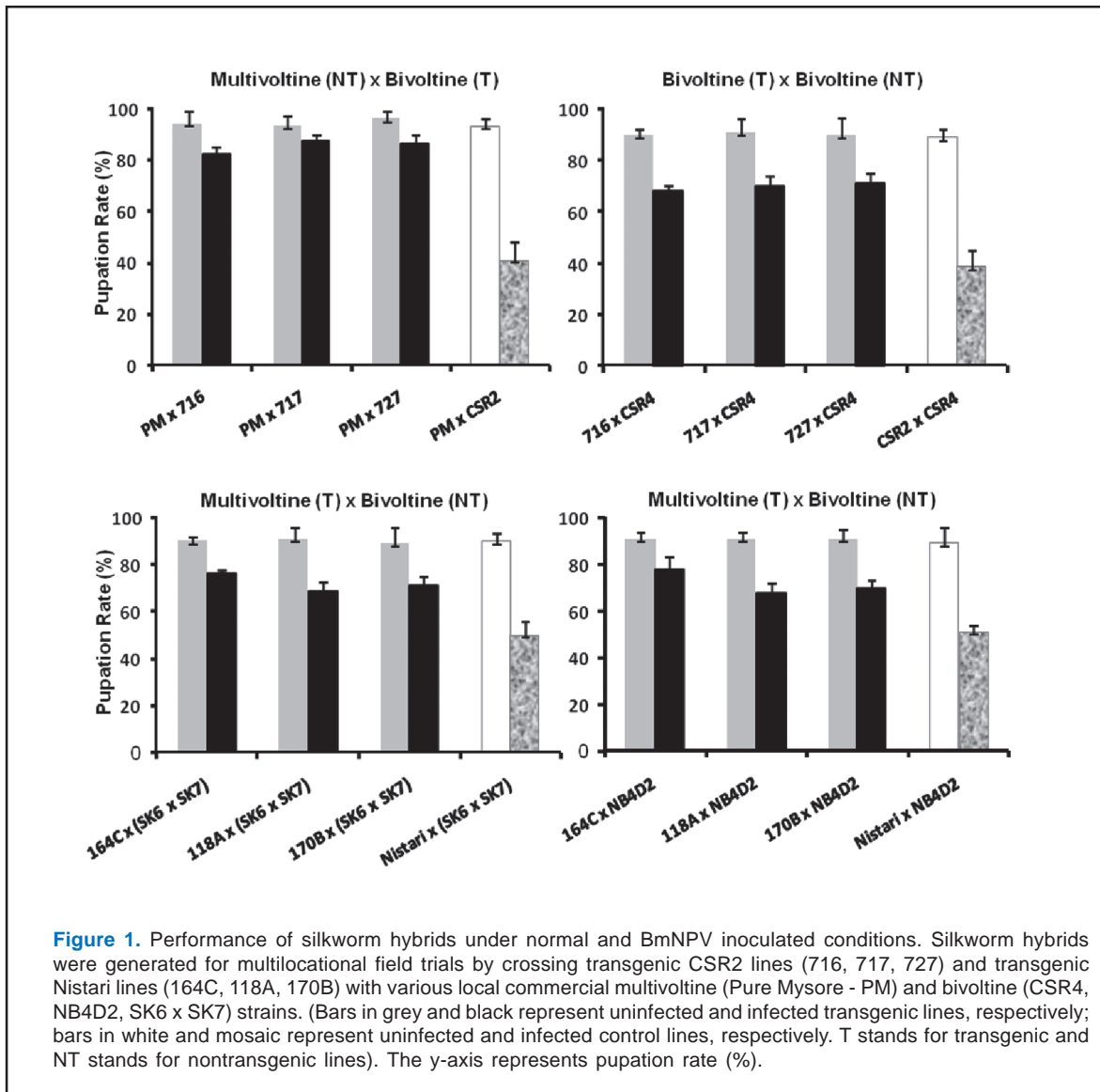
Details of progress made in the current reporting year (April 1, 2013 - March 31, 2014)

Objective 1: Introduction of anti-baculoviral property from transgenic silkworms to commercial silkworm strains followed by limited multilocational field trials.

With an aim of installing a strong antiviral trait, we generated transgenic silkworm harbouring dsRNA-encoding transgenes targeting four essential baculovirus genes namely, *ie1*, *lef1*, *lef3*, and *p74*. Subsequently, the antiviral property of the baculoviral resistant transgenics in the Nistari genetic background was transferred to a high yielding, baculovirus susceptible bivoltine commercial silkworm strain, CSR2 through

transgene (dsRed marker phenotype) selection coupled with microsatellite marker-assisted screening and repeated backcrossing. The recurrent backcrossed lines were advanced to BC₄F₂₇ generation by rigorous selection for various traits such as resistance, cocoon shape, cocoon weight, cocoon shell weight and silk filament length. The transgenic silkworm lines are being maintained at Andhra Pradesh State Sericulture Research and Development Institute (APSSRDI), Hindupur and are being monitored for transgene stability, viral load and unique traits of the strains. During the period under report, hybrids were generated by crossing Nistari and CSR2 transgenic lines with various commercial hybrids and their performance was tested against BmNPV infection (Fig. 1). The best performing breeds were selected for multilocational field trials.

The CDFD in co-ordination with Biotech Consortium India Limited (BCIL) approached Review Committee on Genetic Manipulation (RCGM) by providing a road map for field trials, seeking biosafety regulatory approvals to carry out multilocational contained trials of the transgenic silkworms. RCGM has conveyed its approval for conduct of multilocational field trials in two phases (institutional and farmers level), subject to constitution of IBSCs in each participating institute and constitution of a Co-ordinating Committee. Accordingly, all the participating institutes namely, Andhra Pradesh State Sericulture Research and development Institute (APSSRDI), Hindupur, Central Sericultural Research and Training Institute (CSR&TI), Mysore, CSR&TI, Pampore and CSR&TI, Berhampore have constituted their IBSCs and conveyed their approvals for field trials. As suggested by RCGM, a Coordinating Committee



has also been constituted with members from all participating institutes for effective monitoring of the field trials. The CDFD has submitted a proposal to Biotechnology Industry Research Assistance Council (BIRAC) for funding to the participating institutes to initiate limited contained field trials of transgenic silkworm hybrids, and the proposal has since been recommended for support.

Objective 2: Characterization of *Bombyx mori* nucleopolyhedrosis virus (BmNPV) resistant transgenic silkworm strains.

We observed impaired infectivity of the occlusion bodies (OBs) derived from the transgenic lines expressing dsRNA for multiple essential baculoviral genes (mentioned above) as compared to the OBs

derived from baculovirus infected silkworm strains from farmer's field (wild type). We hypothesized that knockdown of multiple viral genes by dsRNA in the transgenic silkworms would result in less virulent virus particles. We carried out infection experiments with the OBs derived from BmNPV infected transgenic lines and compared infectivity with the wild type OBs. Our results revealed that relative to wild-type OBs, OBs derived from transgenics were both qualitatively and quantitatively different (Fig. 2A). The virus obtained from transgenics failed to compete effectively, and their overall infectivity was significantly less than that of the wild type OBs. Scanning electron microscopy analysis showed structural disturbance on surfaces of the OBs derived from

the transgenic lines (Fig. 2B). Virus growth curves were generated from infection of BmN cells *in vitro* with wild type and transgenic budded virus (BVs). Virus replication kinetics revealed difference in the slope of growth curves between wild type and transgenic BVs (Fig. 2C). However, *in vitro*

experiments revealed that BVs retain their normal infectivity. Overall, the results demonstrated that abrogation of viral genes by the corresponding transgene derived dsRNA would reduce vertical transmission of viral infection conferring an added advantage for large scale field trials.

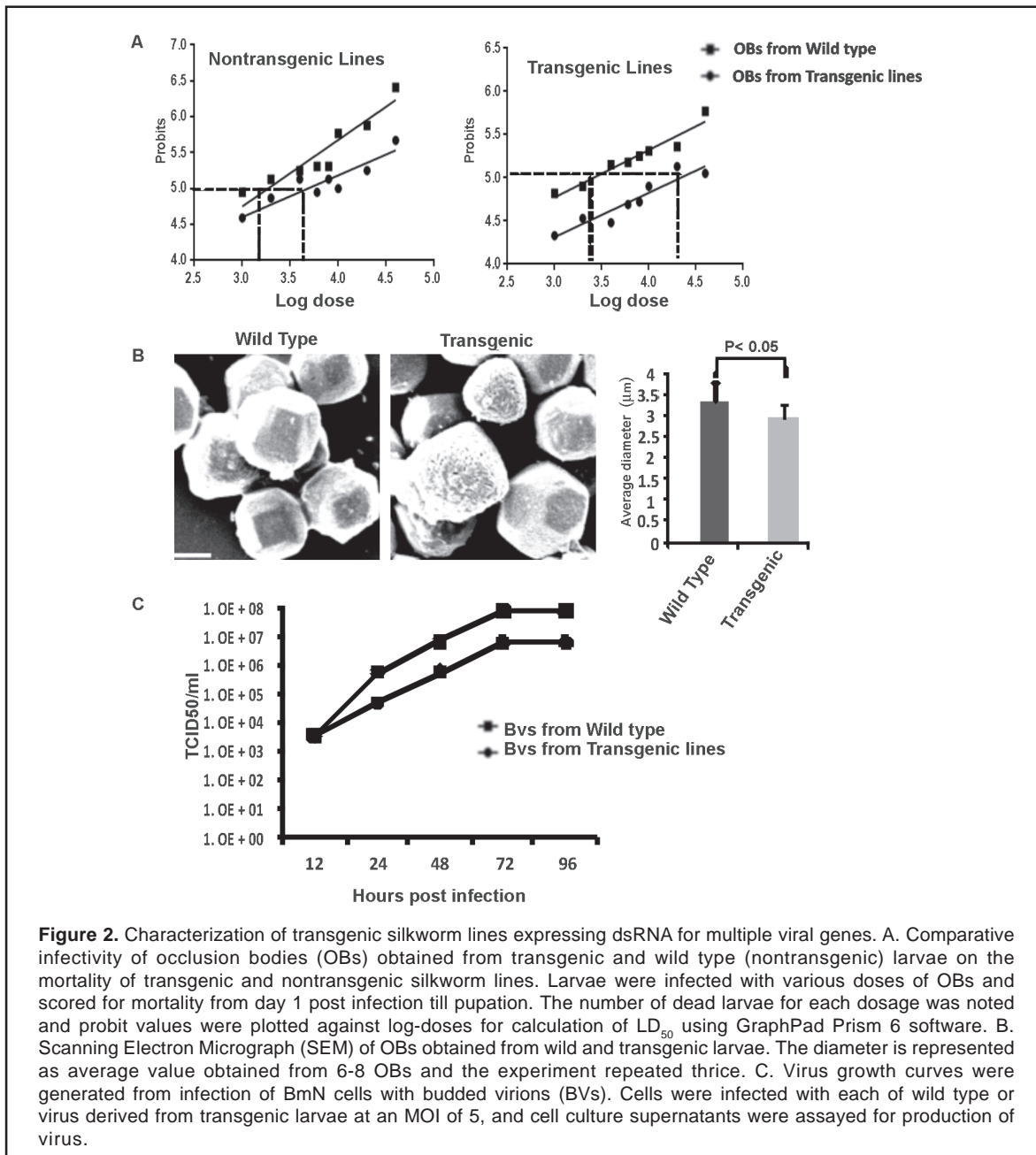


Figure 2. Characterization of transgenic silkworm lines expressing dsRNA for multiple viral genes. A. Comparative infectivity of occlusion bodies (OBs) obtained from transgenic and wild type (nontransgenic) larvae on the mortality of transgenic and nontransgenic silkworm lines. Larvae were infected with various doses of OBs and scored for mortality from day 1 post infection till pupation. The number of dead larvae for each dosage was noted and probit values were plotted against log-doses for calculation of LD₅₀ using GraphPad Prism 6 software. B. Scanning Electron Micrograph (SEM) of OBs obtained from wild and transgenic larvae. The diameter is represented as average value obtained from 6-8 OBs and the experiment repeated thrice. C. Virus growth curves were generated from infection of BmN cells with budded virions (BVs). Cells were infected with each of wild type or virus derived from transgenic larvae at an MOI of 5, and cell culture supernatants were assayed for production of virus.

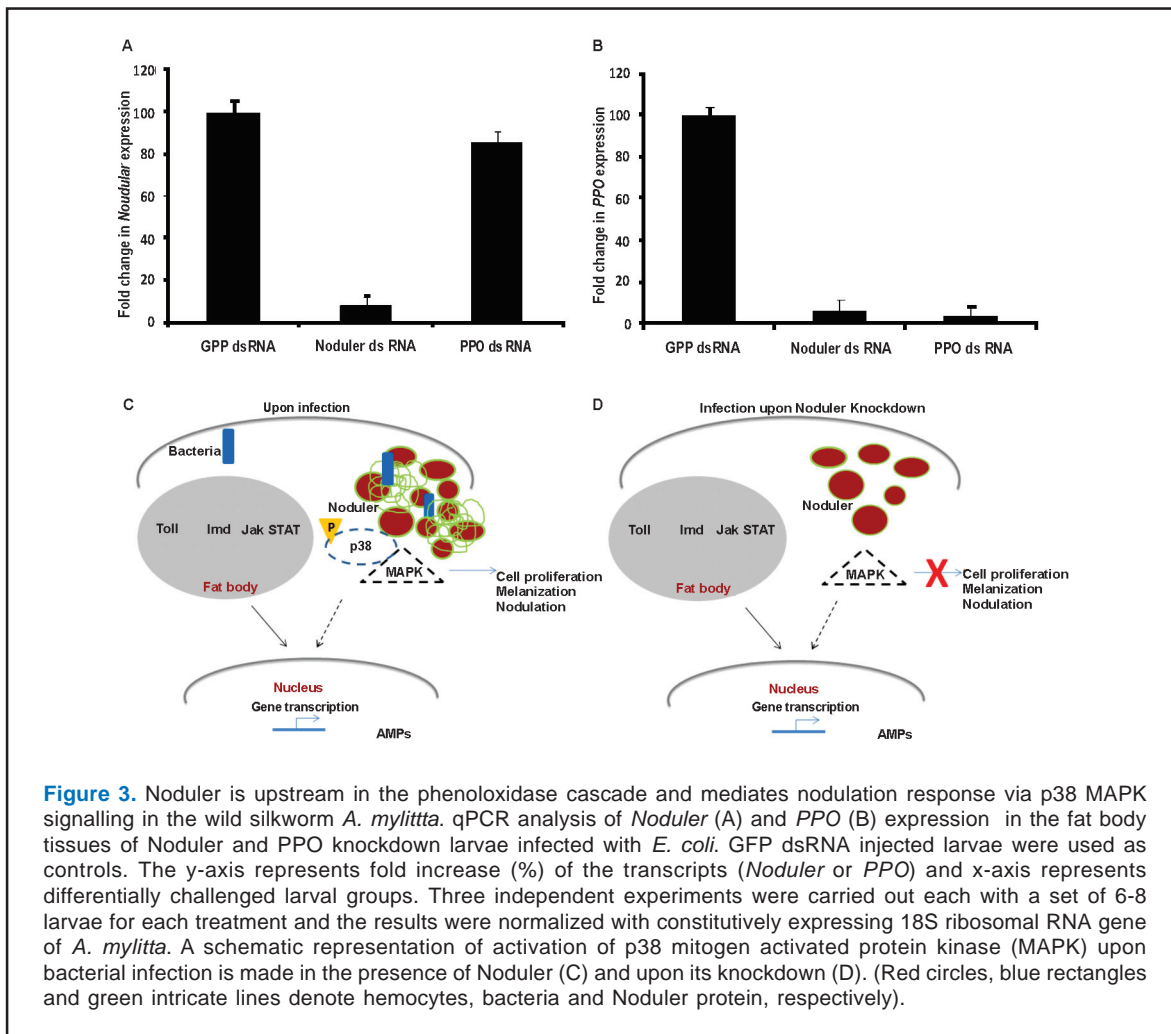
Objective 3: Identification and functional characterization of novel genes involved in immune response pathways of silkmths.

In insects upon infection, the humoral and cellular arms of innate immune system orchestrate

recognition of pathogens facilitating effector responses like production of antimicrobial peptides, phagocytosis, encapsulation or nodulation through various signalling pathways. Despite progress made in understanding the complexity of cellular

immune responses like phagocytosis, our knowledge on nodulation response still remains incomplete. In a previous study, we identified a novel immune protein Noduler which binds specific bacterial components and hemocytes leading to nodulation response in the wild silkworm, *Antheraea mylitta*. However, no molecular mechanism underlying nodulation is elucidated in Lepidopterans. During the period under report, we investigated functional connection between Noduler with various signalling pathways. Our experiments involving RNAi and qPCR analyses inferred that Noduler is upstream in the phenoloxidase cascade

and it augments cell proliferation through activation of p38 mitogen activated protein kinase (MAPK) (Figs. 3A-D). We consolidated information on the nodulation response in insects and made an analogy with that of vertebrate system (Fig. 4). The present study offers cue towards understanding nodulation response across diverse species. Future work includes functional analysis of Noduler homologues in *Drosophila* and mammalian systems, elucidation of the structural basis for preferential recognition of microorganisms by Noduler, and identification of the molecular components of the signalling pathway.



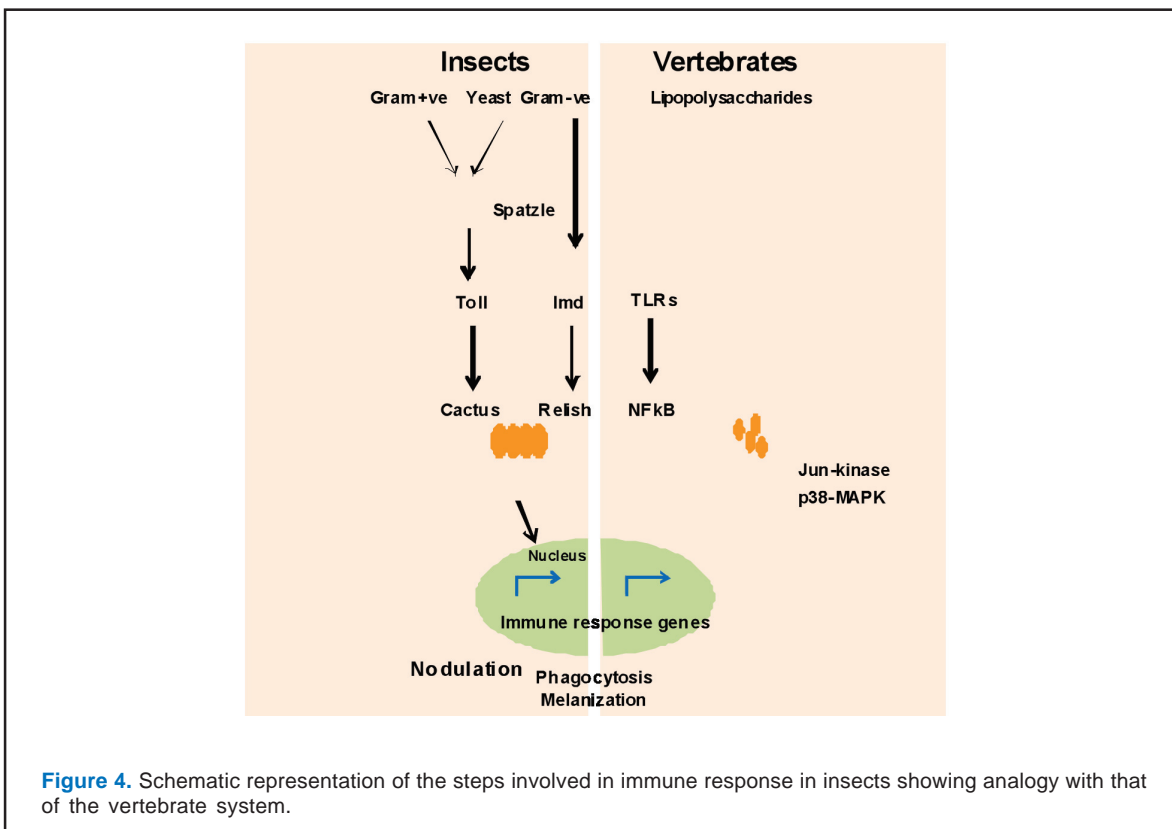
Objective 4: Development of baculovirus resistant silkworm strains using marker assisted selection.

As the molecular events associated with BmNPV infection are well understood, it necessitates utilization of this knowledge in development of

resistant strains of *B. mori*. We have screened multivoltine and bivoltine silkworm strains that are relatively tolerant and susceptible to BmNPV using microsatellite markers. Several markers polymorphic between these two strains were identified. Apart from it, we have performed a

second generation Illumina sequencing to generate 8 pair-end libraries for the midgut and fat body tissues of baculovirus infected and control larvae of both the above mentioned strains. We generated over 200 million paired end reads. Based on bioinformatic pipeline, the transcript abundance was scored in the NPV infected versus control samples and the genes up/down regulated were identified. A number of genes were found to be differentially expressed between the resistant and susceptible silkworm strains. Among them, nine potential candidates having a role in viral entry, namely amino acid transporter, Profilin, Aminopeptidase N, Zn transporter, Cu transporter,

Sugar transporter, DnaJ (Hsp40), VLDL R and Tetraspanin were found to express highly in the susceptible strain. Similarly, we identified serine proteases, serpins, cecropins and noncoding RNAs (lncRNAs) differentially up/down regulated in the resistant and susceptible strains. RNAi-mediated knockdown of Serpins and Tolls in the midgut tissues indicated Serpin 2, lncRNA 4, Toll 2 and Toll 4 to have a significant role in antiviral immunity. Future work includes validation of identified genes by qPCR, their functional analysis by RNAi, and transfer of candidate genes to high yielding susceptible strain through marker assisted recurrent backcross strategy.



Publications

1. Malathi S, Lakshminarayan RV, Hameedunnisa B, Purushotham Reddy B, Neetasri C, Nagaraju J, Anwar SY and Siddiq EA (2013). Population structure and genetic analysis of different utility types of mango (*Mangifera indica* L.) germplasm of Andhra Pradesh state of India using microsatellite markers. **Plant Systematics and Evolution** 299: 1215-1229.
2. Subbaiah EV, Royer C, Kanginakudru S, Satyavathi VV, Babu AS, Sivaprasad V, Chavancy G, Darocha M, Jalabert A, Mauchamp B, Basha I, Couple P and Nagaraju J (2013). Engineering silkworms for resistance to baculovirus through multigene RNA interference. **Genetics** 193: 63-75.
3. Suetsugu Y, Futahashi R, Kanamori H, Kadono-Okuda K, Sasanuma S, Narukawa J, Ajimura M, Jouraku A, Namiki N, Shimomura M,

- Sezutsu H, Osanai-Futahashi M, Suzuki MG, Daimon T, Shinoda T, Taniai K, Asaoka K, Niwa R, Kawaoka S, Katsuma S, Tamura T, Noda H, Kasahara M, Sugano S, Suzuki Y, Fujiwara H, Kataoka H, Arunkumar KP, Tomar A, Nagaraju J, Goldsmith MR, Feng Q, Xia Q, Yamamoto K, Shimada T and Mita K (2013). Large scale full-length cDNA sequencing reveals a unique genomic landscape in a lepidopteran model insect, *Bombyx mori*. **G3: Genes, Genomes, Genetics** 3: 1481-1492.
4. Archak S and Nagaraju J (2014). Computational analyses of protein coded by rice (*Oryza sativa japonica*) cDNA (GI: 32984786) indicate lectin like Ca(2+) binding properties for Eicosapenta Peptide Repeats (EPRs). **Bioinformatics** 10: 63-67.
 5. Nagaraju J, Gopinath G, Sharma V and Shukla JN (2014). Lepidopteran sex determination: a cascade of surprises. **Sexual Development** 8: 104-112.
 6. Saranathan R, Tomar A, Sudhakar P, Arunkumar KP and Prashanth K (2014). Draft genome sequence of a MDR *Acinetobacter baumannii* PKAB 07 clinical strain belonging to ST 195 from India. **Genome Announcements** 2: e00184.
 7. Satyavathi VV, Asha Minz and Nagaraju J. Nodulation: An unexplored cellular defense mechanism in insects. **Cellular Signalling** (In press).
 8. Singh CP, Singh J and Nagaraju J. Regulation of viral late genes by a baculovirus-encoded miRNA. **Insect Biochemistry and Molecular Biology** (In press).
 9. Singh CP, Vaishna RL, Kakkar A, Arunkumar KP and Nagaraju J. Characterization of antiviral and antibacterial activity of *Bombyx mori* seroin proteins. **Cellular Microbiology** (In press).

LABORATORY OF MOLECULAR ONCOLOGY

Genomics and molecular genetics of cancer and genetic disorders

Faculty	Murali D Bashyam	Staff Scientist
PhD Students	P Ramaswamy	Senior Research Fellow
	A Sita Rama Raju	Senior Research Fellow
	Raju Kumar	Junior Research Fellow
	A Srinivas	Junior Research Fellow
	Pratyusha Bala	Junior Research Fellow (Since Jul. 2013)
Other Members	Vasantha K Bhaskara	CSIR Senior Research Associate
	Ratheesh Raman	Project Associate (Till Dec. 2013)
	Mithu Raychaudhuri	Project Associate (Since Jan. 2014)
	Md Khursheed	Project Senior Research Fellow (Till Nov. 2013)
	Ajay Kumar Chaudhary	Technical Assistant
	K Viswakalyan	Research Assistant
	Sandeep N Madana	Research Assistant (Till Dec. 2013)
	Kamtam Ramesh	Project Junior Research Fellow (Till Oct. 2013)
	Rajender K	Project Assistant
	Neha Anilkumar Gupta	Project Junior Research Fellow (Till Jul. 2013)
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	A Dalal	CDFD, Hyderabad
	G Swarnalatha	Apollo Hospitals, Hyderabad
	N Bherappa	NIMS, Hyderabad
	SG Uppin	NIMS, Hyderabad
	M Srinivasulu	MNJ Hospital, Hyderabad
	S Rao	IACHRC, Hyderabad
	KVVN Raju	IACHRC, Hyderabad
	SC Patnaik	IACHRC, Hyderabad
	S Dhagam	Omega Hospitals, Hyderabad
	M Vamsy	Omega Hospitals, Hyderabad
	D Desai	Hinduja Hospital, Mumbai
	AR Ramadevi	Sandor Proteomics, Hyderabad
	SR Phadke	SGPGIMS, Lucknow
	N Gupta	AIIMS, New Delhi
	M Kabra	AIIMS, New Delhi
	D Suri	PGIMER, Chandigarh
	A Gupta	PGIMER, Chandigarh
	RD Puri	Sir Ganga Ram Hospital, New Delhi
	S Kapoor	Lok Nayak Hospital, New Delhi
	S Danda	CMC, Vellore
Objectives		Summary of work done until the beginning of this reporting year (upto March 31, 2013) Lynch Syndrome (LS):
1.	Identification and characterization of important deregulated genes/pathways in cancers prevalent in India; and	LS is an autosomal dominant familial syndrome causing early-onset colorectal cancer (CRC) that results from failure of DNA mismatch repair (MMR)
2.	Identification and characterization of disease causing mutations in genetic disorders.	

system due to germline mutational inactivation of either of four main MMR genes namely *MLH1*, *MSH2*, *MSH6* and *PMS2* leading to loss of corresponding protein expression (MMR-). LS-associated colorectal tumors also exhibit microsatellite instability (MSI). A preliminary screen of suspected LS samples revealed high frequency of MSI but low frequency of loss of MMR expression. Novel *MLH1/MSH2* mutations were detected in samples exhibiting loss of expression of the corresponding protein.

Details of progress made in the current reporting year (April 1, 2013 - March 31, 2014)

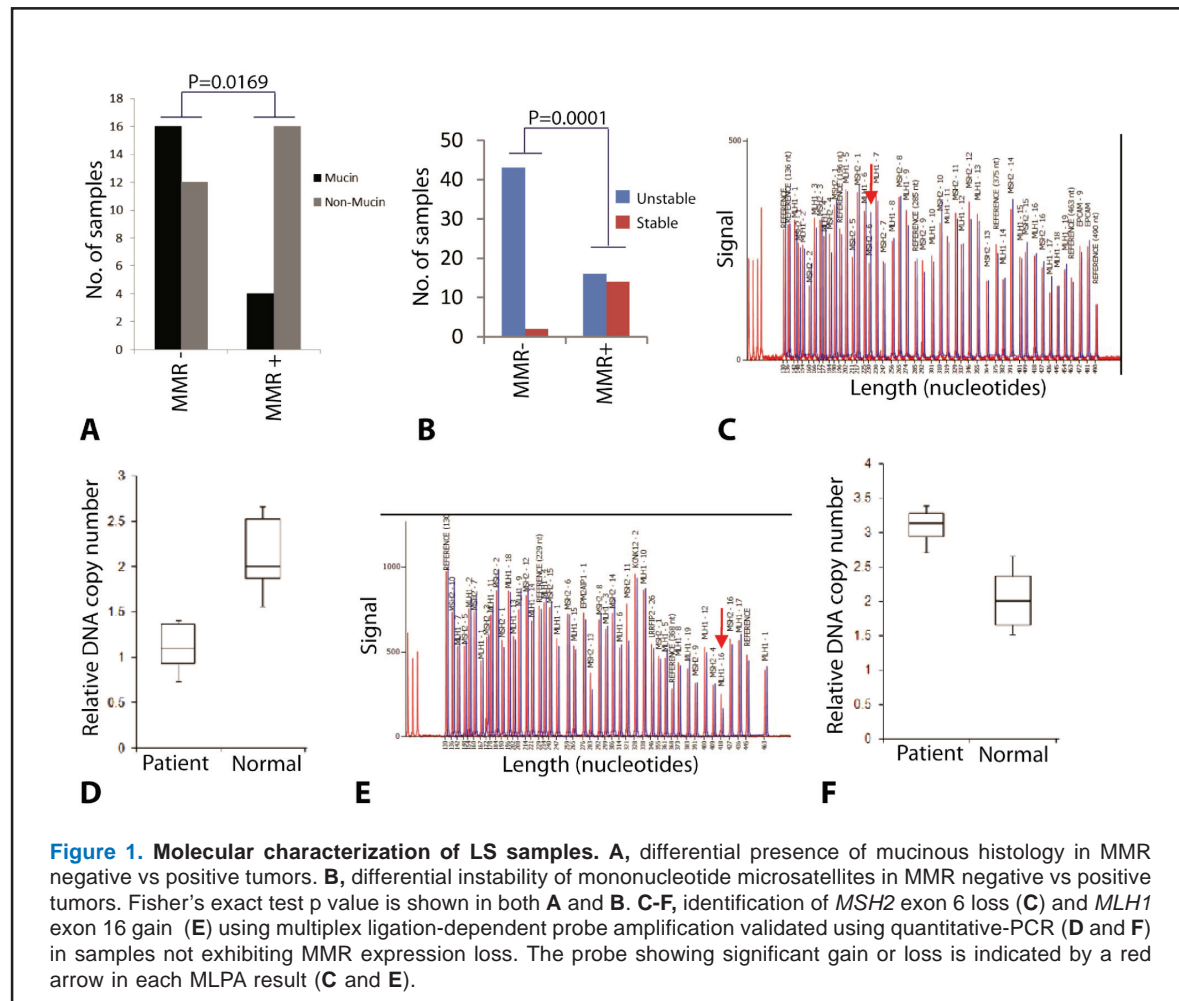
Project 1: Lynch Syndrome (LS).

Of 48 suspected LS cases, 41 exhibited MSI thus confirming association with LS. However, only 23 exhibited loss of *MLH1* or *MSH2* expression, a frequency significantly lower than reported for other countries. We identified several novel disease causing MMR gene point/in-del mutations as well as exonic copy number aberrations in MMR- and

MMR+ samples. Mucin content (Fig. 1A), instability frequency in mononucleotide microsatellites (Fig. 1B) and presence of MMR gene exonic aberrations (Fig. 1C-F) were significantly different between MMR- and MMR+ samples. The work has therefore identified for the first time MMR+ LS-associated CRC despite the presence of MMR gene lesions. Therefore, MMR gene aberrations causing LS do not necessarily cause loss of corresponding protein expression as reported earlier.

Project 2: Clinico-pathological and molecular analysis of squamous cell carcinoma of the oral tongue (SCCOT).

This is a new activity. Unlike other forms of Head and Neck cancer, SCCOT is not significantly associated with elder age and tobacco use. We performed comprehensive clinico-pathological and molecular characterization of more than 120 SCCOT samples. Elevated *EGFR* expression was a frequent occurrence though MSI and HPV



infection were rare. FHIT loss was significantly associated with p53 inactivation especially in patients with no history of tobacco use. Interestingly, p53 inactivation was the only significant prognosticator of SCCOT survival in multivariate analysis.

Project 3: Characterization of TP53 codon 72 Arg/Pro polymorphism in tongue and esophageal cancer.

This is a new activity. The TP53 codon 72 Arg/Pro polymorphism has been extensively studied with respect to many cancers for its possible modulation of tumorigenesis. We assessed its

was detected in ESCC (Table 1). Surprisingly, Pro/Pro genotype was significantly associated with young age in SCCOT (p=0.0314). Interestingly, TP53 DNA binding domain mutation was significantly associated with Pro allele in ESCC (p=0.0015) but not in SCCOT (Table 1).

Project 4: Molecular characterization of Farber lipogranulomatosis (FL).

This is a new activity. FL is a rare autosomal recessive lysosomal storage disorder caused by mutations in the *ASAH1* gene that result in reduced or absent acid ceramidase activity. In the largest ever study, a total of thirteen different mutations

Codon 72 genotype	SCCOT samples (94)		ESCC samples (75)		Normal samples (96)
Pro/Pro	37 (39%)		17 (23%)		22 (23%)
Pro/Arg	39 (42%)		44 (58%)		47 (49%)
Arg/Arg	18 (19%)		14 (19%)		27 (28%)
HWE deviation (p)	0.859		0.752		0.400
Allele frequency					
Proline	0.66		0.48		0.47
Arginine	0.34		0.52		0.53
	**p=0.021				
Codon 72 genotype	p53 mutation				
	Present	Absent	Present	Absent	
Pro/Pro	07	15	11	06	
Pro/Arg	08	19	14	29	
Arg/Arg	03	09	04	10	
			p*=0.057		
Allele					
Proline	NA		23	06	
Arginine	NA		54	65	
			p*=0.0015		

* Fisher's exact test; ** Chi square test; NA, not available (sample no. too small)

Table 1. Comparative analysis of p53 codon 72 polymorphism status in SCCOT, ESCC and normal healthy control samples.

frequency in SCCOT and squamous cell carcinoma of the esophagus (ESCC) with respect to the healthy/normal population and compared individual genotype frequencies with several clinicopathological and molecular parameters. Pro allele was significantly associated with SCCOT compared to healthy controls (p=0.021); no such association

were identified including eleven novel mutations. The IVS6+4A>G splice mutation and the IVS5-16delTTTTTC polypyrimidine tract deletion mutation resulted in skipping of exon 6 (the most common molecular cause for FL) precluding thereby the region responsible for cleavage of enzyme precursor. Using splicing assays based on two independent minigene constructs (pCAS2 and

pcDNA-DUP), a missense mutation (p.V198A) was shown to result in skipping of exon 8 due to inactivation of an exonic splicing enhancer (ESE)

element (Fig. 2). This is the first report of an ESE-inactivating missense mutation in the *ASAH1* gene resulting in FL.

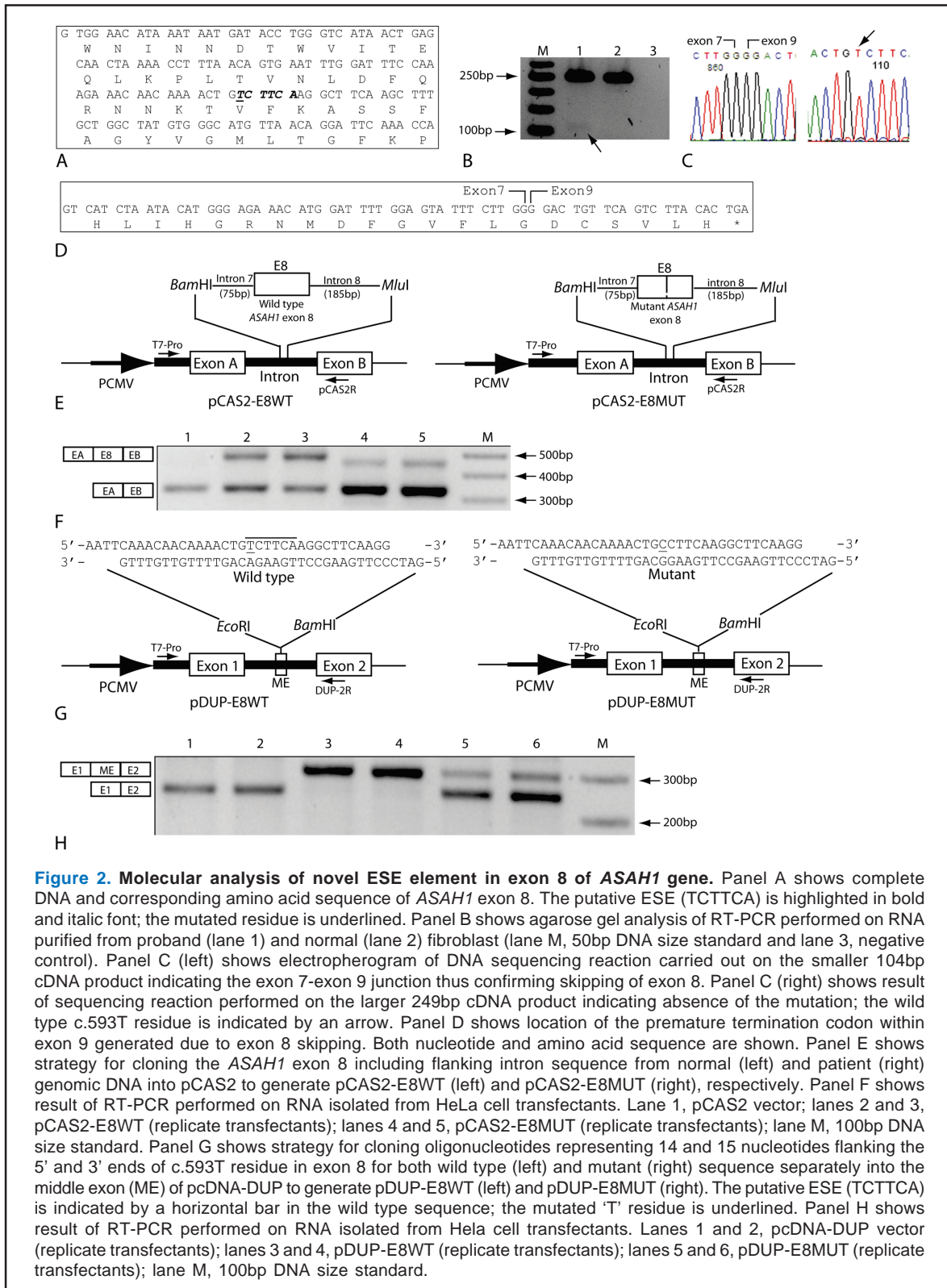


Figure 2. Molecular analysis of novel ESE element in exon 8 of *ASAH1* gene. Panel A shows complete DNA and corresponding amino acid sequence of *ASAH1* exon 8. The putative ESE (TCTTCA) is highlighted in bold and italic font; the mutated residue is underlined. Panel B shows agarose gel analysis of RT-PCR performed on RNA purified from proband (lane 1) and normal (lane 2) fibroblast (lane M, 50bp DNA size standard and lane 3, negative control). Panel C (left) shows electropherogram of DNA sequencing reaction carried out on the smaller 104bp cDNA product indicating the exon 7-exon 9 junction thus confirming skipping of exon 8. Panel C (right) shows result of sequencing reaction performed on the larger 249bp cDNA product indicating absence of the mutation; the wild type c.593T residue is indicated by an arrow. Panel D shows location of the premature termination codon within exon 9 generated due to exon 8 skipping. Both nucleotide and amino acid sequence are shown. Panel E shows strategy for cloning the *ASAH1* exon 8 including flanking intron sequence from normal (left) and patient (right) genomic DNA into pCAS2 to generate pCAS2-E8WT (left) and pCAS2-E8MUT (right), respectively. Panel F shows result of RT-PCR performed on RNA isolated from HeLa cell transfectants. Lane 1, pCAS2 vector; lanes 2 and 3, pCAS2-E8WT (replicate transfectants); lanes 4 and 5, pCAS2-E8MUT (replicate transfectants); lane M, 100bp DNA size standard. Panel G shows strategy for cloning oligonucleotides representing 14 and 15 nucleotides flanking the 5' and 3' ends of c.593T residue in exon 8 for both wild type (left) and mutant (right) sequence separately into the middle exon (ME) of pcDNA-DUP to generate pDUP-E8WT (left) and pDUP-E8MUT (right). The putative ESE (TCTTCA) is indicated by a horizontal bar in the wild type sequence; the mutated 'T' residue is underlined. Panel H shows result of RT-PCR performed on RNA isolated from HeLa cell transfectants. Lanes 1 and 2, pcDNA-DUP vector (replicate transfectants); lanes 3 and 4, pDUP-E8WT (replicate transfectants); lanes 5 and 6, pDUP-E8MUT (replicate transfectants); lane M, 100bp DNA size standard.

Future plans/directions

1. Characterization of MSI+ colorectal cancer in the Indian population.
2. Characterization of tumorigenesis pathways in SCCOT in the absence of p53 inactivation.
3. Characterization of selected mutations affecting transcript stability/processing identified through screening of various genetic disorders.

Publications

1. Bashyam MD, Chaudhary AK, Kiran M, Reddy V, Nagarajaram HA, Dalal A, Bashyam L, Suri D, Gupta A, Gupta N, Kabra M, Puri RD, RamaDevi R, Kapoor S and Danda S (2013). Molecular analyses of novel *ASAH1* mutations causing Farber lipogranulomatosis: analyses of exonic splicing enhancer inactivating mutation. *Clinical Genetics* Nov.8. doi.10.1111/cge.12316.
2. Bashyam MD and Raman R (2013). Molecular origins of colon and rectal cancer: Not a Wnt-Wnt situation. *Current Colorectal Cancer Reports* 9: 365-371.
3. Kavela S, Shinde SR, Ratheesh R, Kotapalli V, Bashyam MD, Gowrishankar S, Vamsy M, Pattnaik S, Rao S, Sastry RA, Srinivasulu M, Chen J and Reddy MS (2013). PNUTS functions as a proto-oncogene by sequestering PTEN. *Cancer Research* 73: 205-214.
4. Khursheed M, Kolla JN, Kotapalli V, Gupta N, Gowrishankar S, Uppin SG, Sastry RA, Koganti S, Sundaram C, Pollack JR and Bashyam MD (2013). ARID1B, a member of the human SWI/SNF chromatin remodeling complex, exhibits tumour suppressor activities

in pancreatic cancer cell lines. *British Journal of Cancer* 108: 2056-2062.

5. Pandilla R, Kotapalli V, Gowrishankar S, Chigurupati M, Patnaik S, Uppin S, Rao S, Kalidindi NR, Sastry RA, Sundaram C, Srinivasulu M, Vasala A, and Bashyam MD (2013). Distinct genetic aberrations in oesophageal adeno and squamous carcinoma. *European Journal of Clinical Investigation* 43: 1233-1239.
6. Bashyam MD, Chaudhary AK, Kiran M, Nagarajaram HA, Devi RR, Ranganath P, Dalal A, Bashyam L, Gupta N, Kabra M, Muranjan M, Puri RD, Verma IC, Nampoothiri S and Kadandale JS (2014). Splice, insertion-deletion and nonsense mutations that perturb the phenylalanine hydroxylase transcript cause phenylketonuria in India. *Journal of Cellular Biochemistry* 115: 566-574.
7. Khursheed M and Bashyam MD (2014). Apico-basal polarity complex and cancer. *Journal of Biosciences* 39: 145-155.
8. Ratheesh R, Kotapalli V, Adduri R, Gowrishankar S, Bashyam L, Chaudhary AK, Chigurupati M, Patnaik S, Srinivasulu M, Sastry RA, Rao S, Vasala A, Kalidindi NR, Pollack JR, Murthy S and Bashyam MD (2014). Evidence for possible non-canonical pathway(s) driven early-onset colorectal cancer in India. *Molecular Carcinogenesis* 53: E181-E186.
9. Ratheesh R, Kongara R, Kotapalli V, Gowrishankar S, Sastry RA, Nagari B, Bashyam MD. Pathological stage significantly predicts survival in colorectal cancer patients: a study from two tertiary care centres in India. *Colorectal Cancer* (In press).

LABORATORY OF NEUROSPORA GENETICS

Does sequence heterozygosity in a cross make wild-isolated *Neurospora* strains behave like suppressors of meiotic silencing? And, what do we hope to find by introgressing translocations from *N. crassa* into *N. tetrasperma*?

Faculty	DP Kasbekar	Haldane Chair
PhD student	Dev Ashish Giri	Junior Research Fellow (Since Jul. 2013)
Other Members	A Sheeba	Technical Officer
	K Sreethi Reddy	Technical Assistant
	S Rekha	Technical Assistant (Since Dec. 2013)

Objectives

Project 1: A majority of wild-isolated *Neurospora crassa* strains, when crossed with tester strains from the standard laboratory Oak Ridge (OR) background, appear to suppress meiotic silencing by unpaired DNA (MSUD). We hypothesized that sequence heterozygosity between the wild and OR genomes might cause one or more MSUD gene to become unpaired and silence itself, thus switching off the MSUD machinery. To test this idea we have now constructed isogenic *mat a* and *mat A* strains from the wild-isolated Bichpuri-1 a (B) and Spurger A (S) suppressor backgrounds (see Annual Report for 2012-13). We are now making new testers to ask whether MSUD occurs in a tester-heterozygous but otherwise isogenic cross in the new B/S hybrid background.

Project 2: By introgressing translocations (*T*) from *N. crassa* into *N. tetrasperma* we hope to create [(*T*) + (*M*)] heterokaryon strains, whose self-crosses should generate [(*T*) + (*M*)] and [(*Dp*) + (*Df*)] progeny (for explanation of terms used see below). Ordinarily, we expect the *Df* nuclei to be rescued by the *Dp* nuclei, therefore both [*T* + *M*] and [*Dp* + *Df*] heterokaryons should be self-fertile, and their self-crosses should again produce [*T* + *M*] and [*Dp* + *Df*] progeny types. But what if a *Df* is not rescued by the corresponding *Dp* nucleus? Will the [*Dp* + *Df*] type effectively regress into a self-sterile [*Dp*] type? Although genes whose null allele (Δ) is not complemented by the wild-type (*WT*) in a [(*WT*) + (Δ)] heterokaryon have not yet been reported, their existence cannot be ruled out, especially in light of the putative “nucleus-limited” behavior found by others for the *N. crassa* *scor*^c mutant (J. Bacteriol. 1972), and for the MatIS process in *Aspergillus nidulans* (Genetics 2013). Any phenotype difference we find between the [*T* + *M*] and [*Dp* + *Df*] heterokaryons would signal the presence of one or more genes with putative nucleus-limited effects.

Summary of work done until the beginning of this reporting year (upto March 31, 2013)

(1) MSUD is an RNAi-mediated process that eliminates the transcripts of any gene that is not properly paired during meiosis with a homologous sequence at an allelic position. The *::Bml*^r and *::mei-3* tester strains contain a copy of the *Bml* (β -tubulin) or *mei-3* gene inserted ectopically in the *his-3* locus on chromosome 1. In the cross of a tester with an OR strain of opposite mating type, the ectopic copy is unpaired in meiosis and induces the synthesis of small interfering RNA which silences it as well as its paired native homologs. Since β -tubulin and MEI-3 protein are essential for ascus development, the silencing results in ascus development abnormalities. Homozygous *tester A* x *tester a* crosses do not show MSUD, nor do crosses of the testers with the semi-dominant *Sad* and *Sms* suppressors of meiotic silencing, and the asci develop normally. The suppressor alleles are presumed to prevent the proper pairing of their wild-type homologues and induce them to autogenously silence themselves. Only eight of 80 wild-isolated strains examined silenced both *bml* and *mei-3* in crosses with the testers and they were designated as “OR” type, four failed to silence both genes and were designated the “Sad” type, and the remaining 68 silenced *bml* but not *mei-3*^r and were designated the “Esm” type. Additional results suggested that MSUD persists throughout the duration of the cross with the OR type, is very fleeting in the cross with the Sad type, and lasts for an intermediate duration in crosses with the Esm type strains. We hypothesized that sequence polymorphism between the tester and wild genomes might cause one or more gene essential for MSUD to become unpaired, silence itself, and thus shorten MSUD duration. To test this idea we are constructing new testers in isogenic *mat a* and *mat A* strains that we derived from the Sad type wild-isolates Bichpuri-1 a (B) and Spurger A (S). Our hypothesis predicts

that MSUD will be seen in a tester-heterozygous cross that is otherwise isogenic for the B/S background.

(2) *N. tetrasperma* is closely related to *N. crassa*, but their life cycles are significantly different. In a *N. crassa* sexual cross, the eight haploid nuclei produced following meiosis and the post-meiotic mitosis become partitioned into the eight ascospores that form within each ascus. Upon ascospore germination the resulting mycelium is homokaryotic and all nuclei have the same mating type. Since mycelia from two ascospores (1 *mat A* + 1 *mat a*) are needed to complete the sexual cycle, *N. crassa* is said to be heterothallic. In contrast, in *N. tetrasperma* the eight haploid nuclei produced following meiosis and post-meiotic mitosis are packaged as four non-sister pairs (1 *mat A* + 1 *mat a*) into the four ascospores that form per ascus. Since the mycelium from a single ascospore contains nuclei of both mating types it is competent to complete the sexual cycle, therefore *N. tetrasperma* is pseudohomothallic. *N. tetrasperma* mycelia can by chance produce some homokaryotic conidia that generate self-sterile single-mating-type derivative strains which can cross with like strains of the opposite mating type. Also, *N. tetrasperma* asci occasionally produce five or more (upto eight) ascospores instead of the normal four, where a pair of smaller homokaryotic ascospores replaces a dikaryotic ascospore. The dominant *Eight-spore* (*E*) mutation substantially increases the frequency of such replacement. The mycelium produced from a small ascospore is homokaryotic and self-sterile and it can cross with a like strain of the opposite mating type. Therefore *N. tetrasperma* is a facultatively heterokaryotic species.

An insertional translocation (*IT*) transfers a segment of a donor chromosome to a recipient chromosome without any reciprocal exchange. Three breakpoint junctions define any *IT*, viz, "A", created by the deletion of the translocated segment from the donor chromosome, and "B" and "C" (proximal and distal), created by its insertion into the recipient chromosome. In a *N. crassa* cross between an *IT* and a normal sequence strain (*IT* x *N*), alternate segregation results in formation of 4 *N* + 4 *T* ascospores that are viable and blacken (B). That is, they do not remain immature and unpigmented (white, W). Therefore, the ascus is of type 8B:0W. Adjacent 1 segregation produces four viable (B) ascospores containing a duplication

(*Dp*) of the translocated segment and four inviable (W) ascospores containing the complementary deficiency (*Df*) and the asci are 4B:4W. Since alternate and adjacent 1 segregation are equally likely, obtaining equal numbers of 8B:0W and 4B:4W asci is a hallmark of *IT* x *N* crosses. Isequential crosses (i.e., *N* x *N* or *IT* x *IT*) yield mostly 8B:0W. Breakpoint junctions of several translocations were defined in our laboratory, and PCR with junction-specific primers can unambiguously establish the progeny genotype from *T* x *N*. The *T* progeny contain all three breakpoints (A, B, and C), *N* progeny contain none, and *Dp* progeny contain B (and C), but not A. PCR-based progeny typing has made it possible to introgress these translocations into *N. tetrasperma*.

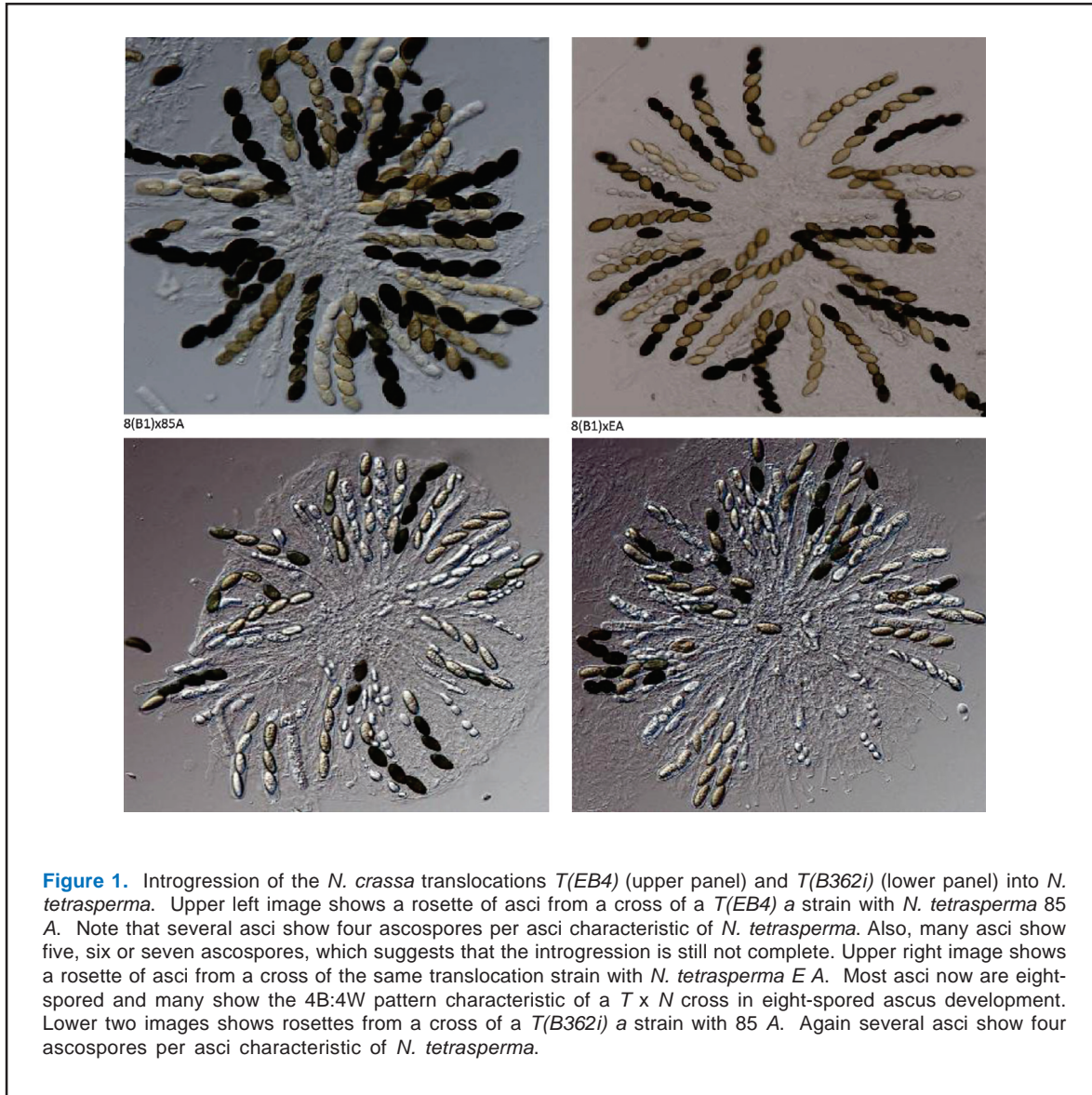
Details of progress made in the current reporting year (April 1, 2013 - March 31, 2014)

(1) We knocked out the *mus-51* gene in the B/S line using the mutational process called RIP. RIP occurs in the premeiotic dikaryon and induces G:C to A:T hypermutation in all copies of DNA sequences that are present in more than one copy in the otherwise haploid genome. The MUS-51 protein is needed for non-homologous end joining (NHEJ), therefore in a *mus-51* mutant strain any transforming DNA can only integrate by homologous recombination, and one can use targeted integration to create well-defined reporter strains.

To make the RIP-induced *mus-51* mutant we transformed the B/S *mat A* strain with a DNA construct containing the *hph* gene for hygromycin-resistance together with a 1683 bp genome fragment stretching from 205 bp upstream of the MUS-51 ORF start codon to 1478 bp down stream. The complete MUS-51 ORF is 2046 bp in length. The majority of hygromycin-resistant transformants were due to ectopic integrations of the transforming DNA, and they were duplicated for ~70% of the *mus-51* ORF (*Dp(mus-51)*). We crossed the *Dp(mus-51)* transgene into B/S *mat a* to set up *Dp(mus-51)*-homozygous crosses. Since RIP frequency increases with the age of the cross, 40 progeny from late harvested ascospores were screened for RIP-induced alterations in restriction site in the endogenous *mus-51* gene. Five progeny that showed evidence of RIP were further examined by sequencing of their *mus-51* gene, and three mutants were found to contain many RIP-induced mutations, including in-frame stop codons. One of

these presumptive *mus-51* null mutant alleles was segregated into both mating types in the B/S background. Next, these strains will be used to generate new testers by targeted integration of transforming DNA.

(2) We made significant progress in introgressing the *N. crassa* translocations *T(IVR > I)B362i* and *T(VR > VII)EB4* (abbreviated henceforth to *T(B362i)* and *T(EB4)*) into *N. tetrasperma* (see Fig. 1).



T(B362i) transfers a 118,782 bp segment of chromosome 4R into a site on chromosome 1L, and *T(EB4)* translocates a 145,282 bp segment of chromosome 5 to a proximal site in chromosome 7L. Since crosses between *N. crassa* and *N. tetrasperma* strains are almost completely sterile, we had to first cross the translocation strains with the bridging strain C4T4 *a*, identify the *T* progeny, then once or twice more repeat the crosses of the *T* progeny with C4T4 *a*, and only then were we able to obtain *T* strains that were capable of

crossing with the opposite mating type derivatives of *N. tetrasperma* strain 85. After two or three more rounds of crosses with 85 A or *a*, we started obtaining progeny containing the translocation breakpoints and that were also of dual mating specificity. That is, these strains could cross with both 85A and *a*, and they could also self-cross. The *T* strains are now being crossed with *E* strains of opposite mating type to increase the fraction of the *N. tetrasperma* genome.

Publications

1. Kasbekar DP (2013). Neurospora duplications and genome defense by RIP and meiotic silencing. In: Neurospora: Genomics and Molecular Biology. Edited by DP Kasbekar and K McCluskey. **Caister Academic Press, Norfolk, UK** 109-127.
2. Nagasowjanya T, Kranthi Raj B, Sreethi Reddy K and Kasbekar DP (2013). An apparent increase in meiotic silencing strength in crosses involving inbred *Neurospora crassa*

strains. **Fungal Genetics and Biology** 56: 158-162.

Other Publications

1. Kasbekar DP (2013). Book review: "March of the microbes: Sighting the unseen". **Current Science** 104: 971.
2. Kasbekar DP (2013). Myth versus mutant: story of *o*. **Journal of Biosciences** 38: 1.
3. Kasbekar DP (2014). Editorial. Lesser models. **Journal of Biosciences** 39: 1.

LABORATORY OF PLANT-MICROBE INTERACTIONS

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

Faculty	Subhadeep Chatterjee	Staff Scientist
PhD Students	Rikky Rai	Senior Research Fellow
	Sheo Shankar Pandey	Senior Research Fellow
	Akanksha Kakkar	Senior Research Fellow
	Raj Kumar Verma	Senior Research Fellow
	Biswajit Samal	Junior Research Fellow
	Prashantee Singh	Junior Research Fellow (Since Feb. 2014)
Other Members	Binod Bihari Pradhan	Technical officer
	Anil Kondreddy	CSIR Research Associate
	Sree Gowrinadh Javvadi	Project-Junior Research Fellow
	L Santhosh Kumar	Project-Junior Research Fellow
	Pradeep Kumar Patnana	Project-Junior Research Fellow (Since Jan. 2014)

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; and
4. Role of PAMP in pathogen recognition and plant defense response

Summary of work done until the beginning of this reporting year (upto March 31, 2013)

We are trying to understand the virulence mechanisms of important *Xanthomonas* pathogens like, *Xanthomonas campestris* pv. *campestris* (Xcc; a pathogen of crucifers), *Xanthomonas oryzae* pv. *oryzae* and *Xanthomonas oryzae* pv. *oryzicola* (Xoo, Xoc; pathogens of rice). In *Xanthomonas*, cell-cell (quorum sensing) is mediated by the production and sensing of fatty acid like signaling molecule known as Diffusible signaling factor. We have shown that DSF in Xoo plays an important role in transition of planktonic to biofilm lifestyle. Our studies have shown that DSF in closely related phytopathogens regulate virulence associated traits in a contrasting fashion. To understand the role of DSF in adaptation to different lifestyle we have characterized the role of DSF in the virulence of Xoo and Xoc. Characterization of DSF deficient $\Delta rpfF$ mutant of Xoc revealed that DSF promotes in planta growth by regulating ferric iron uptake.

We are presently studying the role of DSF in regulating virulence associated function in *Xanthomonas oryzae* pv. *oryzicola* (Xoc) and its contribution to adaptation to host environment. Previously we have isolated a novel adhesin of Xoo, XadM, which is required for virulence. Analysis of XadM adhesins indicated that it is primarily present in xylem vessel colonizing pathogens. In order to gain more insight into its role in xylem colonization, we used Xoc, a rice parenchyma tissue colonization pathogen, as a gain of function approach to study the significance of this adhesin in the biology of vascular vs. non-vascular pathogen.

To understand the dynamics of quorum sensing, we have previously constructed several biosensor strains to study quorum sensing response in individual cells in the population. We have used also an *E. coli* system to reconstitute the AHL mediated QS system to study QS in a heterologous host. Our study has indicated that bacteria exhibit non genetic phenotypic heterogeneity in social behavior and may contribute to bet hedging strategy to changing environmental condition.

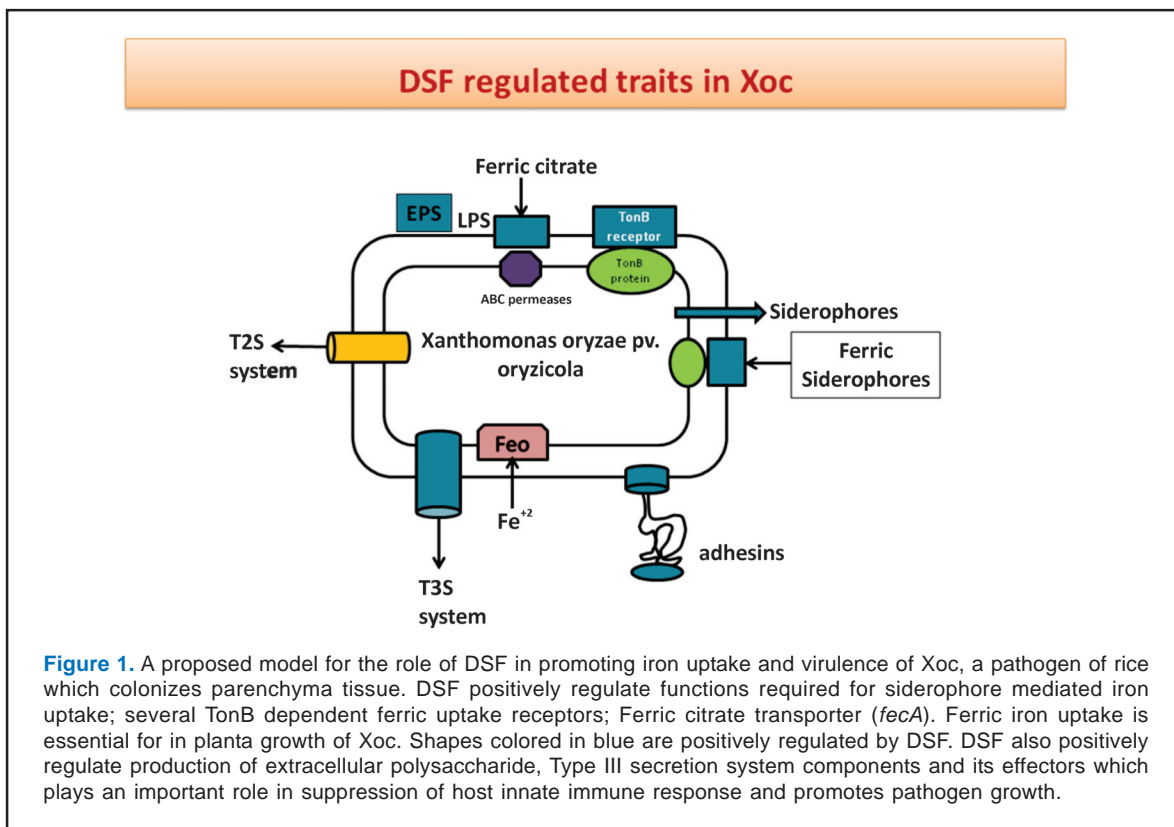
Details of the progress made in the current reporting year (April 1, 2013–March 31, 2014)

Project 1: Role of DSF in virulence of *Xanthomonas oryzae* pv. *oryzicola* (Xoc).

Since DSF exhibits atypical regulation of virulence associated functions in closely related *Xanthomonas*, we wanted to understand the role

of DSF in *Xanthomonas oryzae* pv. *oryzicola* (Xoc). Xocola has an atypical lifestyle as compare to Xoo (a xylem dwelling pathogen), as it infects the rice plant by gaining entry through stomata and grows in the parenchyma tissue. Xoc exhibit streak like symptoms as oppose to long lesions exhibited by Xoo on the mid vein of rice leaves. To understand how DSF promotes in planta growth of Xoc, we have characterized the DSF deficient $\Delta rpfF$ mutant of Xoc. Mutant analysis by expression analysis, radiolabelled iron uptake studies and growth under low iron conditions indicated that DSF positively regulates ferric iron uptake. Further, the DSF deficient mutant of *X. oryzae* pv. *oryzicola* exhibited a reduced capacity to use ferric form of iron for

growth under low-iron conditions. Exogenous iron supplementation in the rice leaves rescued the *in planta* growth deficiency of the DSF deficient mutant of *Xanthomonas oryzae* pv. *oryzicola*. These data suggest that DSF promotes in planta growth of Xoc by positively regulation functions involved in ferric iron uptake which is important for its virulence. 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 *Xanthomonas* group of plant pathogens. We have proposed a model for the role of DSF in regulating iron uptake and metabolism in Xoc (Fig. 1).



Project 2: Dynamics of cell-cell signaling (quorum sensing) in bacteria.

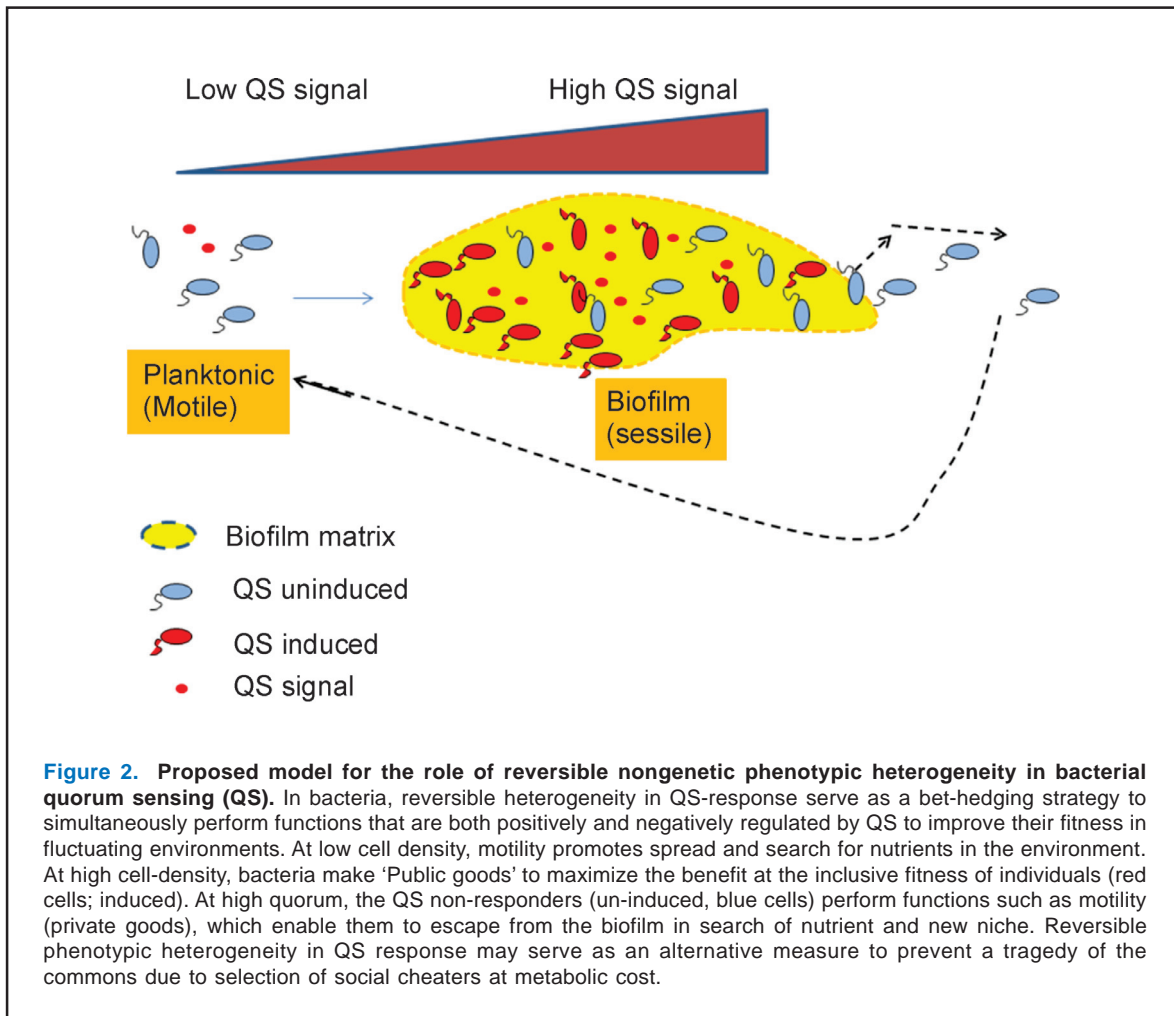
Bacteria coordinate their social behavior in a density dependent manner by production of diffusible signal molecules by a process known as quorum sensing (QS). It is generally assumed that in homogenous environments and at high cell density, QS synchronizes cells in the population to perform collective social tasks in unison which maximize the benefit at the inclusive fitness of individuals. However, evolutionary theory predicts

that maintaining phenotypic heterogeneity in performing social tasks is advantageous as it can serve as a bet-hedging survival strategy. Previous studies to understand the mechanism of quorum sensing population behavior was done primarily on in bulk culture. However, very little information was available on -How single cells behave in a population, particularly in the state of quorum. QS-responses have been characterized primarily in bulk populations, and it is likely that such measurements would mask non-genetic

phenotypic heterogeneity exhibited by individual cells. Therefore, to understand how individuals behave in populations undergoing QS, we addressed this process in *Pseudomonas syringae* pv. *syringae* (Pss) and *Xanthomonas campestris* pv. *campestris* (Xcc) as model organisms that use two diverse classes of QS signals. Using *Pseudomonas syringae* and *Xanthomonas campestris* as model organisms, which use two diverse classes of QS signals, we have shown that two distinct subpopulations of QS-responsive and non-responsive cells exist in the QS-activated population. Addition of excess exogenous QS signal does not significantly alter the distribution of QS-responsive and non-responsive cells in the population. We further show that progeny of cells derived from these subpopulations also exhibited heterogeneous distribution patterns similar to their respective parental strains. We have also demonstrated that heterogeneity in QS-response

is exhibited by bacterial cells in performing social behavior such as swarming motility. Overall, these results support the model that bacteria maintain QS-responsive and non-responsive subpopulations at high cell densities in a bet-hedging strategy to simultaneously perform functions that are both positively and negatively regulated by QS to improve their fitness in fluctuating environments (Fig. 2). 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.

In the future should prove fruitful to study whether the inherent stochastic heterogeneity in QS-responses plays a role in the adaptation of bacteria to fluctuating environmental conditions in their natural habitats. Strategies aimed at altering nongenetic phenotypic heterogeneity in cells undergoing QS-responses may have implications for QS-interference mediated disease control.



Publications

1. Chatterjee S and Pandey A (2013). Signaling in plant-microbe interactions. *Plant Stress* 7: 52-59.
2. Pradhan BB and Chatterjee S. Reversible non-genetic phenotypic heterogeneity in bacterial

quorum sensing. *Molecular Microbiology* (In press).

Other Publications

1. Chatterjee S (2013). Review of: Annual Review of Microbiology, 2011. Susan Gottesman and Caroline S Harwood (eds). *Current Science* 104: 653-654.

LABORATORY OF TRANSCRIPTION

Mechanism of transcription termination and antitermination in Escherichia coli

Faculty	Ranjan Sen	Staff Scientist
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	Rajesh Sashni	Senior Research Fellow (Till Dec. 2013)
	Sourabh Mishra	Senior Research Fellow
	Mohd Zuhaib Qayyum	Senior Research Fellow
	V Vishalini	Senior Research Fellow
	Gairika Ghosh	Junior Research Fellow
	Richa Gupta	Junior Research Fellow (Since Jul. 2013)
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	Amitabh Ranjan	Research Associate (Since Feb. 2014)
	Debashish Dey	DBT-Research Associate
	Rajeshwari Hosammani	DBT-Research Associate (Till Jun. 2013)
	Savita Sharma	Research Associate (Till May 2013)
	Sudha Kalyani	Research Associate
	M Pallavi	Project-Junior Research Fellow
	Ragini Mishra	Project-Junior Research Fellow
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	M Jayavardhan Reddy	Technical Assistant (Since Dec. 2013)
Collaborators	Udayaditya Sen	SINP, Kolkata
	V Nagaraja	IISc., Bangalore
	Akira Ishihama	Hosei University, Japan

Objectives

Fundamental questions in the area of mechanism of transcription termination and antitermination processes is still not very clear and offers an exciting subject for study. In my laboratory, we have undertaken following studies. 1) Mechanism of action of transcription termination factor, Rho. 2) Molecular basis of Rho-NusG interaction. 3) Mechanism of conversion of NusA into an antiterminator by N. 4) Mechanism of action of transcription antitermination of Rho-dependent termination by an anti-rho factor, Psu. 5) In vivo cross-talks between Rho dependent termination and other biological processes. 6) Designing transcription modulators using synthetic biology approaches.

Summary of work done until the beginning of this reporting year (upto March 31, 2013)

- We have established the structural and mechanistic basis of antitermination of Rho

dependent transcription termination by a bacteriophage capsid protein, Psu.

- We have redefined the interaction surface of the bacterial transcription elongation factor, NusA, required for complex formation with the antiterminator, N, during transcription antitermination.

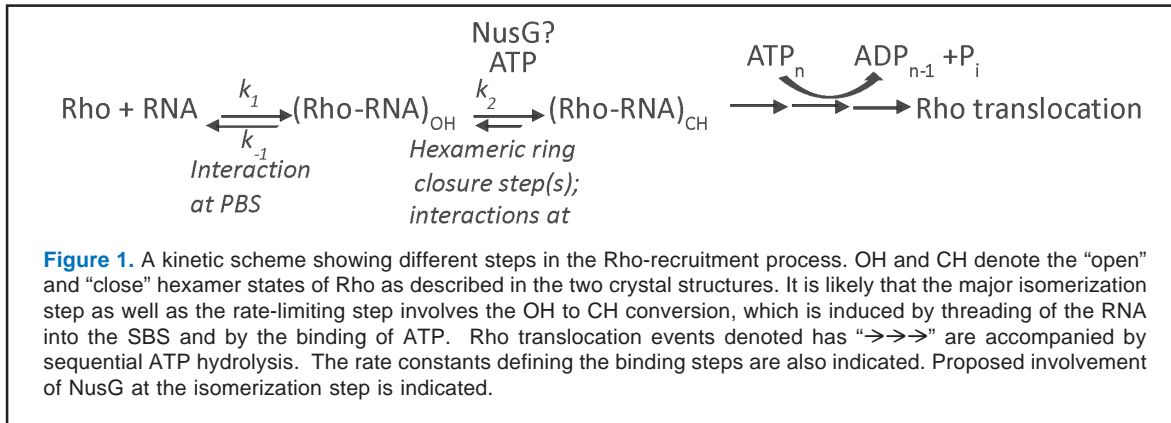
Details of progress made in the current reporting year (April 1, 2013 - March 31, 2014)

1) Redundancy of primary RNA-binding functions of the bacterial transcription terminator, Rho.

The bacterial transcription terminator, Rho, terminates transcription at half of the operons. According to the classical model, derived from *in vitro* assays on a few terminators, Rho is recruited to the transcription elongation complex (EC) by recognizing specific sites (*rut*) on the nascent RNA. Here we explored the mode of *in vivo* recruitment process of Rho. We show that sequence specific

recognition of the *rut* site in majority of the Rho-dependent terminators can be compromised to a great extent without seriously affecting the genome-wide termination function as well as the viability of *E.coli*. These terminators function optimally only through a NusG-dependent assisted recruitment and activation of Rho. Our data also

indicate that at these terminators, Rho-EC-bound NusG interaction facilitates the isomerization of Rho into a translocase-competent form by stabilizing the interactions with mRNA in its secondary RNA binding site, thereby overcoming the defects of the primary RNA binding functions (Fig.1).



2) Transcription elongation factor, NusA, is a general antagonist of Rho-dependent termination.

NusA is an essential, multi-domain and multi-functional protein that binds to RNA polymerase (RNAP) and also binds to nascent RNA. Earlier *in vivo* and *in vitro* observations implicated the involvement of NusA in Rho-dependent termination. Based on the multifunctional nature of NusA, we envisioned the following scenarios during Rho dependent termination. 1) NusA and Rho can both compete for same sites on the nascent RNA, thereby the former can act as an antagonist of the later. 2) Binding of NusA to RNAP induces pausing to the ECs, which in turn can influence the Rho-dependent termination. 3) NusA may modulate Rho-NusG interactions during the termination process.

We screened NusA mutants defective for Rho-dependent termination. We isolated a NusA mutant, G181D, and also tested another reported NusA mutant, R258C. Upon detailed *in vivo* and *in vitro* analyses we made the following conclusions. 1) The NusA mutants inhibit Rho-function in a *nut* site (the only known high affinity NusA-binding site)-dependent manner. 2) This behavior of NusA mutants can be reproduced in the presence of higher concentrations of either WT NusA or its SKK(RNA binding) domain. 3) The enhanced inhibition by NusA mutants arose due to their higher

affinity for nut site. Similarly Rho is inhibited at ribosomal operon by tight-binding of NusA to AT box. 4) Genome wide expression profiles of NusA revealed NusA-induced Rho-inhibition at specific operons having nut-like sequences. We propose that NusA, like Hfq, functions as a general antagonist of Rho. These antagonistic mechanisms have evolved to control the aggressive Rho-function.

3) NusG homologue Rv0639 of Mycobacterium tuberculosis does not interact with Rho but forms stable complex with NusE.

NusG, an elongation factor, binds to the termination factor Rho and facilitates the termination process. It also binds to the ribosomal protein S10 (NusE), which may couple transcription and translation process. In this study, we have attempted to characterize Rv0639, a NusG homologue, to understand transcription processes of *Mycobacterium tuberculosis*.

Using *E.coli* as host, we observed that Rv0639 was unable to complement the functions of *E.coli* NusG in Rho-dependent termination. Even though the homology model of *M.tb* NusG is similar to that of *E.coli*, it did not bind to either *E.coli* or *M. tuberculosis* Rho proteins. It also did not show any *in vitro* function with *E. coli* RNAP as well as Rho. Interestingly, Rv0639 formed a stable complex specifically with *M. tuberculosis* S10. These

results led us to hypothesize that Rv0639 have different conformations, and might have evolved to function differently.

We probed its conformations in detail and made the following observations. 1) Rv0639 has a 50 amino acids extra N-terminal region that folds over its c-terminal domain (CTD), and its deletion reduced the solubility of the protein. 2) In solution, it exists as a monomer, but has more β -sheet content compared to its *E.coli* counterpart, and also migrates anomalously in SDS-PAGE. 3) Its CTD has a distinct CD-spectrum compared to the *E.coli* one, and appeared to be more compact and relatively resistant to trypsin cleavage. These results clearly suggest that the CTD of Rv0639 is structurally different and has evolved only to interact with S10. And hence, *M. tuberculosis* NusG may only have functions in transcription-translation coupling and does not facilitate Rho-dependent termination.

Future plans/directions

The following projects, being pursued in my lab, are in different stages of completion. 1) Mechanism of NusG mediated stimulation of Rho. 2)

Mechanism of conversion of NusA into an antiterminator by N. iii) Mode of *in vivo*-Rho RNA interactions. iv) Cross-talks between Rho-dependent termination and other physiological process.

Publications

1. Mishra S, Mohan S, Godavarthi S and Sen R (2013). The interaction surface of a bacterial transcription elongation factor required for complex formation with an antiterminator during transcription antitermination. ***Journal of Biological Chemistry*** 288: 28089–28103.
2. Ranjan A, Banerjee R, Pani B, Sen U and Sen R (2013). The moonlighting function of bacteriophage P4 capsid protein, Psu, as a transcription antiterminator. ***Bacteriophage*** 3: e25657.
3. Ranjan A, Sharma S, Banerjee R Sen U and Sen R (2013). Structural and mechanistic basis of antitermination of Rho-dependent transcription termination by a bacteriophage P4 capsid protein Psu. ***Nucleic Acids Research*** 41: 6839-6856.

अन्य वैज्ञानिक सेवाएँ / सुविधाएँ
Other Scientific Services / Facilities

LABORATORY ANIMAL FACILITY

Faculty Coordinators	Rashna Bhandari	Staff Scientist & WT-DBT India Alliance Senior Fellow
	Sanjeev Khosla	Staff Scientist
Other Members	Hole Jayant Pundalikrao	Officer In -Charge
	Sridhar Kavela	Technical Officer (Since Dec. 2013)
	Suman Komjeti	Technical Assistant

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. Maintain inbred transgenic strains of mice in a controlled environment, as per CPCSEA guidelines;
3. 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; and
4. Comply with regulatory government body requirements, meet the needs of animals, and promote environment stability to reduce inconsistency in personnel performance and operational costs.

Summary of work done until the beginning of this reporting year (upto March 31, 2013)

The CDFD Laboratory Animal Facility (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 and Forests, Govt. of India, at M/s Vimta Labs Ltd. Until March 2013, the facility housed approximately 900 mice of five different strains, and supplied users with 567 mice for IAEC approved experimentation.

Details of the progress made in the current reporting year (April 1, 2013 - March 31, 2014)

During this reporting year, CDFD LAF has housed five inbred mouse strains, including *Ip6k1*, *Nnat*, C57BL/6, *FoxN1^{nu}* and Balb/c. Mice were bred to expand the colonies and meet users' requirement. Currently this facility has approximately 885 mice housed in 380 IVC cages (Table 1). During the year, 821 mice were supplied to users for IAEC approved experimentation.

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 2013-14 are highlighted below:

Strains	Total (Male + Female)	Under Breeding (Male + Female)	Supplied during 2013-14
<i>Ip6k1</i>	160+156	08+16	128
<i>Nnat</i>	169+136	06+06	106
Balb/c	15+15	06+11	355
C57BL/6	26+30	08+20	100
<i>Foxn1^{nu}</i>	08+09	05+10	10

Table 1. Strain-wise break up of mouse strains housed at LAF as on March 31, 2014, and supplied to users during 2013-14.

- 55 Balb/c mice were injected subcutaneously with protein antigens and polyclonal antibodies were generated successfully.
 - 6 *Ip6k1* heterozygous mice were used to collect 13.5 day embryos to generate mouse embryonic fibroblast (MEFs) for further research.
 - Attempts were made in 24 *Ip6k1* and 2 *Nnat* strains of mice to collect two cell stage embryos by super-ovulation, followed by vitrified cryopreservation for live embryo retrieval and surgical transfer into 6 Balb/c pseudo-pregnant surrogate mothers by using 6 Balb/c vasectomised males. This procedure is still under standardization.
 - 11 *Ip6k1* heterozygous males were injected with retrovirus carrying *Ip6k1* cDNA, and 5 wild type males were injected with retrovirus carrying GFP, into the inter-tubular space of testes surgically, and after 35 days bred with females. Progeny were genotyped for the presence of the transgene. This procedure is still under standardization.
 - 90 C57BL/6 and 116 Balb/c were injected with thioglycolate by intra-peritoneal route of injection for the successful generation of macrophages.
- The IAEC approved projects in progress during this reporting year are mentioned in Table 2.

Sl. No.	Projects in progress
1	Functional analysis of Neuronatin's second intron by knock out strategy
2	Establishment and histopathological characterization of <i>Ip6k1</i> knockout mice - version II
3	Signal transduction pathway in immune cells regulating their innate and effector functions during oxidative stress
4	Protocol for comparative bio-burden study of fifteen strains of <i>Candida glabrata</i> 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	Protocol for the use of nude mice in the study of tumorigenesis
8	Protocol for generation of mouse polyclonal antibodies
9	Isolation of macrophages from Balb/c Mice
10	Cryopreservation of mouse embryo by vitrification
11	Understanding the role of Rab711 in phagosome maturation and immune effector signalling
12	Protocol for establishment and histopathological characterization of <i>Ip6k2</i> knockout mice
13	Protocol for establishment of transgenic mouse model to study the role of <i>Ip6k1</i> in tumorigenesis
14	Studying the immunomodulatory role of some candidate recombinantly purified proteins of mycobacteria
15	Studying the <i>in vivo</i> immunomodulatory role of some candidate PE/PPE proteins of <i>Mycobacterium tuberculosis</i> recombinantly over expressed in the non pathogenic <i>Mycobacterial</i> strains of <i>M. smegmatis</i>

Table 2. IAEC approved projects proposed by various groups at CDFD, in progress during 2013-14.

Future direction

Apart from continuing our current research activities, our goal is to establish a mouse embryo and sperm cryopreservation facility to archive and retrieve mouse strains important for our research.

We are testing recently reported novel methods to develop our own transgenic mouse strains, such as testicular injections of lentivirus expressing transgenes. We are actively involved in the ground work to establish an animal facility in the future CDFD campus.



Figure 1. Blood collection by retro-orbital route for generation of polyclonal antibodies from Balb/c mouse.

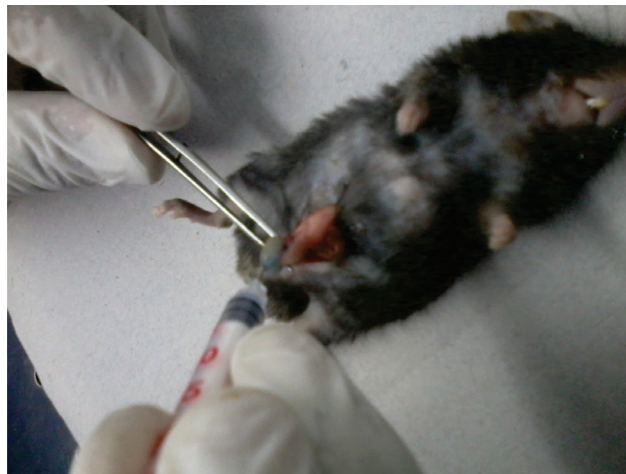


Figure 2. Procedure of intra-testicular injection in mice for transgene expression.

BIOINFORMATICS

Head	HA Nagarajaram	Staff Scientist
Other Members	R Chandra Mohan	Technical Officer
	K Prashanthi	Technical Assistant

Objectives

1. To maintain various servers, workstations, PCs, printers and other peripheral devices;
2. To maintain the 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 the CDFD network from security threats;
5. To integrate Institute's network into National and International grid computing networks; and
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, 2013)

- Activities related to installation, administration and maintenance of servers which provide various services, databases and computational jobs were undertaken.
- Procured two high end servers with 4 processors, 512 GB RAM, 5TB internal storage.
- Internet, web, email-services were provided with enhanced functionalities.
- High-end PCs, workstations, laptops, scanners and printers were procured and installed.
- PC Annual Maintenance Contract was awarded to a new vendor M/s Bharat IT Services.

- Renewed the MoU with CDAC for availing GARUDA-grid facility.
- Upgraded the Firewall, procured additional antivirus licenses.
- Initiated the process of setting up a fail-safe server for the existing email server.

Details of progress made in the current reporting year (April 1, 2013-March 31, 2014)

- Activities related to installation, administration and maintenance of servers which provide various services, databases and computational jobs were undertaken.
- Existing PC Annual Maintenance Contract was renewed.
- Internet, web, email-services have been provided with enhanced functionalities.
- High-end PCs, workstations, laptops, scanners and printers were procured and installed.
- Renewed the MoU with CDAC for availing GARUDA-grid facility.
- Configured Zimbra Email server with a failsafe server.
- Initiated the process of procuring next generation firewall, high end intelligent switches.
- Coordinated the process of procurement and setup of server with workstations and backup facility for CODIS project.

INSTRUMENTATION

Head	Raghavendrachar J	Staff Scientist
Other Members	RN Mishra	Senior Technical Officer
	SD Varalaxmi	Technical Officer
	M Laxman	Technical Officer
	RMK Satyanarayana	Technical Officer
	T Ramakrishna Reddy	Technical 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 (upto March 31, 2013)

During the year 2012-13, we had installed 68 new equipments like Multi mode Reader, Inverted Microscopes, Chemiluminescence Gel Documentation System, PCR Machines, Refrigerated Centrifuges, Shaking waterbaths, Electroporators, -80°C Freezer, -20°C Freezers, Cooled Incubator, Refrigerators etc. and had also completed 491 work orders for repair & maintenance of various laboratory equipments. We had successfully set up the Bio-metric Attendance system at both Tuljaguda and Gruhakalpa complexes registering the accurate attendance of all our staff, Research scholars, Project Staff and administrative staff.

In addition, we were involved in organizing the audio & visual requirements for presentations in various seminars, lectures and workshops, Foundation day lectures, Distinguished Scientist Lectures held in CDFD both at Nampally and Gandipet. We were actively involved in conducting the "Microbial Biology Symposium" at Ramoji Film city, Hyderabad from 11th to 14th December 2012. We had maintained most of the equipment with maximum uptime in the Laboratory.

Details of progress made in the current reporting year (April 1, 2013 -March 31, 2014)

During the year 2013-14, we have installed 61 new equipments like Inverted Microscopes, PCR Machines, Refrigerated Centrifuges, Shaking waterbaths, -20°C Freezers, Cooled Incubator, Refrigerators etc. and we have also completed 498 work orders for repair & maintenance of various laboratory equipments.

We were involved in re-organizing the first floor Lab area and have shifted and re-installed many instruments including Illumina Bead Xpress Next Generation Genotyping System, Pyrosequencer, Laminar Hoods, Fume hood etc.

In addition, we were involved in organizing the audio & visual requirements for presentations in various seminars, lectures and workshops, Foundation day lectures, Distinguished Scientist Lectures. We were actively involved in conducting the Guha Research Conference at Araku Vally and Vizag from 7th to 10th December 2013 and "Young Investigator Meeting" at Ramoji Filmcity, Hyderabad from 8th to 12th February 2014. We have maintained most of the equipment with maximum uptime in the Laboratory. We have installed many instruments in the Animal Experimentation facility of CDFD at Vimta labs and maintaining them too. We are supervising the outsourcing of contracts of both Animal Experimentation facility and outsourcing of specialized instruments at CDFD.

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

* Publications of adjunct faculty of CDFD in which CDFD's affiliation is included.

** Work done elsewhere.

A. Publications during the year 2013

1. Acharya V and Nagarajaram HA (2013). Response to: Statistical analysis of missense mutation classifiers. **Human Mutation** 34: 407.
2. *Aggarwal S (2013). Skeletal dysplasias with increased bone density: evolution of molecular pathogenesis in the last century. **Gene** 528: 41-45
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4. Bairwa G, Balusu S and Kaur R (2013). Aspartyl proteases in human pathogenic fungi: roles in physiology and virulence in the book entitled "**The Fungal Cell Wall**", Editor: Héctor M. Mora-Montes, **Nova Science Publishers** 159-198.
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6. Bashyam MD and Raman R (2013). Molecular origins of colon and rectal cancer: Not a Wnt-Wnt situation. **Current Colorectal Cancer Reports** 9: 365-371.
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17. Gowrishankar J, Krishna Leela J and Anupama K (2013). R-loops in bacterial transcription: Their causes and consequences. **Transcription** 4: 153-157.

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- lepidopteran model insect, *Bombyx mori*. **G3: Genes, Genomes, Genetics** 3: 1481-1492.
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 - B. Publications in 2014 (Till March 31, 2014)**
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 60. Love JM, Prosser D, Love DR, Chintakindi KP, Dalal AB and Aggarwal S (2014). A novel glycine decarboxylase gene mutation in an Indian Family with nonketotic hyperglycinemia. **Journal of Child Neurology** 29:122-127.
 61. *Mon H, Lee JM, Mita K, Goldsmith MR and Kusakabe T (2014). Chromatin-induced spindle assembly plays an important role in metaphase congression of silkworm holocentric chromosomes. **Insect Biochemistry and Molecular Biology** 45: 40-50.
 62. Mudunuri SB, Patnana S and Nagarajaram HA (2014). MICdb3.0: a comprehensive resource of microsatellite repeats from prokaryotic genomes. **Database** doi:10.1093/database/bau005.
 63. Nagaraju J, Gopinath G, Sharma V and Shukla JN (2014). Lepidopteran sex determination: a cascade of surprises. **Sexual Development** 8: 104-112.
 64. Phulera S, Akif M, Sardesai AA and Mande SC (2014). Redox proteins of *Mycobacterium tuberculosis*. **Journal of the Indian Institute of Science** 94: 127-137.
 65. *Ramasarma T (2014). Trendy science research communications. **Current Science** 106: 506-508.
 66. Ratheesh R, Kotapalli V, Adduri R, Gowrishankar G, Bashyam L, Chaudhary AK, Chigurupati M, Patnaik S, Srinivasulu M, Sastry RA, Rao S, Vasala A, Kalidindi NR, Pollack JR, Murthy S and Bashyam MD (2014). Evidence for possible non-canonical pathway(s) driven early-onset colorectal cancer in India. **Molecular Carcinogenesis** 53: E181-E186.

67. Saranathan R, Tomar A, Sudhakar P, Arunkumar KP and Prashanth K (2014). Draft genome sequence of a MDR *Acinetobacter baumannii* PKAB 07 clinical strain belonging to ST 195 from India. **Genome Announcements** 2: e00184.
68. Sharma V and Nandineni MR (2014). Assessment of genetic diversity among Indian potato (*Solanum tuberosum* L.) collection using microsatellite and retrotransposon based marker systems. **Molecular Phylogenetics and Evolution** 73: 10-17.
- C. Publications in Press (as on March 31, 2014)**
69. Aggarwal S, Coutinho MF, Dalal AB, Mohamed Nurul Jain SJ, Prata MJ and Alves S. Prenatal skeletal dysplasia phenotype in severe MLII alpha/beta with novel GNPTAB mutation. **Gene**.
70. Anusha U, Ranganath P, Md Nurul Jain SJ, Krishna Prasad C, Sinha A, Verma IC, Phadke SR, Puri RD, Danda S, Muranjan MN, Jevalikar G, Nagarajaram HA and Dalal AB. Novel mutations of the ARSB gene in Indian patients with Mucopolysaccharidosis Type VI. **Indian Journal of Medical Research**.
71. Basu A, Dasari V, Mishra RK and Khosla S. The CpG island encompassing the promoter and first exon of human DNMT3L gene is a PcG/TrX response element (PRE). **PLoS One**.
72. Dutta UR, Ponnala R and Dalal A. A novel de novo balanced reciprocal translocation t(18;22) associated with recurrent miscarriages: A case report. **Journal of Reproduction and Infertility**.
73. Kantaputra PN, Kayserili H, Guven Y, Kantaputra W, Balci MC, Tanpaiboon P, Tananuvat N, Uttarilli A and Dalal A. Clinical manifestations of 17 patients affected with mucopolysaccharidosis type VI and eight novel ARSB mutations. **American Journal of Medical Genetics**.
74. Pandey SD, Choudhury M, Yousuf S, Wheeler PR, Gordon SV, Ranjan A and Sriharan M. Iron-regulated protein HupB of *Mycobacterium tuberculosis* positively regulates siderophore biosynthesis and is essential for growth in macrophages. **Journal of Bacteriology**.
75. Pradhan BB and Chatterjee S. Reversible non-genetic phenotypic heterogeneity in bacterial quorum sensing. **Molecular Microbiology**.
76. Rajashree N, Phadke SR, Dalal AB and Ranganath P. Novel mutations in the PRG4 gene in two Indian families with the Camptodactyly- arthropathy- coxa vara-pericarditis syndrome. **Indian Journal of Medical Research**.
77. Ratheesh R, Kongara R, Kotapalli V, Gowrishankar S, Sastry RA, Nagari B, Bashyam MD. Pathological stage significantly predicts survival in colorectal cancer patients: a study from two tertiary care centres in India. **Colorectal Cancer**.
78. Raviprakash N and Manna SK. Pulse exposure of cardiac glycoside enhances IL-8-mediated biological responses by increasing cell surface IL-8 receptors. **British Journal of Pharmacology**.
79. Satyavathi VV, Asha Minz and Nagaraju J. Nodulation: An unexplored cellular defense mechanism in insects. **Cellular Signalling**.
80. Shah AH, Singh A, Dhamgaye S, Chauhan N, Vandeputte P, Suneetha KJ, Kaur R, Mukherjee PK, Chandra J, Ghannoum MA, Sanglard D, Goswami SK and Prasad R. Novel role of a family of major facilitator transporters in biofilm development and virulence of *Candida albicans*. **Biochemical Journal**.
81. Singh CP, Singh J and Nagaraju J. Regulation of viral late genes by a baculovirus-encoded miRNA. **Insect Biochemistry and Molecular Biology**.
82. Singh CP, Vaishna RL, Kakkar A, Arunkumar KP and Nagaraju J. Characterization of antiviral and antibacterial activity of *Bombyx mori* seroin proteins. **Cellular Microbiology**.
83. Stephen J, Shukla A, Dalal A, Girisha KM, Shah H, Gupta N, Kabra M, Dabadghao P and Phadke SR. Mutation spectrum of COL1A1 and COL1A2 genes in Indian patients with osteogenesis imperfecta. **American Journal of Medical Genetics**.
84. Tsurusaki Y, Okamoto N, Ohashi H, Mizuno S, Matsumoto N, Makita Y, Fukuda M, Isidor B, Perrier J, Aggarwal S, Dalal AB, Al-Kindy A, Liebelt J, Mowat D, Nakashima M, Saitsu H, Miyake N and Matsumoto N. Coffin-Siris syndrome is a SWI/SNF complex disorder. **Clinical Genetics**.

D. Other Publications

1. Chatterjee S (2013). Review of: Annual Review of Microbiology, 2011. Susan Gottesman and Caroline S Harwood (eds). **Current Science** 104: 653-654.
2. Kasbekar DP (2013). Book review: "March of the microbes: Sighting the unseen". **Current Science** 104: 971.
3. Kasbekar DP (2013). Myth versus mutant: story of *o*. **Journal of Biosciences** 38: 1.
4. Kaur R (2013). Review of: Annual review of microbiology, 2012. Edited by Susan Gottesman, Caroline S. Harwood and Olaf Schneewind. **Current Science** 105: 390-391.
5. Rajashree N, Kumar SR, Ranganath P and Dalal A (2013). Banking of genetic material: A key to the future. **Genetic Clinics** 6: 12-15.
6. Dalal A (2014). Phenylketonuria: Past, present and future. **Genetic Clinics** 7: 19-24.
7. Kasbekar DP (2014). Editorial. Lesser models. **Journal of Biosciences** 39: 1.
8. Ranganath P and Dalal A (2014). Quality issues in medical genetics. **Genetics in Clinical Practice** 1st Edition: 237-243.

E. Patents

(a) Patents granted

1. Mukhopadhyay S, Bhat KH and Khan N. A novel protein as potential candidate for development anti-tuberculosis therapeutics.

US Patent Application No.US-12/551,115
Invention ID: IN-000044-02-US-REG
Patent No.: US-8603739B2
Date of grant: December 10, 2013

(b) Patents filed

1. Gowrishankar J and Shaffiqu TS. Treatment of hides or skins for leather manufacture.

Indian Patent Application No.: 5465/CHE/2013
Date of filing: November 27, 2013

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 streams). 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 March 31, 2014 the Centre has 99 Research Scholars working for their doctorates in different

areas of research. In the reporting year 8 of 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 or Jawaharlal Nehru Centre for Advanced Scientific Research, Bangalore or the Kishore Vigyanik Protsahan Yojna, New Delhi. In the reporting year 23 students 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-on experience in modern biology. In the reporting year, 7 students were given the opportunity to avail training under this programme.

Research Scholars Conferred PhD Degree During the Reporting Period

Scholar	Supervisor	Date of viva voce examination	Title of thesis
Jamshaid Ali	Akash Ranjan	03.05.2013	Computational annotation of typical apicomplexan proteins and biochemical studies of highly conserved plasmodium faciparum acyl CoA binding proteins
Md. Khursheed	MD Bashyam	21.06.2013	"Identification and functional characterization of novel pancreatic cancer gene(s)"
Sidharth Kumar Mahali	SK Manna	02.07.2013	Understanding advanced glycatin end product (Age) - mediated cell signaling and its regulation
Gaurav Bairwa	Rupinder Kaur	19.07.2013	"Studies on virulence factors of <i>Candida glabrata</i>
Syeda Aisha Haneea	J Gowrishankar	22.08.2013	Studies on transcription-translation coupling and the consequences of its failure in <i>Escherichia coli</i> : Tests for compromised genomic integrity and increased propensity for occurrence of RNA-DNA hybrids (R-loops) in rho and nusG mutants
Chandra Pal Singh	KP Arun Kumar	17.01.2014	Elucidation of microRNA(s) role in host-pathogen interaction in silkworm
Nazia Parveen	Sangita Mukhopadhyay	10.02.2014	Understanding <i>Mycobacterium tuberculosis</i> heat shock protein 60 (Mtbhsp60) mediated modulation of macrophage immune responses
Amitabh Ranjan	Ranjan Sen	25.02.2014	Studies on the factor dependent inhibition of Rho dependant termination

पुरस्कार एवं सम्मान
Awards and Honours

AWARDS & HONOURS

FACULTY & STAFF	
Dr Durgadas P Kasbekar	Indian National Science Academy Fellowship (2013)
Dr Murali D Bashyam	1. ICMR International Fellowship for Young Biomedical Scientists (2012-2013) 2. DBT National Bioscience Award for Career Development (2013)
Dr Sanjeev Khosla	Member, Guha Research Conference (2013)
Dr N Madhusudan Reddy	Indo-US Research Fellowship (2013)
Mr Binod B Pradhan	Best poster presentation at the 4th International Conference on Bacterial Blight of Rice (2013)
PhD STUDENTS & PROJECT PERSONNEL	
Dr Khalid Hussain Bhat	1. Best Oral Presentation Award at the 40th Annual Conference of Indian Immunology Society (IMMUNOCON)-2013 2. Young Scientist Award by the Andhra Pradesh Akademi of Sciences (2014)
Dr Nirotpal Mrinal	INSA Young Scientist Medal (2013)
Dr Shiny Nair	INSA Young Scientist Medal (2013)
Mr Aamir Ali	Best Poster Award at the 82nd Annual Meeting of the Society of Biological Chemists (India) (2013)
Mr PV Vivek Reddy	DST-Raman Charpak Fellowship (2013)
Mr Ratheesh Raman	CSIR Travel Award to attend the Annual meeting of the American Association of Cancer Research (2013)
Mr Saurabh Mishra	DP Burma Best Poster Award at the 82nd Annual Meeting of the Society of Biological Chemists (India) (2013)
Ms Narmadha Reddy	Dr KV Rao Research Award (2013-14)
Mr Ajay Kumar Chaudhary	Young Scientist Travel Award of the Indian Society of Human Genetics (ISHG) Conference (2014)
Ms Manasa Chanduri	Best Presentation Award at the 1st AIST International Imaging Workshop at the Biomedical Research Institute, Japan (2014)
Mr Raju Adduri	GP Talwar Immunology Foundation Award of Travel Bursary (2014)

**व्याख्यान, बैठक, कार्यशाला व
अन्य महत्वपूर्ण कार्यक्रम**

**Lectures, Meetings, Workshops
and Important Events**

DISTINGUISHED VISITORS AND LECTURES

Visitor	Title of Lecture	Date
Dr Avinash R Shenoy Yale University, USA	Immunity "On demand": Antimicrobial defences via inducible GTPases	23.04.2013
Dr Souvik Mukherjee National Institute of Biomedical Genomics, Kalyani, India	Signatures of natural selection on human immunity genes and its importance in disease association across populations	10.05.2013
Dr Amartya Sanyal The University of Massachusetts Medical School, USA	The long-range interaction landscape of gene promoters	05.06.2013
Dr Geetanjali Chawla The Indiana University, USA	Regulation of <i>Drosophila</i> let-7-Complex micro RNAs and their role in neuro-degeneration	12.06.2013
Dr Jorg Dojahn AB SCIEX, Germany	Recent trends and developments in mass spectrometric workflows for proteomics	24.06.2013
Dr Saumyadipta Pyne CR Rao Advanced Institute of Mathematics, Statistics & Computer Science, Hyderabad India	Big data in bio-medicine: Handling stochasticity, heterogeneity and noise	02.07.2013
Dr Anil K Ojha University of Pittsburgh, USA	The Yin and Yang of lipid esterases in chronic infection of <i>Mycobacterium tuberculosis</i>	12.07.2013
Dr. Thirumananseri Kumarevel RIKEN SPring-8 Center, Harima Institute, Japan	Structural and functional analysis of histone variants involved in reprogramming	16.07.2013
Dr Gautam V Soni Kavli Institute of Nanoscience The Netherlands	Nanopore biophysics: From gene sequencing to gene silencing	17.07.2013
Dr Arun Kumar Shukla Duke University, USA	Structural basis of β -arrestin dependent regulation and signaling of G protein-coupled receptors	30.07.2013
Dr Santosh Chauhan University of New Mexico, USA	Transcriptional and epigenetic regulation of genes involved in dormancy, cancer and autophagy	16.08.2013
Dr Prim Singh Universitatmedizin Berlin, Germany	Heterochromatin, epigenetics and age reprogramming	19.08.2013

Visitor	Title of Lecture	Date
Dr Gopinath M Institute of Medical Biology, Singapore	Decoding the role of non-coding RNAs in skin physiology and pathology	13.09.2013
Dr Chandra P Chaturvedi The Sprott Centre for Stem Cell Research, The Ottawa Hospital, Canada	Functional role of histone methyltransferase G9a in regulating gene expression program in adult erythroid cells	24.09.2013
Prof Jorg Vogel Institute for Molecular Infection Biology, University of Wurzburg, Germany	An RNA perspective on bad microbes and their hosts	21.10.2013
Prof Steve Busby University of Birmingham, UK	Regulation at simple and complex bacterial promoters	21.10.2013
Dr Bianca Sclavi Laboratory of Biology & Applied Pharmacology, ENS, Cachan, France	Quantitative characterization of the DnaA-dependent transcription network. Coordination of gene expression and DNA replication	28.10.2013
Dr Arati Ramesh University of Texas Southwestern Medical Center, USA	RNA-mediated gene-regulation in bacteria	27.11.2013
Dr Sunil Laxman University of Texas Southwestern Medical Center, USA	From Starvation to Satiety: How sulfur amino acids control cell growth	02.12.2013
Dr Vittorio Venturi ICGEB Trieste, Italy	Rice-Xanthomonas interaction	05.12.2013
Prof Titia Sixma Division of Biochemistry Netherlands Cancer Institute, The Netherlands	Protein-protein interactions regulate ubiquitin conjugation in the DNA damage response	16.12.2013
Dr Areejit Samal University of California, USA	Reconstruction and systems analysis of plant cell wall deconstruction network in filamentous fungus <i>Neurospora crassa</i>	23.12.2013
Dr ALN Rao University of California, USA	Analysis of RNA-protein interactome regulating the replication of Cucumber mosaic virus and its satellite RNA	07.01.2014

Visitor	Title of Lecture	Date
Dr Navratna Vajpai Astra Zeneca Pharmaceuticals, UK	Solution NMR and structure biology: kinase-ligand structure and dynamics	30.01.2014
Dr Syamal Roy Indian Institute of Chemical Biology, Kolkata, India	Poor stability of peptide-MHC-II complex may specify defective cellular immunity in visceral Leishmaniasis	10.02.2014
Prof Leonard Rabinow University of Paris, Paris	Linking sex and death: DOA protein kinase of Drosophila	11.02.2014
Dr TS Suryanarayanan Vivekananda Institute of Tropical Mycology (VINSTROM), Chennai, India	Need for research collaborations in mycology: the endophyte example	12.02.2014
Prof Ashok Venkitaraman The Ursula Zoellner Professor of Cancer Research, University of Cambridge, USA	Macromolecular logistics in the control of genome stability	13.02.2014
Dr Swadhin Jana Monica Bettencourt-Dias Lab, Institute Gulbekian de Ciencia, Portugal	How do cilia become morphologically diverse?	13.02.2014
Dr Pankaj Kumar University of Virginia, USA	MicroDNA and its potential role in genome variation	14.02.2014
Dr Uma Devi Paila Centre for Public Health Sciences, USA	Integrative Exploration of Human Genetics Variation in Disease	14.02.2014
Dr Gyanendra P Dubey The Hebrew University of Jerusalem, Israel	Identification and Characterization of Bacterial Nanotubes	14.02.2014
Dr Ravi Gupta Sci Genom Labs, Cochin, India	NGS technologies changing genomics landscape- Pushing study from gene to genome	21.02.2014
Prof Shamshad Cockcroft Dept. of Neurosciences, Physiology and Pharmacology, University College London (UCL), UK	Lipid Transfer proteins in Phosphoinositide Signalling and membrane traffic	03.03.2014

IMPORTANT EVENTS

Event	Partnering Institutions	Date
27 th Meeting of CDFD Finance Committee		05.04.2013
21 st Meeting of CDFD Building Committee		05.04.2013
33 rd Meeting of CDFD Governing Council		19.04.2013
Fire drill		19.06.2013 - 20.06.2013
Visit of Shri Vidhya Sagar, Legal Officer, CAG, Department of Expenditure, Ministry of Finance, New Delhi.		20.06.2013
Summer Trainee's Colloquium		24.06.2013
Training Program on Medical Laboratory Management Systems & Internal Audit for Diagnostics Division of CDFD	Bureau of Indian Standards (BIS) and CDFD	25.06.2013 - 28.06.2013
15 th Meeting of CDFD Research Area Panels-Scientific Advisory Committee (RAP-SAC)		26.07.2013 - 27.07.2013
Independence Day Celebrations		15.08.2013
Sadbhavana Diwas Pledge		20.08.2013
Hindi Pakhwada Celebrations		14.09.2013
Official Language Implementation Committee (OLIC) Meeting		16.09.2013
Visit of Dr Joan Keutzer, Vice President and Head of Global Scientific Affairs, Rare Diseases Unit at Genzyme, A Sanofi Company, Hyderabad		01.10.2013
MoU to provide DNA Fingerprinting Services	Forensic Science Laboratory, Madhya Pradesh and CDFD	10.10.2013

Event	Partnering Institutions	Date
28 th Meeting of the CDFD Finance Committee		15.10.2013
34 th Meeting of CDFD Governing Council		15.10.2013
Vigilance Awareness Week		28.10.2013
18 th Meeting of CDFD Society		18.11.2013
Visit of Mr Christof Kuhstoß, representative at European Research Council (ERC) in Deutsche Forschungsgemeinschaft (DFG), Bonn		26.11.2013
Visit of Dr Suresh Madhivanan, La Trobe University, Australia		28.11.2013
Guha Research Conference (GRC)-2014 at Araku Valley, Visakhapatnam	Centre for Cellular Molecular Biology (CCMB), University of Hyderabad (UoH) and (CDFD)	06.12.2013 –10.12.2013
Renewal of MoU with NIMS, Hyderabad		20.12.2013
Institutional Bioethics Committee Meeting		16.01.2014
Republic Day celebrations		26.01.2014
MoU with Government of Uttar Pradesh, UP, Lucknow	Police Technical Services, UP, Lucknow; Forensic Science Laboratory, UP, Lucknow and CDFD	27.01.2014
CDFD Foundation Day Celebrations		28.01.2014
Visit of Shri BN Satpathy, Senior Advisor, Planning Commission		05.02.2014
Young Investigator Meeting (YIM) at Ramoji Film City	India BioScience and CDFD	08.02.2014 - 12.02.2014

Event	Partnering Institutions	Date
Visit of Dr Lino Barañao, Hon'ble Minister of Science, Technology & Productive Innovation of Argentina		17.02.2014
Visit of Prof Frank Gannon, CEO, Queensland Institute of Medical Research (QIMR), Australia, along with senior faculty Prof Rajiv Khanna		17.02.2014
2 nd Meeting of the Academic Committee		13.03.2014
Visit of Dr Patrik Stolt from Scan Bi Diagnostics, Sweden along with Dr Lalitha Gowda from CFTRI and Dr Murali Krishna from BCIL under "Phase II Capacity Building Project on Biosafety under UNEP/GEF supported project."	Central Food Technological Research Institute (CFTRI), Biotech Consortium India Limited (BCIL), and Ministry of Environment & Forests (MoEF)	18.03.2014
MoU to institute a fellowship in Clinical Diagnostics	Society for Indian Academy of Medical Genetics, Lucknow, UP	19.03.2014
35 th Meeting of CDFD Governing Council		25.03.2014
22 nd Meeting of CDFD Building Committee		25.03.2014
29 th Meeting of the CDFD Finance Committee		25.03.2014

सी डी एफ डी कर्मचारियों की
विदेशों में प्रतिनियुक्ति
**Deputations Abroad of
CDFD Personnel**

DEPUTATIONS ABROAD - FACULTY & STAFF

Faculty/Staff	Period	Country of Visit and Purpose
J Gowrishankar	07.04.2013 to 10.04.2013	UK : to chair the Indian delegation of the Bureau of Indian Standards to participate in the fourth plenary meeting of ISO/TC 34/SC 16 'Horizontal methods for molecular biomarker analysis' at London
	06.08.2013 to 13.08.2013	USA : (i) to attend the "2013 Molecular Genetics of Bacteria and Phages Meeting" at University of Wisconsin, Madison, USA (ii) to visit the laboratories of Prof. Max E Gottesman, Prof. Anuradha Janakiraman and Prof. EA Nudler in New York
	14.11.2013 to 16.11.2013	Singapore : (i) to participate and to speak as Panel Member at the First Indo-Singapore Workshop 2013 on 'Advances in Chemistry, Biology and Technology for Medicine' that is being jointly organized by DBT and A*STAR Singapore (ii) to visit faculty of the Genome Institute of Singapore
	19.02.2014 to 05.03.2014	Japan : to visit the laboratory of Dr. Tomohiro Shimada (Japanese Investigator), Tokyo Institute of Technology, in connection with implementation of the joint India-Japan research project with Dr. Tomohiro titled "Analysis of co-regulation between DNA replication and amino acid homeostasis by transcription factor LciA / ArgP in <i>Escherichia coli</i> "
DP Kasbekar	02.03.2014 to 15.03.2014	USA : (i) to attend the Neurospora 2014 conference at Asilomar, CA, USA and present his talk (ii) to present his seminar at the School of Plant Sciences, University of Arizona (UofA), Tucson, USA
Ranjan Sen	22.06.2013 to 29.06.2013	USA : to attend the FASEB Summer Conference on "Mechanisms & Regulation of Prokaryotic Transcription" at Vermont Academy, Saxtons River, Vermont, USA.
	26.09.2013 to 03.10.2013	USA : (i) to attend YIM-2013 meeting at Boston. (ii) To visit laboratories of Profs. Max Gottesman and Nudler in New York City.
Murali Dharan Bashyam	06.04.2013 to 10.04.2013	USA : to attend the AACR annual meeting at Washington DC, USA to interact with cancer genomics / translational cancer research experts and also to present latest data from his laboratory.

Faculty/Staff	Period	Country of Visit and Purpose
Nagarajaram HA	27.01.2014 to 05.02.2014	Germany: to attend 1 st HCV-workshop cum exchange visit under New INDIGO project “An attractive and promising strategy for early cancer diagnosis through the assembly of the human cancer volatome” at Rostock University Medical Centre, Rostock, Germany.
Ashwin B Dalal	12.06.2013 to 15.06.2013	Srilanka: (i) to attend the “International Workshop and Symposium on intergrating Genetics in the Medical Curriculum” at Faculty of Medicine, University of Colombo, Colombo, Srilanka. (ii) To visit Diagnostic Unit in the laboratory of Dr. Ranil D Silva, Department of Anatomy, Faculty of Medical Sciences, University of Sri Jayewardenepura (USJP), Nugegoda, Sri Lanka.
N Madhusudan Reddy	15.04.2013 to 20.05.2013 14.10.2013 to 15.02.2014	Germany: to conduct research as Guest Scientist in the laboratory of Prof. Mark Stoneking, Professor for Biological Anthropology, Department of Evolutionary Genetics, Max Planck Institute for Evolutionary Anthropology, Leipzig, Germany against his third 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. USA: to visit the laboratory of Prof. Arthur Eisenberg, Professor and Chairman, Department of Forensic and Investigative Genetics, University of North Texas Health Science Center, Fort Worth, Texas, USA for conducting research work in the area of forensic DNA profiling as a part of Indo-US Research Fellowship.
Sardesai Abhijit Ajit	18.01.2014 to 05.02.2014	Japan: For furthering the Collaborative Research Project under the Indo-Japan S&T Cooperation between the Laboratory of Bacterial Genetics, CDFD and the Laboratory of Dr. Tomohiro Shimada at the Tokyo Institute of Technology (Titech), Yokohama, Japan.
Ankkur Goel	19.07.2013 to 05.08.2013	USA: to attend Khorana Program Technology Transfer Course - 2013 at the University of Wisconsin – Madison (UW), USA.

Faculty/Staff	Period	Country of Visit and Purpose
Venkata Satyavathi	08.11.2013 to 12.11.2013	Malaysia: to present her paper entitled "Identification of long noncoding RNAs (lncRNAs) involved in Immune response during baculoviral infection in Bombyx mori" in the Global Conference on Entomology at Four Points by Sheraton, Kuching, Malaysia.
Shweta Tyagi	07.05.2013 to 12.05.2013	Germany: to attend and present her work at the conference titled "Chromatin and Epigenetics" at EMBL Heidelberg, Germany.
Arun Kumar K P	29.10.2013 to 13.11.2013 05.01.2014 to 08.01.2014	France: To visit as a part of his collaborative research work under the Indo French Centre for the Promotion of Advanced Research (CEFIPRA / IFCPAR) project on "Global transcriptomics of sex-specific splicing" as an exchange visit to the laboratory of Prof. Leonard Rabinow at the Centre de Neurosciences de Paris Sud, Orsay cedex, France. China: To attend "the International Meeting on Spodoptera litura Genome Project" at the State Key Laboratory of Silkworm Genome Biology, Southwest University, Beibei, Chongqing, China and to deliver a talk entitled "Towards physical mapping of Spodoptera litura genome using RAD-seq and SNPs and also to discuss the present status of S litura genome works
Archana Tomar	10.11.2013 to 24.11.2013	Japan: To visit University of Tokyo and National Institute of Agrobiological Sciences, Tsukuba under India-Japan Cooperative Science Programme (IJCSP)

DEPUTATIONS ABROAD - STUDENTS

Name of the Scholar	Period	Country of Visit and Purpose
Asha Minz	03.04.2013 to 09.04.2013	USA: Drosophila Genetics: 54th Annual Drosophila Research Conference
Anujit Sarkar	08.05.2013 to 20.06.2013	Germany: Visit to the laboratory of Prof. Max Stoneking, Max Planck Institute of Evolutionary Anthrapology, Leipzig, Germany (MPI – EVA)
Manjari	19.07.2013 to 23.07.2013	Germany: ISMB / ECCB 2013 Conference
Swarna Gowri Thota	05.11.2013 to 10.11.2013	USA: Cell Biology of Yeasts
Sapan Borah	05.11.2013 to 09.11.2013	USA: Cell Biology of Yeasts
Gajula Gopinath	10.11.2013 to 24.11.2013	Japan: Visit to the University of Tokyo, Tokyo and (b) National Institute of Agrobiological Sciences, Tsukuba under DST sponsored India – Japan Cooperative Science Programme (JCSP)
Nazia Parveen	20.11.2013 to 24.11.2013	USA: Harnessing Immunity prevent and treat disease” conference
PV Vivek Reddy	01.02.2014 to 30.07.2014	France: To work in the laboratory of Dr. Evi Soutoglou, IGBMC, Strasbourg, France
CVL Manasa	19.01.2014 to 25.01.2014	Japan: 1 st AIST International Imaging Workshop 2014
Rakesh Trivedi	27.01.2014 to 28.02.2014	Germany: HCV – Workshop cum exchange visit and Training

सीडीएफडी के संकाय एवं अधिकारी
Faculty and Officers of CDFD

SCIENTIFIC GROUP LEADERS (FACULTY)

Dr J Gowrishankar
Dr DP Kasbekar
Dr Ranjan Sen
Dr Sunil Kumar Manna
Dr Sangita Mukhopadhyay
Dr MD Bashyam
Dr Nagarajaram HA
Dr Akash Ranjan
Dr Rupinder Kaur
Dr Sanjeev Khosla
Dr Ashwin B Dalal
Dr Rashna Bhandari
Dr Devyani Halder
Dr N Madhusudan Reddy
Dr Subhadeep Chatterjee
Dr Sardesai Abhijit Ajit
Dr R Harinarayanan
Dr Shweta Tyagi
Dr Rohit Joshi
Dr MV Subba Reddy
Dr Arun Kumar KP

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
Ms M Kavita Rao
Dr Ankkur Goel (Till 31.10.2013)

SENIOR ADMINISTRATIVE STAFF

Mr J Sanjeev Rao
Mr B Jagannathacharyulu
Mr S Ayub Basha

केन्द्र की समितियाँ

(31.03.2014 तक)

Committees of the Centre

(As on 31.03.2014)

MEMBERS OF CDFD SOCIETY

Shri S Jaipal Reddy Hon'ble Minister for S&T and Earth Sciences	-	President
Prof K VijayRaghavan Secretary, DBT, New Delhi	-	Member
Prof Samir K Brahmachari Director General, CSIR, New Delhi	-	Member (Ex-officio)
Prof P Balaram Director, IISc, Bangalore	-	Member (Ex-officio)
Prof VS Chauhan Director, ICGEB, New Delhi	-	Member
Prof Dipankar Chatterji IISc, Bangalore	-	Member
Shri Inder Kumar Joint Secretary & Legal Adviser Ministry of Law, Justice & Company Affairs, New Delhi	-	Member (Ex-officio)
Shri J R Gaur PSO, BPR&D, New Delhi (Nominee of Director General, BPR&D)	-	Member (Ex-officio)
Joint Secretary (PM) Ministry of Home Affairs, New Delhi	-	Member (Ex-officio)
Ms Anuradha Mitra JS & FA, DBT, New Delhi	-	Member (Ex-officio)
Dr Suman Govil Adviser, DBT, New Delhi	-	Member (Ex-officio)
Dr J Gowrishankar Director, CDFD, Hyderabad	-	Member Secretary

MEMBERS OF CDFD GOVERNING COUNCIL

Prof K VijayRaghavan Secretary, DBT, New Delhi	-	Chairperson
Prof Samir K Brahmachari Director General, CSIR, New Delhi	-	Member (Ex-officio)
Prof P Balaram Director, IISc, Bangalore	-	Member (Ex-officio)
Prof V S Chauhan Director, ICGEB, New Delhi	-	Member
Prof Dipankar Chatterji IISc, Bangalore	-	Member
Mr V Venugopal Director, CFSL, Hyderabad (Nominee of Joint Secretary (PM))	-	Member (Ex-officio)
Mr O Venkateswarlu Dy. Legal Adviser, Ministry of Law, New Delhi (Nominee of Joint Secretary & Legal Adviser)	-	Member (Ex-officio)
Ms Anuradha Mitra Jt. Secretary & Financial Advisor, DBT, New Delhi	-	Member (Ex-officio)
Mr Radhakrishna Kini A Addl. Director General, BPR & D, New Delhi (Nominee of Director General, Bureau of Police Research and Development)	-	Member (Ex-officio)
Dr Suman Govil Adviser, DBT, New Delhi	-	Member (Ex-officio)
Dr J Gowrishankar Director, CDFD, Hyderabad	-	Member Secretary

MEMBERS OF CDFD RESEARCH AREA PANELS – SCIENTIFIC ADVISORY COMMITTEE (RAP-SAC)

Prof P Balaram Director, IISc, Bangalore	-	Chairman
Dr Ramakrishna Ramaswamy UoH, Hyderabad	-	Member
Dr Veena K Parnaik CCMB, Hyderabad	-	Member
Dr SK Apte BARC, Mumbai	-	Member
Dr Ghanshyam Swarup CCMB, Hyderabad	-	Member
Dr Sandhya S Visweswaraiah IISc, Bangalore	-	Member
Dr Usha Vijayraghavan IISc, Bangalore	-	Member
Prof Sanjeev Galande IISER, Pune	-	Member
Dr Chetan E Chitnis ICGEB, New Delhi	-	Member
Dr Jaya Sivaswami Tyagi AIIMS, New Delhi	-	Member
Dr Joyoti Basu Bose Institute, Kolkata	-	Member
Dr Debasisa Mohanty NII, New Delhi	-	Member
Prof MK Mathew NCBS, Bangalore	-	Member
Dr Shubha R Phadke SGPGI, Lucknow	-	Member
Prof Umesh Varshney IISc, Bangalore	-	Member
Dr Suman Govil DBT, New Delhi (Nominee of DBT)	-	Member
Dr K V Prabhu IARI, New Delhi (Nominee of ICAR)	-	Member
Dr K Ghosh NII, Mumbai (Nominee of ICMR)	-	Member
Ministry of Home Affairs, New Delhi	-	Member
Dr J Gowrishankar Director, CDFD, Hyderabad	-	Member Secretary

MEMBERS OF CDFD ACADEMIC COMMITTEE

Prof AS Raghavendra Dean, School of Life Sciences University of Hyderabad,	-	Chairman
Prof Anil K Tyagi University of Delhi, South Campus, New Delhi	-	Member
Dr K Satyamoorthy Director, Manipal Life Sciences Centre Manipal University, Manipal	-	Member
Dr DP Kasbekar Haldane Chair, CDFD, Hyderabad	-	Member
Dr Ranjan Sen Staff Scientist, CDFD, Hyderabad	-	Member
Dr Sanjeev Khosla Staff Scientist & Co-ordinator (Academics) CDFD, Hyderabad	-	Member Convenor

MEMBERS OF THE INSTITUTIONAL BIO-SAFETY COMMITTEE

Dr D P Kasbekar Haldane Chair, CDFD, Hyderabad (Nominee of Director, CDFD)	-	Chairman
Dr Rupinder Kaur Staff Scientist, CDFD, Hyderabad	-	Member Secretary
Dr Ashwin B Dalal Staff Scientist, CDFD, Hyderabad	-	Biosafety Officer
Dr Murali Dharan Bashyam Staff Scientist, CDFD, Hyderabad	-	CDFD Expert
Dr Subhadeep Chatterjee Staff Scientist, CDFD, Hyderabad	-	CDFD Expert
Dr Ashok Khar Former Director, CMBRC, Appollo Hospitals Educational and Research Foundation	-	Outside Expert
Dr Manjula Reddy Senior Principal Scientist, CCMB, Hyderabad	-	DBT Nominee

MEMBERS OF CDFD BUILDING COMMITTEE

Prof VS Chauhan Director, ICGEB, New Delhi	-	Chairman
Dr J Gowrishankar Director, CDFD, Hyderabad	-	Member
Shri S Raghavan Joint Secretary, DBT, New Delhi	-	Member
Shri VH Rao Senior Consultant, NIAB, Hyderabad	-	Member
Shri J Sanjeev Rao Head-Administration, CDFD, Hyderabad	-	Member
Shri BJ Acharyulu Head-F&A, CDFD, Hyderabad	-	Member
Dr V Phani Sree Associate Professor & HOD, Planning JANFAU, Hyderabad	-	Member
Shri BLN Reddy Superintending Engineer, HMDA, Hyderabad	-	Member
Shri V Punnaiah Executive Engineer, CDFD, Hyderabad	-	Member Convenor

MEMBERS OF CDFD MANAGEMENT COMMITTEE

Dr J Gowrishankar Director, CDFD, Hyderabad	-	Chairman
Dr DP Kasbekar Haldane Chair, CDFD	-	Member
Dr Ranjan Sen Staff Scientist, CDFD, Hyderabad	-	Member
Dr MV Subba Reddy Staff Scientist, CDFD, Hyderabad	-	Member
Shri BJ Acharyulu Head-F&A, CDFD, Hyderabad	-	Member
Shri J Sanjeev Rao Head-Administration, CDFD, Hyderabad	-	Member Convenor

MEMBERS OF CDFD FINANCE COMMITTEE

Prof VS Chauhan Director, ICGEB, New Delhi	-	Chairman
Dr Dipankar Chatterji IISc, Bangalore	-	Member
Ms Anuradha Mitra JS&FA, DBT, New Delhi	-	Member
Dr Suman Govil Advisor, DBT, New Delhi	-	Member
Dr J Gowrishankar Director, CDFD, Hyderabad	-	Member
Shri BJ Acharyulu Head-F&A, CDFD, Hyderabad	-	Member Convenor

MEMBERS OF SEXUAL HARASSMENT COMPLAINTS COMMITTEE

Dr Sangita Mukhopadhyay Staff Scientist, CDFD, Hyderabad	-	Chairman
Mr J Sanjeev Rao Head – Administration, CDFD, Hyderabad	-	Member
Ms V Naga Sailaja Technical Officer, CDFD, Hyderabad	-	Member
Ms MV Sukanya Technical Officer, CDFD, Hyderabad	-	Member
Mr MSA Zaman Khan Section Officer, CDFD, Hyderabad	-	Member
Ms P Jamuna Gramya Resource Centre for Women (representing an NGO)	-	Member

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

IMPLEMENTATION OF RIGHT TO INFORMATION (RTI) ACT, 2005

Appellate Authority : J Sanjeev Rao

Central Public Information Officer : M Kavita Rao

Details about the RTI applications and appeals received in CDFD

As received under the RTI Act 2005	Opening Balance an on 01-04-2013	Received during the year 2013-14			Disposed off during the year 2013-14				Closing Balance as on 31-03-14
		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	0	22	14	36	33	3	0	36	0
Appeals	0	2	Not applicable	2	0	2	Not applicable	2	0

बजट एवं वित्त
Budget and Finance

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS HYDERABAD

Budget & Finance 2013-14

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 2013-14

Particulars	Amount in Lakhs	Percentage- %
Plan Grant in Aid	3900.98	75.95
Sponsored Projects	743.60	16.61
CDFD Services	64.08	1.43
Misc Receipts	268.84	6.01
Total	4476.50	100.00

I. Application of Funds during 2013-14 (Plan Grant-in-Aid)

S.No.	Particulars	Amount in Lakhs	Percentage- %
1	Recurring		
	GIA- Salaries	1069.52	28.08
	GIA-General	1696.12	44.44
	Total	2762.64	72.52
2	Non-Recurring		
	GIA- Capital	1046.94	27.48
	Total	1046.94	27.48
	Grand Total	3809.58	100.00

II. Application of Funds during 2013-14 (Extra Mural Projects)

S No	Particulars	Amount in Lakhs	Percentage- %
1	Recurring		
	Salaries	318.84	29.89
	General	498.41	46.73
	Total	817.25	76.62
2	Non-Recurring		
	Capital	249.39	23.38
	Total	249.39	23.38
	Grand Total	1066.64	100.00

लेखा परिक्षक की रिपोर्ट
Auditor's Report

K R Srinivasan & Co

Chartered Accountants

AUDITOR'S REPORT

Date: 18-06-2014

The Director,
Centre for DNA Fingerprinting and Diagnostics,
Nampally, Hyderabad - 500 001

We have audited the attached Balance Sheet of CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, Hyderabad, as at 31st March 2014 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 are 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 those 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 give a true and fair view.
 - a) In so far it relates to the Balance sheet as at 31st March 2014 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 2014.

for **K R Srinivasan & Co**
Chartered Accountants

[K R SRINIVASAN]

Place: Hyderabad

Date: 18/06/2014

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
BALANCE SHEET AS ON 31st MARCH, 2014

(Amount - Rs.)

CORPUS/CAPITAL FUND AND LIABILITIES	Schedule	Current Year	Previous Year
Corpus / Capital Fund	1	1169815289	1142536939.00
Reserves and Surplus	2	0.00	0.00
Earmarked / Endowment funds	3	0.00	6531021.00
Secured Loans & Borrowings	4	0.00	0.00
Unsecured Loans & Borrowings	5	0.00	0.00
Deferred Credit Liabilities	6	0.00	0.00
Current Liabilities and Provisions	7	70814398.00	64750516.00
TOTAL		1240629687.00	1213818476.00
ASSETS			
Fixed Assets	8	966768793.00	932133417.00
Investments- From Earmarked / Endowment Funds	9	19398273.00	62398273.00
Investments - Others	10	23131298.00	25159583.00
Current Assets, Loans, Advances etc.	11	231331323.00	194127203.00
TOTAL		1240629687.00	1213818476.00
Significant Accounting Policies	24	0.00	0.00
Contingent Liabilities and Notes on Accounts	25		
DIRECTOR CDFD	HEAD - FINANCE & ACCOUNTS CDFD		
	For K R SRINIVASAN & CO CHARTERED ACCOUNTANTS (K R SRINIVASAN)		

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD				
INCOME AND EXPENDITURE ACCOUNT FOR THE YEAR ENDING 31st MARCH, 2014				
(Amount - Rs.)				
INCOME	Schedule	Current Year	Previous Year	
Income from Sales/Services	12	6408041.00	3571262.00	
Grants/Subsides	13	250932400.00	210000000.00	
Fees/Subscriptions	14	0.00	0.00	
Income from Investments	15	23220086.00	2768470.00	
Income from Royalty, Publications etc.	16	0.00	0.00	
Interest Earned	17	43238.00	700706.00	
Other Income	18	3620866.00	1350941.00	
Increase/(decrease) in stock of Finished goods and works-in-progress	19	0.00	0.00	
TOTAL (A)		284224631.00	218391379.00	
EXPENDITURE				
Establishment Expenses	20	106712459.00	94843804.00	
Administrative Expenses	21	177267101.00	143988338.00	
Expenditure on Grants, Subsides etc.	22	0.00	0.00	
Interest	23	0.00	0.00	
Depreciation (Net Total at the year-end -corresponding to Schedule 8)			21763702.00	
Less: Transferred to Grants-in-Aid			21763702.00	
Provision For Salaries		3392051.00	6932849.00	
TOTAL (B)		287371611.00	245764991.00	
Balance being excess of Income over Expenditure (A-B)		3146980.00	27373612.00	
Transfer to Special Reserve (Specify each)				
Transfer to/from General Reserve				
BALANCE BEING SURPLUS/(DEFLECT) CARRIED TO	24			
CORPUS/CAPITAL FUND	25			
SIGNIFICANT ACCOUNTING POLICIES				
CONTINGENT LIABILITIES AND NOTES ON ACCOUNTS				
DIRECTOR				HEAD - FINANCE & ACCOUNTS
CDFD				CDFD
				For K R SRINIVASAN & CO
				CHARTERED ACCOUNTANTS
				(K R SRINIVASAN)

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
RECEIPTS AND PAYMENTS ACCOUNT FOR THE YEAR ENDED 31st MARCH 2014					
(Amount - Rs.)					
RECEIPTS	Current Year	Previous Year	PAYMENTS	Current Year	Previous Year
1. Opening Balances			1. Expenses		
a) Cash in hand			a) Establishment Expenses (corresponding to Schedule 20)	106712459.00	94843804.00
b) Bank Balances			b) Administrative Expenses (corresponding to Schedule 21)	177267101.00	143988338.00
i) In current accounts	12223805.10	12333378.80	c) Schedule 22	0.00	0.00
ii) In deposit accounts					
iii) Savings accounts	20909457.77	73301897.29			
2. Grants Received			2. Payments made against funds for various projects		
a) From Government of India	340932400.00	390000000.00	(Name of the fund or project should be shown along with the particulars of payments made for each project)		
b) From State government			Projects (Annexure F)	106664828.00	102820071.00
c) From other sources (details)			CSIR(Stipend)	10743372.00	12496276.00
(Grants for capital & revenue exp. To be shown seperately)			DBT(Stipend)	4534065.00	4595379.00
Research Associates - CSIR(Stipend)	5567737.00	13650331.00	DST(Stipend)	739200.00	527012.00
Research Associates - DBT(Stipend)	5292736.00	1292280.00	ICMR(Stipend)	2901926.00	2079781.00
Research Associates - DST(Stipend)	250400.00	250400.00	IISC(Stipend)	1578752.00	3083960.00
Research Associates - ICMR(Stipend)	2830106.00	2422008.00	UGC(Stipend)	5150772.00	5164427.00
Research Associates - IISC(Stipend)	424400.00	3693877.00			
Research Associates - UGC(Stipend)	0.00	5473330.00			
Projects (Annexure - C)	74360025.00	58652921.00	3. Investments and deposits made		
			a) Out of Earmarked/Endowment funds	203000000.00	190000000.00
			b) Out of Own Funds (Investments-Others)	0.00	0.00
DIRECTOR	For K R SRINIVASAN & CO				HEAD - FINANCE & ACCOUNTS
CDFD	CHARTERED ACCOUNTANTS				CDFD
	(K R SRINIVASAN)				

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
RECEIPTS AND PAYMENTS ACCOUNT FOR THE YEAR ENDED 31st MARCH 2014					
(Amount - Rs.)					
RECEIPTS	Current Year	Previous Year	PAYMENTS	Current Year	Previous Year
3. Income on Investments from					
a) Earmarked/Endow. Funds	23220086.49	2768470.13	4. Expenditure on Fixed Assets & Capital		
b) Own Funds (Oth. Investment)			a) Purchases of Fixed Assets:		
Investments EnCash	246000000.00	190000000.00	Work-in-Progress Books & Journals	562565.00	707417.00
			Equipment -Lab/Office/Furniture	33772456.00	45729192.00
4. Interest Received			b) Expenditure on Capital Work-in-Progress:	59875026.00	143151561.00
a) On Bank deposits	0.00	0.00			
b) Loans, Advances etc			5. Refund of surplus money/Loans		
Interest on LC	43238.00	700706.00	a) To the Government of India	0.00	0.00
Interest on Computer Advance,	12488.00	21531.00	b) To the State Government	0.00	0.00
Conveyance Advance and HBA			c) To other providers of funds	0.00	0.00
5. Other Income(Specify)			6. Finance Charges (Interest)	0.00	0.00
a) Analysis Charges	6408041.00	3571262.00			
6. Any Other Receipts(Give Details)			7. Other Payments (Specify)		
I-Remittances (Annexure-A)	20611562.00	17787572.00	Advances (Annexure-D)	55068123.00	32478185.00
CPF-SUB,Arrears and adv.Refund	12641274.70	16864561.35	I-Remittances (Annexure-E)	19987907.00	17726793.00
Sundry Receipts	3150268.00	1152808.30	CPF A/c	8630042.00	11239240.00
Application Fee	384730.00	69202.00	New Pension Scheme	2935894.00	2123569.00
Provident Fund Salwage	0.00	0.00			
Free Gifts - Donations	0.00	0.00	8. Closing Balances		
Sale OF Tender Forms	19500.00	63000.00	a) Cash in hand		
Leave Salary-Pension Contribution	0.00	0.00	b) Bank Balances		
License Fee	53880.00	44400.00	i) In current accounts	26417751.96	12223805.10
Welfare Fund	0.00	0.00	ii) In deposit accounts		
NPS	2935894.00	2123569.00	iii) Savings accounts	4383078.10	20909457.77
Advance/Refunds/Recovery/Adj(Annexure-B)	52653289.00	49650763.00			
TOTAL	830925318.06	845888267.87	TOTAL	830925318.06	845888267.87
DIRECTOR	For K R SRINIVASAN & CO				HEAD - FINANCE & ACCOUNTS
CDFD	CHARTERED ACCOUNTANTS				CDFD
	(K R SRINIVASAN)				

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2014

(Amount - Rs.)

	Current Year		Previous Year
SCHEDULE 1 - CORPUS/CAPITAL FUND :			
Balance as at the beginning of the year		1142536939.00	996575609.00
Add : Contribution towards Corpus/Capital Fund			
CDFD Core - Plan (Non-Recurring)	90000000.00	180000000.00	
Capitalised portion of Capital Expenditure of projects	24938777.00	9729088.00	189729088.00
Less: Lumpsum Depreciation for the year 1996 to 2012	0.00	271409382.00	
Less : Depreciation For the Year 2013-2014 transferred from General Reserve Account	84513447.00	21763702.00	293173084.00
Less : Balance of net income/(Expenditure) transferred	0.00		
	3146980.00	3146980.00	249405326.00
BALANCE AS AT THE YEAR - END		1169815289.00	1142536939.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2014

(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	0.00		276778938.00	
Addition during the year	0.00		0.00	
Less : Deductions during the year	0.00	0.00	27373612.00	0.00
Less : Transfer to Grant in Aid			249405326.00	
			249405326.00	0.00
Total		0.00		0.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2014			(Amount - Rs.)	
SCHEDULE 3 - EARMARKED/ENDOWMENT FUNDS			Current Year	Previous Year
(Refer Annexures)				
(a) Opening balance of the Funds			6531021.20	50698171.20
(b) Additions to the Funds :				
i. Donations /grants			74360025.00	58652921.00
ii. Income from investments made on account of funds			0.00	0.00
iii. Other additions			0.00	0.00
TOTAL (a+b)			80891046.20	109351092.20
(c) Utilisation/Expenditure towards objective of funds				
(i) Capital Expenditure (Refer Annexures I & II)				
- Fixed Assets			24642024.00	9551279.00
- Others			296753.00	177809.00
- Total				9729088.00
(ii) Revenue Expenditure (Refer Annexures I & II)				
- Salaries, Wages and allowances etc.			31884970.00	31815150.00
- Rent			0.00	0.00
- Other Expenses			49841081.00	61275833.00
Total			81726051.00	93090983.00
TOTAL (c)			106664828.00	102820071.00
NET BALANCE AS AT THE YEAR-END [(a + b)-c]			-25773781.80	6531021.20

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2014

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 4 - SECURED LOANS AND BORROWINGS:				
1. Central Government		0.00		0.00
2. State Government (Specify)		0.00		0.00
3. Financial Institutions				
(a) Term Loans	0.00		0.00	
(b) Interest accrued and due	0.00	0.00	0.00	0.00
4. Banks				
(a) Term Loans	0.00		0.00	
- Interest accrued and due	0.00		0.00	
(b) Other Loans (specify)	0.00		0.00	
- Interest accrued and due	0.00	0.00	0.00	0.00
5. Other Institutions and Agencies		0.00		0.00
6. Debentures and Bonds		0.00		0.00
7. Others (Specify)		0.00		0.00
TOTAL		0.00		0.00
Note: Amount due within one year				

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD			(Amount - Rs.)	
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2014				
			Current Year	Previous Year
SCHEDULE 5 - UNSECURED LOANS AND BORROWINGS:				
1. Central Government			0.00	0.00
2. State Government (Specify)			0.00	0.00
3. Financial Institutions			0.00	0.00
4. Banks				
(a) Term Loans		0.00		0.00
(b) Other Loans (specify)		0.00		0.00
5. Other Institutions and Agencies			0.00	0.00
6. Debentures and Bonds			0.00	0.00
7. Fixed Deposits			0.00	0.00
8. Others (Specify)			0.00	0.00
TOTAL			0.00	0.00
Note: Amounts due within one year				

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CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD			(Amount - Rs.)	
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2014				
			Current Year	Previous Year
SCHEDULE6 - DEFERRED CREDIT LIABILITIES:				
(a) Acceptances secured by hypothecation of capital equipment and other assets			0.00	0.00
(b) Others			0.00	0.00
TOTAL			0.00	0.00
Note: Amount due within one year				

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2014

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 7 - CURRENT LIABILITIES AND PROVISIONS		
A. CURRENT LIABILITIES		
1. Acceptances	0.00	0.00
2. Sundry Creditors	0.00	0.00
3. Advances Received	0.00	0.00
4. Interest accrued but not due on:	0.00	0.00
5. Statutory Liabilities:	0.00	0.00
6. Other current Liabilities		
CDFD,CP Fund A/C(Annexure-G)	37788349.00	35805402.00
Collaboration -Workshop Funds	11300000.00	11300000.00
DG Set Maintenance [Advance]	42000.00	0.00
EMD	2357734.00	2898534.00
GSLI	263362.00	44390.00
Honorarium [Advance]	8000.00	0.00
House Building Advance	129831.00	95087.00
Human Resource Development - Training of Staff - Conferences [Advance]	199000.00	0.00
Income Tax	57955.00	37355.00
Lab Security Deposit & Hostel Security Deposit	1170310.00	1155810.00
LIC	2550.00	2550.00
Medical [Advance]	238481.00	0.00
Others (I-Remittances)	269095.00	178985.00
Others [Maintenance Advance]	1000.00	0.00
Out Standing Liabilities	8453405.00	1520556.00
Postage-Courier [Advance]	1264.00	0.00
Professional Tax	96927.00	99187.00
Public Provident Fund	116345.00	0.00
Royalty & Consultancy	2254142.00	2254740.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2014

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 7 - CURRENT LIABILITIES AND PROVISIONS				
Scientific Workshops - Symposiums - Seminars [Advance]	25000.00		0.00	
Security Deposit	1803775.00		1708475.00	
TDS	604383.00		478747.00	
Works Tax	239439.00		234688.00	
Workshop & Conference	0.00	67422347.00	3161.00	57817667.00
TOTAL (A)		67422347.00		57817667.00
B.PROVISIONS				
1. For Taxation				
2. Gratuity				
3. Superannuation/Pension				
4. Accumulated Leave Encashment				
5. Trade Warranties/Claims				
6. Others (Specify)		3392051.00		6932849.00
TOTAL (B)		3392051.00		0.00
TOTAL (A+B)		70814398.00		64750516.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2014

(Amount - Rs.)

SCHEDULE 8 - FIXED ASSETS	GROSS BLOCK			DEPRECIATION			NET BLOCK			
	Cost/valuation as at beginning of the year	Additions during the year	Deductions during the year	Cost/Valuation at the year end	As at the beginning of the year	On Additions during the year	On Deductions during the year	Total up to the year end	As at the Current year end	As at the Previous 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	16503928.00	30532266.00	0.00	47036194.00	173016175.00	203548441.00
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	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
not belongs to the entity										
3. PLANT MACHINERY & EQUIPMENT										
a) VEHICLES	532437808.05	58295873.00	0.00	590733681.05	232197871.00	51684909.00	0.00	283882780.00	306850901.05	300239937.05
b) FURNITURE, FIXTURES	4131158.00	21868.00	0.00	4153026.00	3371338.00	117409.00	0.00	3488747.00	664279.00	759820.00
c) OFFICE EQUIPMENT	16456881.00	1775.00	0.00	16458656.00	9500017.00	697460.00	0.00	10197477.00	6261179.00	6956864.00
d) COMPUTER/PERIPHERALS	11548344.00	19121.00	0.00	11567465.00	8223262.00	505026.00	0.00	8728288.00	2839177.00	3325082.00
e) ELECTRIC INSTALLATIONS	0.00	75843.00	0.00	75843.00	0.00	0.00	0.00	0.00	75843.00	0.00
f) LIBRARY BOOKS	15966802.00	859318.00	0.00	16826120.00	15607294.00	853703.00	0.00	16460997.00	365123.00	359508.00
g) TUBEWELLS & WATER SUPPLY	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
h) OTHER FIXED ASSETS	8857898.00	0.00	0.00	8857898.00	7769374.00	122674.00	0.00	7892048.00	965850.00	1088524.00
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	813351260.05	59273798.00	0.00	872625058.05	293173084.00	84513447.00	0.00	377686531.00	494938527.05	520178176.05
B. CAPITAL WORK-IN-PROGRESS	411955240.70	59875026.00	0.00	471830266.70	0.00	0.00	0.00	0.00	471830266.70	411955240.70
TOTAL	1225306500.75	119148824.00	0.00	1344455324.75	293173084.00	84513447.00	0.00	377686531.00	966768793.75	932133416.75

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD		
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2014		
	Current Year	Previous Year
(Amount - Rs.)		
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)	19398273.00	62398273.00
TOTAL	19398273.00	62398273.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD		
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2014		
	Current Year	Previous Year
(Amount - Rs.)		
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	0.00	0.00
5. Subsidiaries and Joint Ventures	0.00	0.00
6. Others (to be specified) - STDRs, (CPF), CDFD CP FUND A/C	23131298.00	25159583.00
TOTAL	23131298.00	25159583.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2014

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 11 - CURRENT ASSETS, LOANS, ADVANCES ETC.				
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	
b) Others-Life Membership Fees	165935.00	165935.00	165935.00	165935.00
3. Cash balances in hand (including cheques/drafts and imprest)				
4. Bank Balances:				
a) With Scheduled Banks:				
-On Current Accounts	26417751.96		12223805.10	
-On Deposit Accounts (includes margin money)	0.00		0.00	
-On Savings Accounts	4383078.10	30800830.06	20909457.77	33133262.87
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)		30966765.06		33299197.87

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2014

(Amount - Rs.)

SCHEDULE 11 - CURRENT ASSETS, LOANS, ADVANCES, ETC.	Current Year		Previous Year	
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)	151473164.51		136321662.51	
b) Prepayments - Deposits (Annexure-I)	23117612.00		24506343.00	
c) Others	0.00	174590776.51	0.00	160828005.51
3. Income Accrued:				
a) On Investments from Earmarked/Endowments Funds	0.00		0.00	
b) On Investments - Others	0.00		0.00	
c) On Loans and Advances	0.00		0.00	
d) Others	0.00	0.00	0.00	0.00
4. Claims Receivable		25773782.00		0.00
TOTAL (B)		200364558.51		160828005.51
TOTAL (A+B)		231331323.57		194127203.38

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD		
SCHEDULES FORMING PART OF INCOME & EXPENDITURE AS AT 31st MARCH 2014		
(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)	6408041.00	3571262.00
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	6408041.00	3571262.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD		
SCHEDULES FORMING PART OF INCOME & EXPENDITURE AS AT 31st MARCH 2014		
(Amount - Rs.)		
	Current Year	Previous Year
SCHEDULE 13 - GRANTS/SUBSIDIES		
(Irrevocable Grants & Subsidies Received)		
1) Central Government (DBT Plan Grant-in-Aid)	250932400.00	210000000.00
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	250932400.00	210000000.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD		
SCHEDULES FORMING PART OF INCOME & EXPENDITURE AS AT 31st MARCH 2014		
(Amount - Rs.)		
SCHEDULE 14 - FEES/SUBSCRIPTIONS	Current Year	Previous Year
1) Entrance Fees	0.00	0.00
2) Annual Fees/Subscriptions	0.00	0.00
3) Seminar/Program Fees	0.00	0.00
4) Consultancy Fees	0.00	0.00
5) Others (Specify)	0.00	0.00
TOTAL	0.00	0.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD			
SCHEDULES FORMING PART OF INCOME & EXPENDITURE AS AT 31st MARCH 2014			
(Amount - Rs.)			
SCHEDULE 15 - INCOME FROM INVESTMENTS	Investment from Earmarked Fund		Investments - Others
(Income on Invest from Earmarked/Endowment Funds transferred to Funds)	Current Year	Previous Year	Current Year
1) Interest:			
a) On Govt. Securities	0.00		0.00
b) Other Bonds/Debentures	0.00	0.00	0.00
2) Dividends:			
a) On Shares	0.00	0.00	0.00
b) On Mutual Fund Securities	0.00	0.00	0.00
3) Rents	0.00	0.00	0.00
4) Others (Specify) STDRs	23220086.00	2768470.00	0.00
TOTAL	23220086.00	10566572.00	0.00
TRANSFERRED TO EARMARKED/ENDOWMENT FUNDS			

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD		
SCHEDULES FORMING PART OF INCOME & EXPENDITURE AS AT 31st MARCH 2014		
	Current Year	Previous Year
	(Amount - Rs.)	
SCHEDULE 16 - INCOME FROM ROYALTY, PUBLICATIONS ETC.		
1) Income from Royalty	0.00	0.00
2) Income from Publications	0.00	0.00
3) Others (Specify)	0.00	0.00
TOTAL	0.00	0.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD		
SCHEDULES FORMING PART OF INCOME & EXPENDITURE AS AT 31st MARCH 2014		
	Current Year	Previous Year
	(Amount - Rs.)	
SCHEDULE 17 - INTEREST EARNED		
1) On Term Deposits		
a) With Schedule Banks	43238.00	700706.00
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	0.00	0.00
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	0.00	0.00
b) Others	0.00	0.00
4) Interest on Debtors and Other Receivables	0.00	0.00
TOTAL	43238.00	700706.00
Note :- Tax deducted at source to be indicated		

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD		
SCHEDULES FORMING PART OF INCOME & EXPENDITURE AS AT 31st MARCH 2014		
	Current Year	Previous Year
	(Amount - Rs.)	
SCHEDULE 18 - OTHER INCOME		
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	0.00	0.00
5) Other Receipts		
Sundry Receipts	3150268.00	1152808.00
Application Fee	384730.00	69202.00
Sales Of Tender Forms	19500.00	63000.00
Licence Fee	53880.00	44400.00
Interest On Computer Advance, Conveyance Advance And HBA	12488.00	21531.00
Leave Salary-Pension Contribution	0.00	0.00
Provident Fund Salwage	0.00	0.00
Free.Gifts-Donations	0.00	0.00
TOTAL	3620866.00	1350941.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD		
SCHEDULES FORMING PART OF INCOME & EXPENDITURE AS AT 31st MARCH 2014		
	Current Year	Previous Year
	(Amount - Rs.)	
SCHEDULE 19 - INCREASE/(DECREASE) IN STOCK OF FINISHED GOODS & WORK IN PROGRESS		
a) Closing stock		
-Finished Goods	0.00	0.00
-Work-in-progress	0.00	0.00
Total (a)	0.00	0.00
b) Less: Opening Stock		
-Finished Goods	0.00	0.00
-Work-in-progress	0.00	0.00
Total (b)	0.00	0.00
NET INCREASE/(DECREASE) [a-b]	0.00	0.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD		(Amount - Rs.)	
SCHEDULES FORMING PART OF INCOME & EXPENDITURE AS AT 31st MARCH 2014			
SCHEDULE 20 - ESTABLISHMENT EXPENSES		Current Year	Previous Year
a) Salaries and Wages		54269773.00	54234162.00
b) Allowances and Bonus		41267382.00	33816395.00
c) Contribution to Provident Fund		3213621.00	2112193.00
d) Contribution to Other Fund (NPS)		1736649.00	1788473.00
e) Staff Welfare Expenses - Medical charges		2195107.00	2801565.00
f) Expenses on Employees Retirement and Terminal Benefits		4029927.00	91016.00
g) Others (specify) - Staff leased House		0.00	0.00
TOTAL		106712459.00	94843804.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
SCHEDULES FORMING PART OF INCOME & EXPENDITURE AS AT 31st MARCH 2014

(Amount - Rs.)

SCHEDULE 21 - OTHER ADMINISTRATIVE EXPENSES, ETC.	Current Year	Previous Year
a) Purchases	54146570.00	30974267.00
b) Electricity and power	20703811.00	18257125.00
c) Water charges	592058.00	616153.00
d) Insurance	106691.00	80030.00
e) Repairs and maintenance	13737055.00	18347984.00
f) Rent, Rates and Taxes	18691350.00	20625866.00
g) Vehicles Running and Maintenance	949931.00	953329.00
h) Postage, Telephone and Communication Charges	2198082.00	3809722.00
i) Printing and Stationary	1701402.00	1151153.00
j) Travelling and Conveyance Expenses	9099650.00	6819565.00
k) Expenses on Seminar/Workshops	654385.00	1029747.00
l) Subscription Expenses	60872.00	163532.00
m) Expenses on Fees	322746.00	294361.00
n) Auditors Remuneration	71326.00	28090.00
o) Hospitality Expenses	826450.00	891110.00
p) Professional Charges	3722520.00	3329870.00
q) Advertisement and Publicity	2821705.00	4082079.00
r) Bank Charges	14931.00	35206.00
s) Security & Cleaning Contract Charges	18839558.00	16177366.00
t) Training Course /Symposia	211800.00	23752.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD		
SCHEDULES FORMING PART OF INCOME & EXPENDITURE AS AT 31st MARCH 2014		
(Amount - Rs.)		
SCHEDULE 21 - OTHER ADMINISTRATIVE EXPENSES, ETC.	Current Year	Previous Year
u) Other Contingencies	1502085.00	2178819.00
v) Liveries & Blankets	102830.00	1170.00
w) Other Research Expenses	26140605.00	14099459.00
x) Office Books	48688.00	18583.00
TOTAL	177267101.00	143988338.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD		
SCHEDULES FORMING PART OF INCOME & EXPENDITURE AS AT 31st MARCH 2014		
(Amount - Rs.)		
SCHEDULE 22 - EXPENDITURE ON GRANTS, SUBSIDIES, ETC.	Current Year	Previous Year
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, HYDERABAD		
SCHEDULES FORMING PART OF INCOME & EXPENDITURE AS AT 31st MARCH 2014		
(Amount - Rs.)		
SCHEDULE 23 - INTEREST	Current Year	Previous Year
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, and
Schedule 25: Contingent Liabilities & Notes on Account
for the period ended 31/03/2014**

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. Investments:

Investments in STDR's are stated at book values.

7. 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.

8. The previous year balances have been regrouped/rearranged, wherever necessary.

Director, CDFD

Head Finance & Accounts

for K R Srinivasan & Co
Chartered Accountants
[K R SRINIVASAN]

Place: Hyderabad

Date: 18/06/2014

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

CLARIFICATION ON NOTES ON ACCOUNTS: 2013-14

- ◆ 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 informative 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 7: Advances:
The observation of the audit has been noted. The action has already been initiated to reconcile the objection book register.

B JACHARYULU
Head Finance & Accounts
CDFD

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
Details of closing balances of various Earmarked / Endowment Funds (Refer Sch-3)
for the year ended 31st March 2014

Amount in Rs.

Previous year	P No	Particulars	Current Year
-3110519.00	COE-I	COE for Genetics and Genomics of silkmoths	-9645531.00
-8969700.00	COE-II	DBT Centre of Excellence for Microbial Biology	-12818181.00
-630047.00	P-03	"Transgenesis and Genetic basis of Pathogen Resistance in the Silkworm, Bombyx Mori	-630047.00
244305.00	P-09	"NMITLI Project on – Latent M.Tuberculosis: New targets, Drug delivery systems, Bio enhancers & Therapeutics"	244305.00
-9645531	COE1	COE for Genetics and Genomics of silkmoths	-13869143
-12818181	COE2	DBT Centre of Excellence for Microbial Biology	-23581573
-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 reactive oxygen species on T-Cell immune response: An approach to understand the molecular mechanism of immunosuppression during tuberculosis - National Bioscience Award	-576590
4364267	P-101	Role of inositol pyrophosphates in cell physiology: Investigating the biochemical significance of protein pyrophosphorylation - Senior Fellowship	3727878
-430020	P-102	Understanding the role of Mycobacterium tuberculosis heat shockprotein 60 as Th1/Th2 immuno modular	-27922
-600000	P-103	National Bioscience Award - Regulation of mast cell signaling, apoptosis and surface receptors	-300000
-2017875	P-104	Virtual Centre of Excellence on Epigenetics	-3307223
-844946	P-105	Cloning, Characterization and analysis of chromosomal rearrangements in human genetic disorders	-862685
-189211	P-106	Clinical, Biochemical and molecular analysis of treatable lysosomal storage disorders	-227909
435	P-107	IYBA Project - Mechanism and role of bacterial cell-cell signaling molecules in plant defense response	15400
-392965	P-108	Establishment of EBV transformed cell lines from families with rare genetic disorders	-454643
94426	P-109	Molecular dissection of PI3-Kinase/Akt pathway by using proteomics based approach: A study to identify novel potential oncogenes and tumor suppressors	57690
-191391	P-110	India-Japan research project title"Identification and analysis of sex determining genes in silkmoths"	-191391
550416	P-111	Ramalingaswami Fellowship - Refractoriness mechanism in Mosquito: cracking molecular codes at genomic scale	450416
-1036754	P-113	Clinical and molecular genetic analysis of squamous cell carcinoma of the tongue	0
-450859	P-114	Evaluating the Calcineurin-NFAT Pathway and its regulators superoxide dismutase (SOD) AND RCAN1 (regular of Calcineurin) Down Syndrome	-450859
-5	P-115	Setting up of the National Institute of Animal Biotechnology	0
-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
-1132629	P-119	Analysis of DNA copy number alterations in esophageal cancer	-2892
-600218	P-120	Effect of reactive oxygen species on macrophage signalosome: impact on antigen presentation functions and T Cell priming responses	-1474723
-1130866	P-121	Identification and characterization of PTEN regulators	-1130866
13089682	P-122	Understanding the role of Hox genes in anterior-posterior axis determination of the central nervous system	4377125
1151969	P-123	Establish a Max Planck Partner Group for Genetic Diversity Studies at CDFD	513310
-549916	P-124	Preparation and characterization of peroxometal compounds and studies and their biological significance in cellular signalling	-549916
-480981	P-125	Mechanistic studies on the role of protein kinase Snf1k in cell cycle and cancer	172619
-685428	P-126	Rho-dependent transcription termination machinery: mechanism of action	35390
4162538	P-127	Systematic studies on the functional network of phosphatases in cell life and death	283993
537771	P-128	Mechanism of iron acquisition and iron homeostasis in an opportunistic human pathogen Candida glabrata	-608942
6737	P-13	"Programme to delineate gene functions in the post – genomics era by a systematic two gene knockout method"	6737
465973	P-130	Comparative genetic analysis of sex chromosomes and sex determining genes in silkmoths	2865531
-768669	P-131	Structural and functional studies of Acyl CoA Binding proteins from plasmodium falciparum	-1245339
-1228480	P-132	Characterization of tumor suppressor function of ARID1B, a component of the human SWI/SNF chromatin remodelling complex	-2166471
969489	P-133	Investigating the role of Hox gene deformed in central nervous system patterning in Drosophila melanogaster	534614
-141437	P-134	Exploration of wild silk moth biodiversity in Manipur and their genetic characterization using molecular markers	-156437
5376566	P-135	Sys TB: A Network Program for Resolving the Intracellular Dynamics of Host Phthogen Interaction in TB Infection	-298323

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
Details of closing balances of various Earmarked / Endowment Funds (Refer Sch-3)
for the year ended 31st March 2014

Amount in Rs.

Previous year	P No	Particulars	Current Year
77980	P-136	Raf Kinase - a key target for modern-day therapy against tumors	13618
685020	P-137	Signaling pathways involved in down regulation of proinflammatory responses by PPE18 protein of Mycobacterium tuberculosis: Implication of PPE18 as therapeutics	44141
903944	P-138	Co-evaluation of Dnmt3l and Genomic imprinting	-638079
1223583	P-139	Evaluating the role of Sirtuins and epigenetic changes during cellular senescence in context of p53 status	20000
556091	P-140	Development of baculovirus resistant silkworms strains through synthetic miRNA based knockdown of essential viral genes	146091
1463	P-141	Evaluating the functional role of PTEN interacting proteins in cell survival signaling and tumor suppression	-223537
360148	P-142	Identification of H3K4 TRI Demethylase involved in erasing H3K4 trimethylation marks at E2F Responsive promoters	-401878
146284	P-143	Microarray based characterisation of squamous cell carcinoma of the tongue occurring in non smokers	-751303
2208206	P-145	H3K4 HMT family regulates cell cycle progression	-1064782
812209	P-146	Role of MLL in ribosomal RNA transcription	763439
315642	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	41311
20326	P-148	Transcriptional regulation of novel tumor suppressor genes in Pancreatic Cancer	0
1770286	P-149	Role of SUMOylation in the pathobiology of Candida Glabrata	270865
164706	P-150	Genetic and genomic basis of the evolution of bombycid and sturniid silkworms	-28096
1993200	P-151	Human Exome Sequencing to Identify Novel Genes for Medelian Disorders	594981
0	P-152	Global transcriptomics of sex specific splicing	1114145
3000000	P-153	An attractive and promising strategy for early cancer diagnosis through the assembly of the human cancer volatome"	3613562
0	P-154	Rational design, synthetic strategies for developing organometallic anticancer compounds based on organotin and organoiron	87432
335194	P-155	Studies on the cellular roles of calcium signalling proteins in Neurospora crassa	335194
0	P-156	Targeting microbial quorum sensing to demonstrate potential application of cell-cell signaling molecules from Xanthomonas group of plant pathogen in disease control	926632
0	P-157	Identification of novel antifungal drug and delineation of drug resistance mechanisms in an opportunistic human fungal pathogen Candida glabrata	944665
0	P-158	Modulation of host immune responses by a PPE Protein of Mycobacterium tuberculosis: Understanding its role in host - pathogen cross-talk	621787
0	P-159	Gene Targeting of microbial isolates to demonstrate potential plant growth promoting (PGP) traits by third generation sequencing	300000
0	P-160	Understanding the role of novel adhesins of Xanthomonas oryzae PV oryzae in Virulence and colonization in Rice	363884
0	P-161	Analysis of co-regulation between DNA replication activity and amino acid homeostasis by transcription factor IcaA/ArgP in Escherichia coli	350000
0	P-162	Characterization and design of inhibitors of Mycobacterium tuberculosis transcription	235671
0	P-163	Unravelling new functions for the H-NS family of proteins in Gram-negative bacterial pathogens	2006048
0	P-164	A Yeast based screen for discovery of novel sirtuin inhibitors as anticancer agents	-26671
0	P-165	Identification and functional characterization of immune response genes in silkworms	1569682
-687887	P-17	"Studies on inositol-phosphate synthesis - a novel enzyme from Mycobacterium tuberculosis H37RV" - Transfer from IMTECH, Chandigarh	-687887
-274286	P-18	"Mapping of receptor binding site on the Erythrocyte binding of malaria parasite"	-274286
-1888111	P-20	"Genomic Micro array R&D Programmes on infectious diseases and Neurological Disorders"	-1888111
0.5	P-22	"Biotechnology for leather - towards cleaner processing"	0.5
-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 protein 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
2045696	P-30	Transcription termination and anti termination in E-coli	2045696
746453	P-31	Role of K-ras in Lung type II epithelial cells	746453
-234000	P-33	"Molecular and Epidemiological characterisation of cryptosporidium - An enteric protozoan parasite"	-234000
26334	P-34	"Molecular analysis of lepidopteran - specific immune proteins from silkworms"	26334
-283883	P-35	"Identification, Characterization and Physical mapping of Z-Chromosome linked genes of the silk worm, Bombyx mori"	-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
-2237285	P-42	"Structural and functional studies on Mycobacterium tuberculosis heat shock proteins".	-2237285
685906.7	P-43	"A generalized mechanism of transcription termination in prokaryotes: a quest for mechanism based transcription inhibitors for microbial pathogens".	685906.7
-457538	P-44	"Understanding of role of Ras and NO / iNOS signalling in promotion of hepatocellular carcinomas with persistent HBV infection"	-457538
605714	P-45	Specialized chromatin structures as epigenetic imprints to distinguish parental alleles".	605714
-1586965	P-47	Research cum Training for DRDO Programme	-1586965

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Details of closing balances of various Earmarked / Endowment Funds (Refer Sch-3)
for the year ended 31st March 2014

Amount in Rs.

Previous year	P No	Particulars	Current Year
151826	P-48	'Molecular characterization of human liver stem cells for use in the treatment of hepatic diseases'.	151826
308361	P-49A	International Atomic Energy Agency (IAEA)	804660
-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 nucleoid protein H-NS"	-280000
-278928	P-62	"HIV – 1 Pathogenesis: Role of Integrase in Reverse Transcription and Nuclear Transport of Viral Genome"	-278928
-837574	P-63	"Upgradation of the existing computing infrastructure at the Bioinformatics facility at CDFD"	-837574
-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 Helicobacter pylori"	-582647
19734821	P-65A	APEDA-CDFD Centre for Basmati DNA Analysis	20617169
-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
-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 - ± 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
562620	P-81A	Financial assistance for award of J C Bose Fellowship to Dr J Gowrishankar	463453
-367721	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 silkworms	-65698
218818	P-88	Introduction of anti-baculoviral property in commercial silkworm strains by expression of multiple RNAi viral targets	218818
-636286	P-90	Role of Yapsins in the Pathobiology of Candida Glabrata	-636286
-1098900	P-91	DMMT3L: epigenetic correlation with cancer	-1098900
-3090255	P-92	Swarnajayanti fellowship proj on "Designing transcription anti-terminators: a novel approach for making new inhibitors of gene expression"	268823
-661454	P-93/A1	Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against tuberculosis	-605745
-2446997	P-93/A2	Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against Mycobacterium tuberculosis	-2469833
-146870	P-97	Proteome-wide Analysis of Serine pyrophosphorylation by inositol pyrophosphates	-276552
-255844	P-98	Role of cell - cell signaling mediated by Diffusible signaling factor (DSF) in Xanthomonas virulence	-203419
-315780	P-99	Role of inositol Pyrophosphates in eukaryotic cell growth, proliferation and ribosome biogenesis	-567516
6531021.2			-25773781.8

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
Details of Fixed Assets Fund (Capitalised portion of Project Grants)
for the year ended 31st March 2014

Amount in Rs.

Previous year	P No	Particulars	Current Year
11713327	COE-I	COE for Genetics and Genomics of silkmoths	11713327
10000000	COE-II	DBT Centre of Excellence for Microbial Biology	10000000
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
11713327	COE-I	COE for Genetics and Genomics of silkmoths	11713327
10000000	COE-II	DBT Centre of Excellence for Microbial Biology	10000000
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 reactive oxygen species on T-Cell immune response: An approach to understand the molecular mechanism of immunosuppression during tuberculosis - National Bioscience Award	17784
10645294	P-101	Role of inositol pyrophosphates in cell physiology: Investigating the biochemical significance of protein pyrophosphorylation - Senior Fellowship	12024311
681121	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
915278	P-109	Molecular dissection of PI3-Kinase/Akt pathway by using proteomics based approach: A study to identify novel potential oncogenes and tumor suppressors	915278
0	P-111	Ramalingaswami Fellowship - Refractoriness mechanism in Mosquito: cracking molecular codes at genomic scale	206800
0	P-112	Ramanujan Fellowship	0
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 (regulator 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
459324	P-122	Understanding the role of Hox genes in anterior-posterior axis determination of the central nervous system	9889367
453095	P-123	Establish a Max Planck Partner Group for Genetic Diversity Studies at CDFD	540436
385404	P-126	Rho-dependent transcription termination machinery: mechanism of action	402016
2897196	P-127	Systematic studies on the functional network of phosphatases in cell life and death	6281319
1594393	P-128	Mechanism of iron acquisition and iron homeostasis in an opportunistic human pathogen Candida glabrata	1609427
1334600	P-13	"Programme to delineate gene functions in the post – genomics era by a systematic two gene knockout method"	1334600
0	P-130	Comparative genetic analysis of sex chromosomes and sex determining genes in silkmoths	81500
474792	P-133	Investigating the role of Hox gene deformed in central nervous system patterning in Drosophila melanogaster	964215
0	P-135	Sys TB: A Network Program for Resolving the Intracellular Dynamics of Host Phthogen Interaction in TB Infection	5500000
0	P-137	Signaling pathways involved in down regulation of proinflammatory responses by PPE18 protein of Mycobacterium tuberculosis: Implication of PPE18 as therapeutics	130979
0	P-138	Co-evaluation of Dnmt3l and Genomic imprinting	565518
0	P-139	Evaluating the role of Sirtuins and epigenetic changes during cellular senescence in context of p53 status	500000
5163243	P-14	"Comparative and functional genomics approaches for the identification and characterization of genes responsible for multi drug resistance of mycobacterium tuberculosis"	5163243
424914	P-142	Identification of H3K4 TRI Demethylase involved in erasing H3K4 trimethylation marks at E2F Responsive promoters	624495
0	P-145	H3K4 HMT family regulates cell cycle progression	1546279
359711	P-146	Role of MLL in ribosomal RNA transcription	686219
0	P-149	Role of SUMOylation in the pathobiology of Candida Glabrata	468720
6000000	P-15	"The Helicobacter Pylori genome programme – Genome sequencing, functional analysis and comparative genomics of the strains obtained from Indian patients"	6000000
0	P-157	Identification of novel antifungal drug and delineation of drug resistance mechanisms in an opportunistic human fungal pathogen Candida glabrata	380852
1814901	P-16	NMITLI Project on – Latent M.Tuberculosis: New targets, Drug delivery systems, Bio enhancers & Therapeutics	1814901
244400	P-17	"Studies on inositol-phosphate synthesis – a novel enzyme from Mycobacterium tuberculosis H37RV" – Transfer from IMTECH, Chandigarh	244400
344020	P-18	"Mapping of receptor binding site on the Erythrocyte binding of malaria parasite"	344020
7246511	P-19	"Construction of Integrated RAPD, RFLP and Microsatellite linkage map of the Silkworm, Bombyx mori and its correlation 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

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
Details of Fixed Assets Fund (Capitalised portion of Project Grants)
for the year ended 31st March 2014

Amount in Rs.

Previous year	P No	Particulars	Current Year
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 Transcription 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
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 - ± 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
195728	P-81A	Financial assistance for award of J C Bose Fellowship to Dr J Gowrishankar	195728
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
655403	P-93/A2	Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against Mycobacterium tuberculosis	840648
246320	P-95	Construction of regulatory networks in prokaryotes through protein: Protein interaction predictions and transcription regulation predictions. (MOU with Russian Foundation)	246320
966602	P-97	Proteome-wide Analysis of Serine pyrophosphorylation by inositol pyrophosphates	1000000
2789420	P-98	Role of cell - cell signaling mediated by Diffusible signaling factor (DSF) in Xanthomonas virulence	2783795
2963482	P-99	Role of inositol Pyrophosphates in eukaryotic cell growth, proliferation and ribosomae biogenesis	2963482
264585874			3E+08

**CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2014**

Annexure: A Forming part of Receipts & Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	I-Remittances	
4976630.00	TDS	4872379.00
6114373.00	Income Tax	6930770.00
4586.00	Works Tax	4751.00
1335912.00	LIC	1501203.00
219721.00	GSLI	450115.00
1904410.00	Public Provident Fund	2201735.00
574296.00	Professional Tax	568281.00
1979139.00	Service Tax	2739240.00
678505.00	Others (I-Remittances)	1142963.00
0.00	Health Insurance	200125.00
17787572.00		20611562.00

**CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2014**

Annexure: B Forming part of Receipts & Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	Advance refunds/recovery/Adjst.	
239083.00	Advance for purchases by Staff	432548.00
0.00	AMC for Equipment [Advance]	34547.00
355710.00	CDFD Staff reserve Fund	0.00
0.00	Chemicals [Advance]	25209032.00
70004.00	Computer Advance [Research Fellows]	83449.00
46200.00	Computer Advance [Staff]	35900.00
0.00	Consumables, glassware and Spares [Advance]	33400.00
31080.00	Conveyance Advance	47140.00
0.00	DG Set Maintenance [Advance]	42000.00
1800.00	EMD	339200.00
42903643.00	Equipment [Advance]	13093454.00
110625.00	Festival Advance	103125.00
1585218.00	General Deposits And Advances	1999431.00
0.00	Honorarium [Advance]	23000.00
0.00	House Building Advance	34744.00
0.00	Human Resource Development - Training of Staff - Conferences [Advance]	199000.00
220200.00	Lab Security Deposit & Hostel Security Deposit	141500.00
239061.00	LTC [Advance]	864513.00
0.00	Medical [Advance]	538481.00
0.00	Other Research Expenses [Advance]	28090.00
39075.00	Others [Advances]	405179.00
0.00	Others [Maintenance Advance]	1000.00

**CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2014**

Annexure: B Forming part of Receipts & Payment a/c

Previous Year Amount	Rs.	Particulars	Current Year Amount	Rs.
	0.00	Postage-Courier [Advance]		1264.00
	307394.00	Revolving Advance		319669.00
	1200000.00	Royalty & Consultancy		0.00
	0.00	Scientific Workshops - Symposiums - Seminars [Advance]		25000.00
	71000.00	Security Deposit		95300.00
	0.00	TA Abroad [Advance]		157412.00
	1690485.00	TA With in India [Advance]		4352911.00
	4000.00	Trainee Security Deposit		13000.00
	536185.00	Workshop & Conference		4000000.00
49650763.00			52653289.00	

**CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2014**

Annexure: C Forming part of Receipts & Payment a/c

Previous Year Amount	Rs.	Particulars	Current Year Amount	Rs.
		Projects - Receipts		
	4000000.00	COE1/CORE		3814000.00
	0.00	COE1/P-I		750000.00
	0.00	COE1/P-II		643000.00
	0.00	COE1/P-III		1009000.00
	4478000.00	COE2/CORE		0.00
	924000.00	COE2/P-2		0.00
	777000.00	COE2/P-A		0.00
	791000.00	COE2/P-B		0.00
	911000.00	COE2/P-C		0.00
	0.00	COE-I/P-IV		450000.00
	0.00	P-101		6230314.00
	503782.00	P-102		457596.00
	0.00	P-103		300000.00
	1437000.00	P-104		0.00
	505153.00	P-106		0.00
	817000.00	P-107		0.00
	566000.00	P-109		0.00
	1487000.00	P-111		1490000.00
	0.00	P-113		1419047.00
	760000.00	P-114		0.00
	143232.00	P-115		0.00
	1252800.00	P-119		1328000.00
	4880510.00	P-122		4986110.00
	1047000.00	P-123		1203108.00
	0.00	P-125		1374000.00
	0.00	P-126		1780400.00
	4637410.00	P-127		6910824.00
	1017200.00	P-128		0.00

**CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2014**

Annexure: C Forming part of Receipts & Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
0.00	P-130	4300000.00
0.00	P-131	1768900.00
763000.00	P-133	981000.00
0.00	P-134	425000.00
0.00	P-135	2057700.00
0.00	P-136	759000.00
0.00	P-137	473256.00
1799600.00	P-138	0.00
500000.00	P-139	520000.00
1850000.00	P-140	394000.00
500000.00	P-141	300000.00
1514000.00	P-142	211000.00
714000.00	P-143	0.00
3885200.00	P-145	0.00
1850000.00	P-146	872000.00
805900.00	P-147	500000.00
700000.00	P-148	0.00
1979600.00	P-149	1059500.00
210000.00	P-150	0.00
1993200.00	P-151	0.00
0.00	P-152	2872300.00
3000000.00	P-153	937000.00
0.00	P-154	1030000.00
335194.00	P-155	0.00
0.00	P-156	2104400.00
0.00	P-157	2760800.00
0.00	P-158	1933141.00
0.00	P-159	300000.00
0.00	P-160	382000.00
0.00	P-161	350000.00
0.00	P-162	799600.00
0.00	P-163	2006048.00
0.00	P-165	1569682.00
222000.00	P-40	0.00
0.00	P-49A	496299.00
1068000.00	P-65A	1062000.00
1360000.00	P-81A	1360000.00
126140.00	P-83A	0.00
680000.00	P-88	0.00
0.00	P-92	4000000.00
645000.00	P-93/A1	645000.00
0.00	P-93/A2	985000.00
1217000.00	P-99	0.00
58652921.00		74360025.00

**CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2014**

Annexure: D Forming part of Receipts & Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	Advances	
229702.00	Advance for purchases by Staff	340050.00
38250.00	AMC for Equipment [Advance]	0.00
9660410.00	Chemicals [Advance]	6938893.00
100000.00	Computer Advance [Research Fellows]	32400.00
210000.00	Computer Advance [Staff]	90000.00
714700.00	Consumables, glassware and Spares [Advance]	10610261.00
148200.00	Conveyance Advance	180768.00
241800.00	EMD	880000.00
12673898.00	Equipment [Advance]	23577545.00
86250.00	Festival Advance	124875.00
135277.00	GDA [Others]	600700.00
1038800.00	General Deposits And Advances	10000.00
5000.00	Honorarium [Advance]	10000.00
93000.00	Lab Security Deposit & Hostel Security Deposit	127000.00
0.00	Liveries & Blankets [Advance]	31000.00
1229250.00	LTC [Advance]	1417120.00
300000.00	Medical [Advance]	0.00
22700.00	Office Equipment [Advance]	0.00
28090.00	Other Research Expenses [Advance]	9166.00
113479.00	Others [Advances]	1023456.00
326500.00	Revolving Advance	370500.00
493382.00	Royalty & Consultancy	598.00
2500.00	Security Deposit	0.00
0.00	TA Abroad [Advance]	614715.00
2876325.00	TA With in India [Advance]	3847364.00
12500.00	Trainee Security Deposit	13500.00
1698172.00	Workshop & Conference	4218212.00
32478185.00		55068123.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS FOR THE YEAR ENDED 31st MARCH 2014		
Annexure: E Forming part of Receipts & Payment a/c		
Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	I-Remittances	
202410.00	GSLI	231143.00
0.00	Health Insurance	334610.00
6154006.00	Income Tax	6910170.00
1335912.00	LIC	1501203.00
499520.00	Others (I-Remittances)	1052853.00
478411.00	Professional Tax	570541.00
2012875.00	Public Provident Fund	1999815.00
2155621.00	Service Tax	2640829.00
4883964.00	TDS	4746743.00
4074.00	Works Tax	0.00
17726793.00		19987907.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS FOR THE YEAR ENDED 31st MARCH 2014		
Annexure: F Forming part of Receipts & Payment a/c		
Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	Projects - Expenditure	
10535012.00	COE1	10889612
11729481.00	COE2	10763392
241590.00	P-100	0.00
9365134.00	P-101	6866703.00
1016456.00	P-102	55498.00
300000.00	P-103	0.00
2060009.00	P-104	1289348.00
754102.00	P-105	17739.00
885316.00	P-106	38698.00
880165.00	P-107	-14965.00
462890.00	P-108	61678.00
787200.00	P-109	36736.00
22712.00	P-110	0.00
1368315.00	P-111	1590000.00
1571384.00	P-113	382293.00
1262412.00	P-114	0.00
8182978.00	P-115	-5.00
962946.00	P-116	0.00
1646824.00	P-119	198263.00
724818.00	P-120	874505.00
533680.00	P-121	0.00
3269871.00	P-122	13698667.00
1969087.00	P-123	1841767.00
717200.00	P-124	0.00
634981.00	P-125	720400.00

**CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2014**

Annexure: F Forming part of Receipts & Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
2267043.00	P-126	1059582.00
5527587.00	P-127	10789369.00
2533016.00	P-128	1146713.00
306000.00	P-129	0.00
3721027.00	P-130	1900442.00
1951604.00	P-131	2245570.00
1862803.00	P-132	937991.00
1342511.00	P-133	1415875.00
395437.00	P-134	440000.00
2041634.00	P-135	7732589.00
759220.00	P-136	823362.00
814980.00	P-137	1114135.00
895656.00	P-138	1542023.00
1743617.00	P-139	1723583.00
1293909.00	P-140	804000.00
498537.00	P-141	525000.00
1153852.00	P-142	973026.00
567716.00	P-143	897587.00
267184.00	P-144	0.00
1676994.00	P-145	3272988.00
1037791.00	P-146	920770.00
490258.00	P-147	774331.00
679674.00	P-148	20326.00
209314.00	P-149	2558921.00
45294.00	P-150	192802.00
0.00	P-151	1398219.00
0.00	P-152	1758155.00
0.00	P-153	323438.00
0.00	P-154	942568.00
0.00	P-156	1177768.00
0.00	P-157	1816135.00
0.00	P-158	1311354.00
0.00	P-160	18116.00
0.00	P-162	563929.00
0.00	P-164	26671.00
132589.00	P-49A	0.00
271200.00	P-65A	179652.00
15829.00	P-71	0.00
860000.00	P-81A	1459167.00
523580.00	P-82	1300.00
461182.00	P-88	0.00
1829794.00	P-92	640922.00
630644.00	P-93/A1	589291.00
1615921.00	P-93/A2	1007836.00
48406.00	P-97	129682.00
192825.00	P-98	-52425.00
270880.00	P-99	251736.00
102820071.00		106664828.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS		
FOR THE YEAR ENDED 31st MARCH 2014		
Annexure: G Forming part of Receipts & Payment a/c		
Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	CDFD C.P.F ACCOUNT	
29159376.00	Opening Balance	35805401.67
	Add:	
5355840.00	Employee subscription/ refunds	4801908.00
0.00	Transfer from other departments	0.00
2112193.00	Institute contribution (inc. Projects staff)	0.00
3277120.00	Interest received	980581.70
4099128.00	Less: Advances/withdrawals/Transfer/Adjst	3799542.00
35805401.00		37788349.37

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS		
FOR THE YEAR ENDED 31st MARCH 2014		
Annexure: H Forming part of Receipts & Payment a/c		
Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	LOANS AND ADVANCES	
238838.00	Advance for purchases by Staff	146340.50
4310.00	Advances [Previous Years]	4310.00
38250.00	AMC for Equipment [Advance]	3703.00
79326670.00	Chemicals [Advance]	61056531.00
67909.00	Computer Advance [Research Fellows]	16860.00
88500.00	Computer Advance [Staff]	142600.00
714700.00	Consumables, glassware and Spares [Advance]	11291561.00
44620.00	Conveyance Advance	178248.00
37848368.45	Equipment [Advance]	48332459.45
53550.00	Festival Advance	75300.00
0.00	Health Insurance	134485.00
5000.00	Honorarium [Advance]	0.00
0.00	Liveries & Blankets [Advance]	31000.00
1972353.00	LTC [Advance]	2524960.00
300000.00	Medical [Advance]	0.00
22700.00	Office Equipment [Advance]	22700.00
28090.00	Other Research Expenses [Advance]	9166.00
5333288.00	Others [Advances]	5951565.00
85575.00	Public Provident Fund	0.00
304569.00	Rent [Advance]	304569.00
8468959.00	Research Fellows-Associates	19751667.00
102343.00	Revolving Advance	153174.00
176554.00	Service Tax	78143.00
0.00	TA Abroad [Advance]	457303.00
1069516.56	TA With in India [Advance]	563968.56
27000.00	Trainee Security Deposit	27500.00
0.00	Workshop & Conference	215051.00
136321663.01		151473164.51

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS FOR THE YEAR ENDED 31st MARCH 2014		
Annexure: I Forming part of Balance Sheet		
Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	DEPOSITS	
24371066.00	General Deposits And Advances	22381635.00
135277.00	GDA [Others]	735977.00
24506343.00		23117612.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS FOR THE YEAR ENDED 31st MARCH 2014		
Annexure: J Forming part of Balance sheet		
Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	INVESTMENT A/C	
51098273.00	Investments	8098273.00
11300000.00	Other Investments	11300000.00
62398273.00		19398273.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS FOR THE YEAR ENDED 31st MARCH 2014		
Annexure: K Forming part of Balance sheet		
Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	CDFD C.P.F INVESTMENT A/C	
23202519.00	Deposit with Banks	25159583.00
7140112.00	Employee subscription	4830500.00
5183048.00	Less Transfer To Bank A/C	6858785.00
25159583.00		23131298.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-03: "Transgenesis and Genetic basis of Pathogen Resistance in the Silkworm, Bombyx Mori									
Pi:									
Receipts and Payments Account from 01/04/2013 to 31/03/2014									
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	
0.00			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	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-09: "NMITLI Project on – Latent M.Tuberculosis: New targets, Drug delivery systems, Bio enhancers & Therapeutics"									
Pi: Dr Seyed E Hasnain									
Receipts and Payments Account from 01/04/2013 to 31/03/2014									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
244305.00		Opening Balance	244305.00		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	
244305.00			244305.00		0.00			0.00	
0.00		Excess of Expenditure over Income	0.00		244305.00		Closing Balance	244305.00	
244305.00			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 01/04/2013 to 31/03/2014					
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	0.00	Transfer of Funds	0.00
0.00		0.00	28332.00		28332.00
28332.00	Excess of Expenditure over Income	28332.00	0.00	Closing Balance	0.00
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 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
6737.00	Opening Balance	6737.00	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
6737.00		6737.00	0.00		0.00
0.00	Excess of Expenditure over Income	0.00	6737.00	Closing Balance	6737.00
6737.00		6737.00	6737.00		6737.00

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-17: "Studies on inositol-phosphate synthesis – a novel enzyme from Mycobacterium tuberculosis H37RV" – Transfer from IMTECH, Chandigarh P.I: Dr Sekhar C Mande Receipts and Payments Account from 01/04/2013 to 31/03/2014</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
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

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-18: "Mapping of receptor binding site on the Eythrocyte binding of malaria parasite" P.I: Dr Akash Ranjan Receipts and Payments Account from 01/04/2013 to 31/03/2014</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
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 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	1888111.00	
0.00		Grant In Aid	0.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			0.00			1888111.00	
1888111.00		Excess of Expenditure over Income	1888111.00		Closing Balance	0.00	
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 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	34495.00	
0.00		Grant In Aid	0.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			0.00			34495.00	
34495.00		Excess of Expenditure over Income	34495.00		Closing Balance	0.00	
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 01/04/2013 to 31/03/2014									
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: Dr Mahalingam & Dr Mande									
Receipts and Payments Account from 01/04/2013 to 31/03/2014									
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 PI: Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		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" PI: Dr K Prashanth Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		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-30: Transcription termination and anti termination in E-coli P.I: Dr Ranjan Sen Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
2045696.00	Opening Balance	2045696.00	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
2045696.00		2045696.00	0.00		0.00
0.00	Excess of Expenditure over Income	0.00	2045696.00	Closing Balance	2045696.00
2045696.00		2045696.00	2045696.00		2045696.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-31: Role of K-ras in Lung type II epithelial cells P.I: Dr Gayatri Ramakrishna Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
746453.00	Opening Balance	746453.00	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
746453.00		746453.00	0.00		0.00
0.00	Excess of Expenditure over Income	0.00	746453.00	Closing Balance	746453.00
746453.00		746453.00	746453.00		746453.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-33: "Molecular and Epidemiological characterisation of cryptosporidium – An enteric protozoan parasite"									
P.I: Dr Radha Rama Devi									
Receipts and Payments Account from 01/04/2013 to 31/03/2014									
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 proteins from silkworms"									
P.I: Dr J Nagaraju									
Receipts and Payments Account from 01/04/2013 to 31/03/2014									
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		Salaries - Manpower	0.00	
0.00		Grant In Aid	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	

<p align="center">P-35: "Identification, Characterization and Physical mapping of Z-Chromosome linked genes of the silk worm, Bombyx mori" Centre for DNA Fingerprinting and Diagnostics, Hyderabad P.I: Dr J Nagaraju Receipts and Payments Account from 01/04/2013 to 31/03/2014</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		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

<p align="center">P-36: "Development of Artificial retina using Bacterio rhodospin and genetically engineered analogues " Centre for DNA Fingerprinting and Diagnostics, Hyderabad P.I: Dr Sekhar C Mande Receipts and Payments Account from 01/04/2013 to 31/03/2014</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
2073896.00	Opening Balance	2073896.00	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
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 01/04/2013 to 31/03/2014									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		226058.00		Opening Balance	4058.00	
222000.00		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	
222000.00			0.00		226058.00			4058.00	
4058.00		Excess of Expenditure over Income	4058.00		0.00		Closing Balance	0.00	
226058.00			4058.00		226058.00			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 01/04/2013 to 31/03/2014									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
1873605.00		Opening Balance	1873605.00		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	
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-42: "Structural and functional studies on Mycobacterium tuberculosis heat shock proteins".					
P.I: Dr Sekhar C Mande					
Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
0.00	Opening Balance	0.00	2237285.00	Opening Balance	2237285.00
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	2237285.00		2237285.00
2237285.00	Excess of Expenditure over Income	2237285.00	0.00	Closing Balance	0.00
2237285.00		2237285.00	2237285.00		2237285.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-43: "A generalized mechanism of transcription termination in prokaryotes: a quest for mechanism based transcription inhibitors for microbial pathogens".					
P.I: Dr Ranjan Sen					
Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
685906.70	Opening Balance	685906.70	0.00	Salaries - Manpower	0.00
0.00	Grant In Aid	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
685906.70		685906.70	0.00		0.00
0.00	Excess of Expenditure over Income	0.00	685906.70	Closing Balance	685906.70
685906.70		685906.70	685906.70		685906.70

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 01/04/2013 to 31/03/2014									
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-45: Specialized chromatin structures as epigenetic imprints to distinguish parental alleles". P.I: Dr Sanjeev Khosla Receipts and Payments Account from 01/04/2013 to 31/03/2014									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
605714.00		Opening Balance	605714.00		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	
605714.00			605714.00		0.00			0.00	
0.00		Excess of Expenditure over Income	0.00		605714.00		Closing Balance	605714.00	
605714.00			605714.00		605714.00			605714.00	

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 01/04/2013 to 31/03/2014									
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 Khosia Receipts and Payments Account from 01/04/2013 to 31/03/2014									
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		Salaries - Manpower	0.00	
0.00		Grant In Aid	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 01/04/2013 to 31/03/2014									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
440950.00	0.00	Opening Balance	308361.00	0.00	0.00	0.00	Salaries - Manpower	0.00	0.00
		Grant In Aid	496299.00	0.00	0.00	0.00	Consumables	0.00	0.00
			0.00	0.00	0.00	0.00	Contingencies	0.00	0.00
			0.00	0.00	132589.00	0.00	Travel	0.00	0.00
			0.00	0.00	0.00	0.00	Overheads	0.00	0.00
			0.00	0.00	0.00	0.00	Equipment	0.00	0.00
			0.00	0.00	0.00	0.00	Books	0.00	0.00
			0.00	0.00	0.00	0.00	AMC	0.00	0.00
			0.00	0.00	0.00	0.00	Others	0.00	0.00
			0.00	0.00	0.00	0.00	Transfer of Funds	0.00	0.00
440950.00	0.00		804660.00	0.00	132589.00	0.00		0.00	0.00
		Excess of Expenditure over Income	0.00	0.00	308361.00	0.00	Closing Balance	804660.00	804660.00
440950.00			804660.00		440950.00			804660.00	

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 01/04/2013 to 31/03/2014									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00	0.00	Opening Balance	0.00	0.00	284065.00	0.00	Opening Balance	284065.00	0.00
0.00	0.00	Grant In Aid	0.00	0.00	0.00	0.00	Salaries - Manpower	0.00	0.00
0.00	0.00		0.00	0.00	0.00	0.00	Consumables	0.00	0.00
0.00	0.00		0.00	0.00	0.00	0.00	Contingencies	0.00	0.00
0.00	0.00		0.00	0.00	0.00	0.00	Travel	0.00	0.00
0.00	0.00		0.00	0.00	0.00	0.00	Overheads	0.00	0.00
0.00	0.00		0.00	0.00	0.00	0.00	Equipment	0.00	0.00
0.00	0.00		0.00	0.00	0.00	0.00	Books	0.00	0.00
0.00	0.00		0.00	0.00	0.00	0.00	AMC	0.00	0.00
0.00	0.00		0.00	0.00	0.00	0.00	Others	0.00	0.00
0.00	0.00		0.00	0.00	0.00	0.00	Transfer of Funds	0.00	0.00
0.00	0.00		0.00	0.00	284065.00	0.00		284065.00	0.00
284065.00		Excess of Expenditure over Income	284065.00	0.00	0.00	0.00	Closing Balance	284065.00	284065.00
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 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	1231118.00	
0.00		Grant In Aid	0.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			0.00			1231118.00	
1231118.00		Excess of Expenditure over Income	1231118.00		Closing Balance	0.00	
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 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	37877.00	
0.00		Grant In Aid	0.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			0.00			37877.00	
37877.00		Excess of Expenditure over Income	37877.00		Closing Balance	0.00	
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 01/04/2013 to 31/03/2014									
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		Salaries - Manpower	0.00	
0.00		Grant In Aid	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 01/04/2013 to 31/03/2014									
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 01/04/2013 to 31/03/2014									
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 01/04/2013 to 31/03/2014									
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		Salaries - Manpower	0.00	
0.00		Grant In Aid	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 nucleoid protein H-NS"							
P.I: Dr Abhijit A Sardesai							
Receipts and Payments Account from 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	280000.00	
0.00		Grant In Aid	0.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			0.00			280000.00	
280000.00		Excess of Expenditure over Income	280000.00		Closing Balance	0.00	
280000.00			280000.00			280000.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-62: "HIV – 1 Pathogenesis: Role of Integrase in Reverse Transcription and Nuclear Transport of Viral Genome"							
P.I: Dr S Mahalingam							
Receipts and Payments Account from 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	278928.00	
0.00		Grant In Aid	0.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			0.00			278928.00	
278928.00		Excess of Expenditure over Income	278928.00		Closing Balance	0.00	
278928.00			278928.00			278928.00	

<p align="center">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 01/04/2013 to 31/03/2014</p>									
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	837574.00	
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		837574.00			837574.00	
837574.00		Excess of Expenditure over Income	837574.00		0.00		Closing Balance	0.00	
837574.00			837574.00		837574.00			837574.00	

<p align="center">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 01/04/2013 to 31/03/2014</p>									
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	

P-65: "Molecular, genetic and functional analysis of the chromosomal plasticity region of the gastric pathogen Helicobacter pylori" PI: Dr Ayesha Alvi Receipts and Payments Account from 01/04/2013 to 31/03/2014									
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	

P-65A: APEDA-CDFD Centre for Basmati DNA Analysis PI: Dr J Nagaraju Receipts and Payments Account from 01/04/2013 to 31/03/2014									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
18938021.00		Opening Balance	19734821.00		0.00		Opening Balance	0.00.00	
0.00		Grant in aid	0.00		271200.00		Salaries- Manpower	179652.00	
1068000.00		Basmati Analysis Charges	1062000.00		0.00		Consumables	0.00	
0.00					0.00		Contingencies	0.00	
					0.00		Travel	0.00	
					0.00		Overheads	0.00	
					0.00		Consultancy & Knowledge fee	0.00	
					0.00		Vehicle	0.00	
					0.00		Equipment	0.00	
20006021.00		Excess of expenditure over income	20796821.00		271200.00			179652.00	
20006021.00			20796821.00		19734821.00		Closing Balance	20617169.00	
					20006021.00			20796821.00	

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 Khosia Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs.		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

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 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs.		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 01/04/2013 to 31/03/2014									
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 01/04/2013 to 31/03/2014									
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 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		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 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		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 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		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 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		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 01/04/2013 to 31/03/2014					
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	Salaries - Manpower	0.00
0.00	Grant In Aid	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 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
1304.00	Opening Balance	1304.00	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
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 01/04/2013 to 31/03/2014									
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 01/04/2013 to 31/03/2014									
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		Consumables	0.00	
					0.00		Contingencies	0.00	
					0.00		Travel	0.00	
					0.00		Overheads	0.00	
					0.00		Equipment	0.00	
					0.00		Books	0.00	
					0.00		AMC	0.00	
					0.00		Others	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 01/04/2013 to 31/03/2014					
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	Salaries - Manpower	0.00
0.00	Grant In Aid	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 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
62620.00	Opening Balance	562620.00	300000.00	Opening Balance	0.00
1360000.00	Grant In Aid	1360000.00	342185.00	Salaries - Manpower	300000.00
0.00		0.00	0.00	Consumables	1000000.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	157815.00	Travel	99167.00
0.00		0.00	60000.00	Overheads	60000.00
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
1422620.00		1922620.00	860000.00		1459167.00
0.00	Excess of Expenditure Over Income	0.00	562620.00	Closing Balance	463453.00
1422620.00		1922620.00	1422620.00		1922620.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-82: Functional genomic analysis of Candida Glabrata-macrophage PI: Dr Rupinder Kaur Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
155859.00	Opening Balance	0.00		Opening Balance	367721.00
0.00	Grant In Aid	0.00	284787.00	Salaries - Manpower	1300.00
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	38793.00	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
155859.00		0.00	523580.00		369021.00
367721.00	Excess of Expenditure Over Income	369021.00	0.00	Closing Balance	0.00
523580.00		369021.00	523580.00		369021.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-83: Prokaryotic Transcription termination factor, Rho: Mechanism of Action and Biology PI: Dr Ranjan Sen Receipts and Payments Account from 01/04/2013 to 31/03/2014					
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

<p align="center">P-83A: Understanding the mechanism of Azadirachtin-mediated cell signaling: role in anti-inflammation and anti-tumorigenesis Centre for DNA Fingerprinting and Diagnostics, Hyderabad P.I: Dr Sunil Kumar Manna Receipts and Payments Account from 01/04/2013 to 31/03/2014</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
0.00	Opening Balance	0.00	126140.00	Opening Balance	0.00
126140.00	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	126140.00		0.00
126140.00	Excess of Expenditure over Income	0.00	0.00	Closing Balance	0.00
126140.00		0.00	126140.00		0.00

<p align="center">P-84: Preparing for vaccine efficacy trials: Baseline epidemiology, improved diagnosis, markers of protection and phase I/II trials Centre for DNA Fingerprinting and Diagnostics, Hyderabad P.I: Dr Niyaz Ahmed Receipts and Payments Account from 01/04/2013 to 31/03/2014</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
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 PI: Dr Madhusudan Reddy Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		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 PI: Dr Akash Ranjan Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		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	1118755.00		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 silkworms P.I: Dr J Nagaraju Receipts and Payments Account from 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	65698.00	
0.00		Grant In Aid	0.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			0.00			65698.00	
65698.00		Excess of Expenditure over Income	65698.00		Closing Balance	0.00	
65698.00			65698.00			65698.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-88: Introduction of anti-baculoviral property in commercial silkworm strains by expression of multiple RNAi viral targets P.I: Dr J Nagaraju Receipts and Payments Account from 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	218818.00		Opening Balance	0.00	
680000.00		Grant In Aid	0.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
680000.00			218818.00			0.00	
0.00		Excess of Expenditure Over Income	0.00		Closing Balance	218818.00	
680000.00			218818.00			218818.00	

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 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year. Amount	Payments	Current Year Amount
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: DM1T3L: epigenetic correlation with cancer P.I: Dr Sanjeev Khosla Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year. Amount	Payments	Current Year Amount
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

P-92: Swarnajayanti fellowship proj on "Designing transcription anti-terminators: a novel approach for making new inhibitors of gene expression" Centre for DNA Fingerprinting and Diagnostics, Hyderabad P.I.: Dr Ranjan Sen Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	1260461.00	Opening Balance	3090255.00
0.00	Grant In Aid	4000000.00	696542.00	Salaries - Manpower	410922.00
0.00		0.00	1000000.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	30000.00
0.00		0.00	133252.00	Travel	0.00
0.00		0.00	0.00	Overheads	200000.00
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		4000000.00	3090255.00		3731177.00
3090255.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	268823.00
3090255.00		4000000.00	3090255.00		4000000.00

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P - 93: DBT Project on "Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against tuberculosis" Centre for DNA Fingerprinting and Diagnostics, Hyderabad P.I.: Dr Shekar C Mande & Dr Sangita Mukhopadhyay Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	1506886.00	Opening Balance	3108451.00
645000.00	Grant in aid	1630000.00	1341030.00	Salaries- Manpower	1407023.00
			546166.00	Consumables	0.00
			0.00	Contingencies	0.00
			0.00	Travel	4859.00
			0.00	Overheads	0.00
			359369.00	Equipment	185245.00
645000.00		1630000.00	3753451.00		4705578.00
3108451.00	Excess of expenditure over income	3075578.00	0.00	Closing Balance	0.00
3753451.00		4705578.00	3753451.00		4705578.00

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 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	98464.00	Opening Balance	146870.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	96284.00
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	48406.00	Equipment	33398.00
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	146870.00		276552.00
146870.00	Excess of Expenditure Over Income	276552.00	0.00	Closing Balance	0.00
146870.00		276552.00	146870.00		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 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	63019.00	Opening Balance	255844.00
0.00	Grant In Aid	0.00	187200.00	Salaries - Manpower	-46800.00
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	5625.00	Equipment	-5625.00
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	255844.00		203419.00
255844.00	Excess of Expenditure Over Income	203419.00	0.00	Closing Balance	0.00
255844.00		203419.00	255844.00		203419.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-99: Role of inositol Pyrophosphates in eukaryotic cell growth, proliferation and ribosome biogenesis					
P.I: Dr Rashna Bhandari					
Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	1261900.00	Opening Balance	315780.00
1217000.00	Grant In Aid	0.00	206491.00	Salaries - Manpower	251736.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	20000.00	Contingencies	0.00
0.00		0.00	2636.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	41753.00	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	1532780.00		567516.00
1217000.00		0.00			
315780.00	Excess of Expenditure Over Income	567516.00	0.00	Closing Balance	0.00
1532780.00		567516.00	1532780.00		567516.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-100: Effect of reactive 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 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	335000.00	Opening Balance	576590.00
0.00	Grant In Aid	0.00	77479.00	Salaries - Manpower	0.00
0.00		0.00	164111.00	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
0.00		0.00	0.00		0.00
576590.00	Excess of Expenditure Over Income	576590.00	576590.00	Closing Balance	0.00
576590.00		576590.00	576590.00		576590.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-101: Role of inositol pyrophosphates in cell physiology: Investigating the biochemical significance of protein pyrophosphorylation - Senior Fellowship					
P.I: Dr Rashna Bhandari					
Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
13729401.00	Opening Balance	4364267.00	1818930.00	Opening Balance	0.00
0.00	Grant In Aid	6230314.00	2300000.00	Salaries - Manpower	1440715.00
0.00		0.00	849721.00	Consumables	3409058.00
0.00		0.00	27452.00	Contingencies	0.00
0.00		0.00	0.00	Travel	22013.00
0.00		0.00	0.00	Overheads	615900.00
0.00		0.00	4191222.00	Equipment	1379017.00
0.00		0.00	177809.00	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
13729401.00		10594581.00	9365134.00		6866703.00
0.00	Excess of Expenditure Over Income	0.00	4364267.00	Closing Balance	3727878.00
13729401.00		10594581.00	13729401.00		10594581.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-102: ICMR Project on "Understanding the role of Mycobacterium tuberculosis heat shockprotein 60 as Th1/Th2 immuno modular"					
P.I: Dr Sangita Mukhopadhyay					
Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
0.00		0.00	0.00	Opening Balance	430020.00
82654.00	Opening Balance	0.00	386400.00	Salaries- Manpower	0.00
503782.00	Grant in aid	457596.00	600000.00	Consumables	0.00
0.00		0.00	29605.00	Contingencies	19043.00
0.00		0.00	451.00	Travel	0.00
0.00		0.00	0.00	Overheads	19026.00
0.00		0.00	0.00	Equipment	17429.00
586436.00		457596.00	1016456.00		485518.00
430020.00	Excess of expenditure over income	27922.00	0.00	Closing Balance	
1016456.00		485518.00	1016456.00		485518.00

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 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	300000.00	Opening Balance	600000.00
0.00	Grant In Aid	300000.00	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	600000.00		600000.00
600000.00	Excess of Expenditure over Income	300000.00	0.00	Closing Balance	0.00
600000.00		600000.00	600000.00		600000.00

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 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	1394866.00	Opening Balance	2017875.00
1437000.00	Grant In Aid	0.00	452710.00	Salaries - Manpower	354407.00
0.00		0.00	1594206.00	Consumables	884941.00
0.00		0.00	0.00	Contingencies	500000.00
0.00		0.00	13093.00	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
1437000.00		0.00	3454875.00		3307223.00
2017875.00	Excess of Expenditure Over Income	3307223.00	0.00	Closing Balance	0.00
3454875.00		3307223.00	3454875.00		3307223.00

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 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	90844.00	Opening Balance	844946.00
0.00	Grant In Aid	0.00	126202.00	Salaries - Manpower	0.00
0.00		0.00	600000.00	Consumables	0.00
0.00		0.00	20000.00	Contingencies	0.00
0.00		0.00	7900.00	Travel	17739.00
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	84946.00		862685.00
844946.00	Excess of Expenditure Over Income	862685.00	0.00	Closing Balance	0.00
844946.00		862685.00	84946.00		862685.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-106: Clinical, Biochemical and molecular analysis of treatable lysosomal storage disorders					
P.I: Dr Ashwin B Dalal					
Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
190952.00	Opening Balance	0.00	409530.00	Opening Balance	189211.00
505153.00	Grant In Aid	0.00	450000.00	Salaries - Manpower	38698.00
0.00		0.00	25786.00	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
696105.00		0.00	885316.00		227909.00
189211.00	Excess of Expenditure Over Income	227909.00	0.00	Closing Balance	0.00
885316.00		227909.00	885316.00		227909.00

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 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
636000.00	Opening Balance	435.00	187200.00	Salaries- Manpower	78000.00
817000.00	Grant in aid	0.00	674153.00	Consumables	-74153.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	18812.00	Travel	-18812.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
880600.00		435.00	880165.00		-14965.00
0.00		0.00	435.00	Closing Balance	15400.00
880600.00		435.00	880600.00		435.00

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 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
69925.00	Opening Balance	0.00		Opening Balance	392965.00
0.00	Grant In Aid	0.00	174737.00	Salaries - Manpower	42774.00
0.00		0.00	250000.00	Consumables	0.00
0.00		0.00	25000.00	Contingencies	0.00
0.00		0.00	13153.00	Travel	18904.00
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
69925.00		0.00	462890.00		454643.00
392965.00	Excess of Expenditure Over Income	454643.00	0.00	Closing Balance	0.00
462890.00		454643.00	462890.00		454643.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-109: Molecular dissection of PI3-Kinase/Akt pathway by using proteomics based approach: A study to identify novel potential oncogenes and tumor suppressors					
P.I: Dr M Subba Reddy					
Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
315626.00	Opening Balance	94426.00		Opening Balance	0.00
566000.00	Grant In Aid	0.00	187200.00	Salaries - Manpower	36736.00
0.00		0.00	580160.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	19840.00	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
881626.00		94426.00	787200.00		36736.00
0.00	Excess of Expenditure Over Income	0.00	94426.00	Closing Balance	57690.00
881626.00		94426.00	881626.00		94426.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-110: India-Japan research project title "Identification and analysis of sex determining genes in silkworms"					
P.I: Dr J Nagaraju					
Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	168679.00	Opening Balance	191391.00
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	22712.00	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

P-111: Ramalingaswami Fellowship - Refractoriness mechanism in Mosquito: cracking molecular codes at genomic scale P.I: Dr Shweta Tyagi Receipts and Payments Account from 01/04/2013 to 31/03/2014									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
431731.00		Opening Balance	550416.00		1106000.00		Opening Balance		0.00
1487000.00		Grant In Aid	1490000.00		262315.00		Salaries - Manpower	1182684.00	
0.00			0.00		0.00		Consumables	200516.00	
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	206800.00	
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	
1918731.00			2040416.00		1368315.00			1590000.00	
0.00		Excess of Expenditure Over Income	0.00		550416.00		Closing Balance	450416.00	
1918731.00			2040416.00		1918731.00			2040416.00	

P-113: Clinical and molecular genetic analysis of squamous cell carcinoma of the tongue P.I: Dr M D Bashyam Receipts and Payments Account from 01/04/2013 to 31/03/2014									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
534630.00		Opening Balance	0.00		544619.00		Opening Balance	1036754.00	
0.00		Grant In Aid	1419047.00		590000.00		Salaries - Manpower	372663.00	
0.00			0.00		34084.00		Consumables	0.00	
0.00			0.00		1500.00		Contingencies	4000.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		401181.00		Overheads	5630.00	
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	
534630.00			1419047.00		1571384.00			1419047.00	
1036754.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance	0.00	
1571384.00			1419047.00		1571384.00			1419047.00	

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 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
51553.00		Opening Balance	0.00		Opening Balance	450859.00	
760000.00		Grant In Aid	0.00	112840.00	Salaries - Manpower	0.00	0.00
0.00			0.00	960000.00	Consumables	0.00	0.00
0.00			0.00	0.00	Contingencies	0.00	0.00
0.00			0.00	7680.00	Travel	0.00	0.00
0.00			0.00	0.00	Overheads	0.00	0.00
0.00			0.00	181892.00	Equipment	0.00	0.00
0.00			0.00	0.00	Books	0.00	0.00
0.00			0.00	0.00	AMC	0.00	0.00
0.00			0.00	0.00	Others	0.00	0.00
0.00			0.00	0.00	Transfer of Funds	0.00	0.00
811553.00			0.00	1262412.00		450859.00	
450859.00		Excess of Expenditure Over Income	450859.00	0.00	Closing Balance	0.00	0.00
1262412.00			450859.00	1262412.00		450859.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-115: Setting up of the National Institute of Animal Biotechnology P.I: Dr J Gowrishankar Receipts and Payments Account from 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00			0.00	0.00	Opening Balance	5.00	
8039741.00		Opening Balance	0.00	0.00	Salaries- Manpower	0.00	0.00
143232.00		Grant in aid	0.00	0.00	Consumables	0.00	0.00
0.00			0.00	0.00	Contingencies	-5.00	
0.00			0.00	0.00	Travel	0.00	0.00
0.00			0.00	0.00	Equipment	0.00	0.00
0.00			0.00	8182978.00	Transfer of Funds	0.00	0.00
8182973.00			0.00	8182978.00		0.00	
0.00			0.00	0.00	Closing Balance	0.00	0.00
8182973.00			0.00	5000000.00		0.00	

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 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	1251366.00	
0.00		Grant In Aid	0.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			0.00			1251366.00	
1251366.00		Excess of Expenditure Over Income	1251366.00		Closing Balance	0.00	
1251366.00			1251366.00			1251366.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-119: Analysis of DNA copy number alterations in esophageal cancer P.I: Dr M D Bashyam Receipts and Payments Account from 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	1132629.00	
1252800.00		Grant In Aid	1328000.00		Salaries - Manpower	198263.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
1252800.00			1328000.00			1330892.00	
1132629.00		Excess of Expenditure Over Income	2892.00		Closing Balance	0.00	
2385429.00			1330892.00			1330892.00	

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 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
124600.00		Opening Balance	0.00		Opening Balance	600218.00	
0.00		Grant In Aid	0.00		Salaries - Manpower	205400.00	
0.00			0.00		Consumables	619105.00	
0.00			0.00		Contingencies	50000.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
124600.00			0.00			1474723.00	
600218.00		Excess of Expenditure Over Income	1474723.00		Closing Balance	0.00	
724818.00			1474723.00			1474723.00	

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 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	1130866.00	
0.00		Grant In Aid	0.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			0.00			1130866.00	
1130866.00		Excess of Expenditure Over Income	1130866.00		Closing Balance	0.00	
1130866.00			1130866.00			1130866.00	

<p align="center">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 01/04/2013 to 31/03/2014</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
11479043.00	Opening Balance	13089682.00	1340206.00	Opening Balance	0.00
4880510.00	Grant In Aid	4986110.00	1400000.00	Salaries - Manpower	1207355.00
0.00		0.00	342745.00	Consumables	1839406.00
0.00		0.00	165680.00	Contingencies	0.00
0.00		0.00	0.00	Travel	22013.00
0.00		0.00	21240.00	Overheads	1199850.00
0.00		0.00	0.00	Equipment	9430043.00
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
16359553.00		1807592.00	3269871.00		13698667.00
0.00	Excess of Expenditure Over Income	0.00	13089682.00	Closing Balance	4377125.00
16359553.00		1807592.00	16359553.00		1807592.00

<p align="center">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 01/04/2013 to 31/03/2014</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
2074056.00	Opening Balance	1151969.00	564208.00	Opening Balance	0.00
1047000.00	Grant In Aid	1203108.00	670000.00	Salaries - Manpower	438409.00
0.00		0.00	200000.00	Consumables	1016274.00
0.00		0.00	183584.00	Contingencies	100000.00
0.00		0.00	0.00	Travel	199743.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	351295.00	Equipment	87341.00
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
3121056.00		2355077.00	1969087.00		1841767.00
0.00	Excess of Expenditure Over Income	0.00	1151969.00	Closing Balance	513310.00
3121056.00		2355077.00	3121056.00		2355077.00

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 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
167284.00	Opening Balance	0.00	187200.00	Opening Balance	549916.00
0.00	Grant In Aid	0.00	500000.00	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
167284.00		0.00	717200.00		549916.00
549916.00	Excess of Expenditure over Income	549916.00	0.00	Closing Balance	0.00
717200.00		549916.00	717200.00		549916.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-125: Mechanistic studies on the role of protein kinase Snf1k in cell cycle and cancer					
P.I: Dr M Subba Reddy					
Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
154000.00	Opening Balance	0.00	134981.00	Opening Balance	480981.00
0.00	Grant In Aid	1374000.00	500000.00	Salaries - Manpower	220400.00
0.00		0.00	0.00	Consumables	500000.00
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
154000.00		1374000.00	634981.00		1201381.00
480981.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	172619.00
634981.00		1374000.00	634981.00		1374000.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-126: Rho-dependent transcription termination machinery: mechanism of action					
PI: Dr Ranjan Sen					
Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
1581615.00	Opening Balance	0.00	488532.00	Opening Balance	685428.00
0.00	Grant In Aid	1780400.00	1381445.00	Salaries - Manpower	298393.00
0.00		0.00	3081.00	Consumables	704577.00
0.00		0.00	8581.00	Contingencies	40000.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	385404.00	Overheads	0.00
0.00		0.00	0.00	Equipment	16612.00
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
1581615.00		1780400.00	2267043.00		1745010.00
685428.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	35390.00
2267043.00		1780400.00	2267043.00		1780400.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-127: Systematic studies on the functional network of phosphatases in cell life and death					
PI: Dr M Subba Reddy					
Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
5052715.00	Opening Balance	4162538.00	538921.00	Opening Balance	0.00
4637410.00	Grant In Aid	6910824.00	3560000.00	Salaries - Manpower	620441.00
0.00		0.00	506524.00	Consumables	5827231.00
0.00		0.00	250853.00	Contingencies	0.00
0.00		0.00	0.00	Travel	3700.00
0.00		0.00	671289.00	Overheads	953874.00
0.00		0.00	0.00	Equipment	3087370.00
0.00		0.00	0.00	Books	296753.00
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
9690125.00		11073362.00	5527587.00		10789369.00
0.00	Excess of Expenditure Over Income	0.00	4162538.00	Closing Balance	283993.00
9690125.00		11073362.00	9690125.00		11073362.00

<p align="center">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 01/04/2013 to 31/03/2014</p>							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
2053587.00		Opening Balance	537771.00		Opening Balance		0.00
1017200.00		Grant In Aid	0.00		Salaries - Manpower		185732.00
0.00			0.00		Consumables		900000.00
0.00			0.00		Contingencies		30000.00
0.00			0.00		Travel		15947.00
0.00			0.00		Overheads		0.00
0.00			0.00		Equipment		15034.00
0.00			0.00		Books		0.00
0.00			0.00		AMC		0.00
0.00			0.00		Others		0.00
0.00			0.00		Transfer of Funds		0.00
3070787.00			537771.00				1146713.00
0.00		Excess of Expenditure Over Income	608942.00		Closing Balance		0.00
3070787.00			1146713.00				1146713.00

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-130: Comparative genetic analysis of sex chromosomes and sex determining genes in silkworms P.I: Dr J Nagaraju Receipts and Payments Account from 01/04/2013 to 31/03/2014</p>							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
4187000.00		Opening Balance	465973.00		Opening Balance		0.00
0.00		Grant In Aid	4300000.00		Salaries - Manpower		699263.00
0.00			0.00		Consumables		1000000.00
0.00			0.00		Contingencies		100000.00
0.00			0.00		Travel		19679.00
0.00			0.00		Overheads		0.00
0.00			0.00		Equipment		81500.00
0.00			0.00		Books		0.00
0.00			0.00		AMC		0.00
0.00			0.00		Others		0.00
0.00			0.00		Transfer of Funds		0.00
4187000.00			4765973.00				1900442.00
0.00		Excess of Expenditure Over Income	0.00		Closing Balance		2865531.00
4187000.00			4765973.00				4765973.00

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 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
1182935.00	Opening Balance	0.00	414514.00	Opening Balance	768669.00
0.00	Grant In Aid	1768900.00	1488971.00	Salaries - Manpower	311665.00
0.00		0.00	29000.00	Consumables	1861029.00
0.00		0.00	19119.00	Contingencies	50000.00
0.00		0.00	0.00	Travel	22876.00
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
1182935.00		1768900.00	1951604.00		3014239.00
768669.00	Excess of Expenditure Over Income	1245339.00	0.00	Closing Balance	0.00
1951604.00		3014239.00	1951604.00		3014239.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-132: Characterization of tumor suppressor function of ARID1B, a component of the human SWI/SNF chromatin remodelling complex					
P.I: Dr M D Bashyam, Dr Rohit Joshi					
Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
634323.00	Opening Balance	0.00	425016.00	Opening Balance	1228480.00
0.00	Grant In Aid	0.00	1400000.00	Salaries - Manpower	400113.00
0.00		0.00	30000.00	Consumables	484566.00
0.00		0.00	7787.00	Contingencies	20000.00
0.00		0.00	0.00	Travel	33312.00
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
634323.00		0.00	1862803.00		2166471.00
1228480.00	Excess of Expenditure Over Income	2166471.00	0.00	Closing Balance	0.00
1862803.00		2166471.00	1862803.00		2166471.00

<p align="center">P-133: Investigating the role of Hox gene deformed in central nervous system patterning in Drosophila melanogaster Centre for DNA Fingerprinting and Diagnostics, Hyderabad PI: Dr Rohit Joshi Receipts and Payments Account from 01/04/2013 to 31/03/2014</p>					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
1549000.00	Opening Balance	969489.00	239394.00	Opening Balance	0.00
763000.00	Grant In Aid	981000.00	600000.00	Salaries - Manpower	326452.00
0.00		0.00	0.00	Consumables	600000.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	28325.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	474792.00	Equipment	489423.00
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
2312000.00		1950489.00	1342511.00		1415875.00
0.00	Excess of Expenditure Over Income	0.00	969489.00	Closing Balance	534614.00
2312000.00		1950489.00	2312000.00		1950489.00

<p align="center">P-134: Exploration of wild silk moth biodiversity in Manipur and their genetic characterization using molecular markers Centre for DNA Fingerprinting and Diagnostics, Hyderabad PI: Dr K P Arun Kumar Receipts and Payments Account from 01/04/2013 to 31/03/2014</p>					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
254000.00	Opening Balance	0.00	0.00	Opening Balance	141437.00
0.00	Grant In Aid	425000.00	350000.00	Salaries - Manpower	0.00
0.00		0.00	15000.00	Consumables	400000.00
0.00		0.00	30437.00	Contingencies	25000.00
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
254000.00		425000.00	395437.00		581437.00
141437.00	Excess of Expenditure Over Income	156437.00	0.00	Closing Balance	0.00
395437.00		581437.00	395437.00		581437.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-135: Sys TB: A Network Program for Resolving the Intracellular Dynamics of Host Pathogen Interaction in TB Infection					
P.I: Dr. Sanjeev Kholisa					
Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
7418200.00	Opening Balance	5376566.00	0.00	Opening Balance	0.00
0.00	Grant In Aid	2057700.00	2000000.00	Salaries - Manpower	274929.00
0.00		0.00	25000.00	Consumables	1885265.00
0.00		0.00	16634.00	Contingencies	50000.00
0.00		0.00	0.00	Travel	22395.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	5500000.00
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
7418200.00		7434266.00	2041634.00		7732589.00
0.00	Excess of Expenditure Over Income	298323.00	5376566.00	Closing Balance	0.00
7418200.00		7732589.00	7418200.00		7732589.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-136: Raf Kinase - a key target for modern-day therapy against tumors					
P.I: Dr Sunil Kumar Manna					
Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
837200.00	Opening Balance	77980.00	162240.00	Opening Balance	0.00
0.00	Grant In Aid	759000.00	566980.00	Salaries - Manpower	187200.00
0.00		0.00	30000.00	Consumables	606162.00
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	30000.00
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
837200.00		836980.00	759220.00		823362.00
0.00	Excess of Expenditure Over Income	0.00	77980.00	Closing Balance	13618.00
837200.00		836980.00	837200.00		836980.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-137: Signaling pathways involved in down regulation of proinflammatory responses by PPE18 protein of Mycobacterium tuberculosis: Implication of PPE18 as therapeutics							
Pi: Dr Sangita Mukhopadhyay							
Receipts and Payments Account from 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
1500000.00		Opening Balance	685020.00		Opening Balance		0.00
0.00		Grant In Aid	473256.00		Salaries - Manpower		79733.00
0.00			0.00		Consumables		750000.00
0.00			0.00		Contingencies		53423.00
0.00			0.00		Travel		0.00
0.00			0.00		Overheads		100000.00
0.00			0.00		Equipment		130979.00
0.00			0.00		Books		0.00
0.00			0.00		AMC		0.00
0.00			0.00		Others		0.00
0.00			0.00		Transfer of Funds		0.00
1500000.00			1158276.00				1114135.00
0.00		Excess of Expenditure Over Income	0.00		Closing Balance		44141.00
1500000.00			1158276.00				1158276.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-138: Co-evaluation of Dnmt3l and Genomic imprinting							
Pi: Dr Sanjeev Khosla							
Receipts and Payments Account from 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	903944.00		Opening Balance		0.00
1799600.00		Grant In Aid	0.00		Salaries - Manpower		151505.00
0.00			0.00		Consumables		800000.00
0.00			0.00		Contingencies		250000.00
0.00			0.00		Travel		0.00
0.00			0.00		Overheads		0.00
0.00			0.00		Equipment		565518.00
0.00			0.00		Books		0.00
0.00			0.00		AMC		0.00
0.00			0.00		Others		0.00
0.00			0.00		Transfer of Funds		0.00
1799600.00			903944.00				1542023.00
0.00		Excess of Expenditure Over Income	638079.00		Closing Balance		0.00
1799600.00			1542023.00				1542023.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-139: Evaluating the role of Sirtuins and epigenetic changes during cellular senescence in context of p53 status PI: Dr Gayatri Ramakrishna, Dr Sanjeev Khosla Receipts and Payments Account from 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
2467200.00	0.00	Opening Balance	1223583.00	0.00	Opening Balance	0.00	0.00
5000000.00	0.00	Grant In Aid	520000.00	80617.00	Salaries - Manpower	109200.00	109200.00
0.00	0.00		0.00	1623000.00	Consumables	520173.00	520173.00
0.00	0.00		0.00	40000.00	Contingencies	20000.00	20000.00
0.00	0.00		0.00	0.00	Travel	0.00	0.00
0.00	0.00		0.00	0.00	Overheads	0.00	0.00
0.00	0.00		0.00	0.00	Equipment	500000.00	500000.00
0.00	0.00		0.00	0.00	Books	0.00	0.00
0.00	0.00		0.00	0.00	AMC	0.00	0.00
0.00	0.00		0.00	0.00	Others	0.00	0.00
0.00	0.00		0.00	0.00	Transfer of Funds	574210.00	574210.00
2967200.00	0.00	Excess of Expenditure Over Income	1743583.00	1743617.00		1723583.00	1723583.00
2967200.00			0.00	1223583.00	Closing Balance	20000.00	20000.00
			1743583.00	2967200.00		1743583.00	1743583.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-140: Development of baculovirus resistant silkworms strains through synthetic miRNA based knockdown of essential viral genes PI: Dr K P Arun Kumar Receipts and Payments Account from 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00	0.00	Opening Balance	556091.00	0.00	Opening Balance	0.00	0.00
1850000.00	0.00	Grant In Aid	394000.00	193909.00	Salaries - Manpower	204000.00	204000.00
0.00	0.00		0.00	1084288.00	Consumables	600000.00	600000.00
0.00	0.00		0.00	0.00	Contingencies	0.00	0.00
0.00	0.00		0.00	15712.00	Travel	0.00	0.00
0.00	0.00		0.00	0.00	Overheads	0.00	0.00
0.00	0.00		0.00	0.00	Equipment	0.00	0.00
0.00	0.00		0.00	0.00	Books	0.00	0.00
0.00	0.00		0.00	0.00	AMC	0.00	0.00
0.00	0.00		0.00	0.00	Others	0.00	0.00
0.00	0.00		0.00	0.00	Transfer of Funds	0.00	0.00
1850000.00	0.00	Excess of Expenditure Over Income	950091.00	1293909.00		804000.00	804000.00
1850000.00			0.00	556091.00	Closing Balance	146091.00	146091.00
			950091.00	1850000.00		950091.00	950091.00

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 01/04/2013 to 31/03/2014									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	1463.00		0.00		Opening Balance	0.00	
500000.00		Grant In Aid	300000.00		418537.00		Salaries - Manpower	0.00	
0.00			0.00		80000.00		Consumables	425000.00	
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	100000.00	
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			301463.00		498537.00			525000.00	
0.00		Excess of Expenditure Over Income	223537.00		1463.00		Closing Balance	0.00	
500000.00			525000.00		500000.00			525000.00	

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 01/04/2013 to 31/03/2014									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	360148.00		128938.00		Opening Balance	0.00	
1514000.00		Grant In Aid	211000.00		600000.00		Salaries - Manpower	173445.00	
0.00			0.00		0.00		Consumables	600000.00	
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		424914.00		Equipment	199581.00	
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	
1514000.00			571148.00		1153852.00			973026.00	
0.00		Excess of Expenditure Over Income	401878.00		360148.00		Closing Balance	0.00	
1514000.00			973026.00		1514000.00			973026.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-143: Microarray based characterisation of squamous cell carcinoma of the tongue occurring in non smokers P.I: Dr M D Bashyam Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	146284.00	163716.00	Opening Balance	0.00
714000.00	Grant In Aid	0.00	404000.00	Salaries - Manpower	247587.00
0.00		0.00	0.00	Consumables	650000.00
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
714000.00		146284.00	567716.00		897587.00
0.00	Excess of Expenditure Over Income	751303.00	146284.00	Closing Balance	0.00
714000.00		897587.00	714000.00		897587.00

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 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	2208206.00	76994.00	Opening Balance	0.00
3885200.00	Grant In Aid	0.00	1600000.00	Salaries - Manpower	187200.00
0.00		0.00	0.00	Consumables	1500000.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	39509.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	1546279.00
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
3885200.00		2208206.00	1676994.00		3272988.00
0.00	Excess of Expenditure Over Income	1064782.00	2208206.00	Closing Balance	0.00
3885200.00		3272988.00	3885200.00		3272988.00

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 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	812209.00		Opening Balance	0.00
1850000.00	Grant In Aid	872000.00	78080.00	Salaries - Manpower	244262.00
0.00		0.00	600000.00	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	359711.00	Equipment	326508.00
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
1850000.00		1684209.00	1037791.00		920770.00
0.00	Excess of Expenditure Over Income	0.00	812209.00	Closing Balance	763439.00
1850000.00		1684209.00	1850000.00		1684209.00

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 01/04/2013 to 31/03/2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	315642.00		Opening Balance	0.00
805900.00	Grant In Aid	500000.00	40258.00	Salaries - Manpower	187200.00
0.00		0.00	400000.00	Consumables	400000.00
0.00		0.00	50000.00	Contingencies	50000.00
0.00		0.00	0.00	Travel	50831.00
0.00		0.00	0.00	Overheads	86300.00
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
805900.00		815642.00	490258.00		774331.00
0.00	Excess of Expenditure Over Income	0.00	315642.00	Closing Balance	41311.00
805900.00		815642.00	805900.00		815642.00

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-148: Transcriptional regulation of novel tumor suppressor genes in Pancreatic Cancer P.I: Dr K Jayaprakash Narayana Receipts and Payments Account from 01/04/2013 to 31/03/2014</p>									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Previous Year Amount	Rs	Current Year Amount	Rs
0.00		Opening Balance	20326.00		Opening Balance	170484.00		0.00	
700000.00		Grant In Aid	0.00		Salaries - Manpower	0.00		20326.00	
0.00			0.00		Consumables	19081.00		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	490109.00		0.00	
700000.00			20326.00			679674.00		20326.00	
0.00		Excess of Expenditure Over Income	0.00		Closing Balance	20326.00		0.00	
700000.00			20326.00			700000.00		20326.00	

<p align="center">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 01/04/2013 to 31/03/2014</p>									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Previous Year Amount	Rs	Current Year Amount	Rs
0.00		Opening Balance	1770286.00		Opening Balance	59314.00		0.00	
1979600.00		Grant In Aid	1059500.00		Salaries - Manpower	100000.00		187200.00	
0.00			0.00		Consumables	50000.00		1700000.00	
0.00			0.00		Contingencies	0.00		150000.00	
0.00			0.00		Travel	0.00		53001.00	
0.00			0.00		Overheads	0.00		0.00	
0.00			0.00		Equipment	0.00		468720.00	
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	
1979600.00			2829786.00			209314.00		2558921.00	
0.00		Excess of Expenditure Over Income	0.00		Closing Balance	1770286.00		270865.00	
1979600.00			2829786.00			1979600.00		2829786.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-150: Genetic and genomic basis of the evolution of bombycid and sturniid silkworms									
PI: Dr J Nagaraju									
Receipts and Payments Account from 01/04/2013 to 31/03/2014									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	164706.00		0.00		Opening Balance		0.00
210000.00		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		45294.00		Travel	192802.00	
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
210000.00			164706.00		45294.00				192802.00
0.00		Excess of Expenditure Over Income	28096.00		164706.00		Closing Balance		0.00
210000.00			192802.00		210000.00				192802.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-151: Human Exome Sequencing to Identify Novel Genes for Medelian Disorders									
PI: Dr Ashwin B Dalal									
Receipts and Payments Account from 01/04/2013 to 31/03/2014									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	1993200.00		0.00		Opening Balance		0.00
1993200.00		Grant In Aid	0.00		0.00		Salaries - Manpower	74729.00	
0.00			0.00		0.00		Consumables	1200000.00	
0.00			0.00		0.00		Contingencies	100000.00	
0.00			0.00		0.00		Travel	23490.00	
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
1993200.00			1993200.00		0.00				1398219.00
0.00		Excess of Expenditure Over Income	0.00		1993200.00		Closing Balance		594981.00
1993200.00			1993200.00		1993200.00				1993200.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-152 : Global transcriptomics of sex specific splicing P.I: Dr K P Arun Kumar Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year. Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00		Opening Balance	0.00
0.00	Grant In Aid	2872300.00	0.00	Salaries - Manpower	284155.00
0.00		0.00	0.00	Consumables	1474000.00
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		2872300.00	0.00		1758155.00
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	1114145.00
0.00		2872300.00	0.00		2872300.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-153: An attractive and promising strategy for early cancer diagnosis through the assembly of the human cancer volatome” P.I: Dr H A Nagarajaram Receipts and Payments Account from 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year. Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	3000000.00	0.00	Opening Balance	0.00
3000000.00	Grant In Aid	937000.00	0.00	Salaries - Manpower	58877.00
0.00		0.00	0.00	Consumables	70000.00
0.00		0.00	0.00	Contingencies	80000.00
0.00		0.00	0.00	Travel	114561.00
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
3000000.00		3937000.00	0.00		323438.00
0.00	Excess of Expenditure Over Income	0.00	3000000.00	Closing Balance	3613562.00
3000000.00		3937000.00	3000000.00		3937000.00

<p align="center">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 01/04/2013 to 31/03/2014</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	0.00	Opening Balance	0.00
0.00	Grant In Aid	1030000.00	0.00	Salaries - Manpower	192568.00
0.00		0.00	0.00	Consumables	700000.00
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	50000.00
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		1030000.00	0.00		942568.00
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	87432.00
0.00		1030000.00	0.00		1030000.00

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-155: Studies on the cellular roles of calcium signalling proteins in Neurospora crassa P.I: Dr D P Kasbekar Receipts and Payments Account from 01/04/2013 to 31/03/2014</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	335194.00	0.00	Opening Balance	0.00
335194.00	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

P-156 : Targeting microbial quorum sensing to demonstrate potential application of cell-cell signaling molecules from Xanthomonas group of plant pathogen in disease control PI : Dr Subhadeep Chatterjee Receipts and Payments Account from 01/04/2013 to 31/03/2014									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		0.00		Opening Balance	0.00	
0.00		Grant In Aid	2104400.00		0.00		Salaries - Manpower	197768.00	
0.00			0.00		0.00		Consumables	950000.00	
0.00			0.00		0.00		Contingencies	30000.00	
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			2104400.00		0.00			1177768.00	
0.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance	926632.00	
0.00			2104400.00		0.00			2104400.00	

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 01/04/2013 to 31/03/2014									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		0.00		Opening Balance	0.00	
0.00		Grant In Aid	2760800.00		0.00		Salaries - Manpower	165309.00	
0.00			0.00		0.00		Consumables	1200000.00	
0.00			0.00		0.00		Contingencies	50000.00	
0.00			0.00		0.00		Travel	19974.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	380852.00	
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			2760800.00		0.00			1816135.00	
0.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance	944665.00	
0.00			2760800.00		0.00			2760800.00	

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 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	0.00	
0.00		Grant In Aid	1933141.00		Salaries - Manpower	233567.00	
0.00			0.00		Consumables	1000000.00	
0.00			0.00		Contingencies	70000.00	
0.00			0.00		Travel	7787.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			1933141.00			1311354.00	
0.00		Excess of Expenditure Over Income	0.00		Closing Balance	621787.00	
0.00			1933141.00			1933141.00	

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 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	0.00	
0.00		Grant In Aid	300000.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			300000.00			0.00	
0.00		Excess of Expenditure Over Income	0.00		Closing Balance	300000.00	
0.00			300000.00			300000.00	

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-160 : Understanding the role of novel adhesins of Xanthomonas oryzae PV oryzae in Virulence and colonization in Rice PI : Dr Subhadeep Chatterjee Receipts and Payments Account from 01/04/2013 to 31/03/2014</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	0.00	Opening Balance	0.00
0.00	Grant In Aid	382000.00	0.00	Salaries - Manpower	18116.00
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		382000.00	0.00		18116.00
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	363884.00
0.00		382000.00	0.00		382000.00

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-161 : Analysis of co-regulation between DNA replication activity and amino acid homeostasis by transcription factor IciA/ArgP in Eschericia coli PI : Dr J Gowrishankar Receipts and Payments Account from 01/04/2013 to 31/03/2014</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	0.00	Opening Balance	0.00
0.00	Grant In Aid	350000.00	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		350000.00	0.00		0.00
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	350000.00
0.00		350000.00	0.00		350000.00

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 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00		Opening Balance	0.00
0.00	Grant In Aid	799600.00	0.00	Salaries - Manpower	70955.00
0.00		0.00	0.00	Consumables	477974.00
0.00		0.00	0.00	Contingencies	15000.00
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		799600.00	0.00		563929.00
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	235671.00
0.00		799600.00	0.00		799600.00

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 01/04/2013 to 31/03/2014					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00		Opening Balance	0.00
0.00	Grant In Aid	2006048.00	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		2006048.00	0.00		0.00
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	2006048.00
0.00		2006048.00	0.00		2006048.00

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 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current 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		Salaries - Manpower	26671.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			0.00			26671.00	
0.00		Excess of Expenditure Over Income	26671.00		Closing Balance	0.00	
0.00			26671.00			26671.00	

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 01/04/2013 to 31/03/2014							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	0.00	
0.00		Grant In Aid	1569682.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			1569682.00			0.00	
0.00		Excess of Expenditure Over Income	0.00		Closing Balance	1569682.00	
0.00			1569682.00			1569682.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD COE on Genetics and Genomic of Silkworms - P.I. Dr J Nagaraju RECEIPTS AND PAYMENTS ACCOUNT FROM 01.04.2013 TO 31.03.2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	3110519.00	7760392.00	Opening Balance	9645531.00
4000000.00	Grant in aid	0.00	2429598.00	Salaries- Manpower	8202456.00
		6666000.00	100000.00	Consumables	2550000.00
			245022.00	Contingencies	0.00
			0.00	Travel	137156.00
			0.00	Workshop / Training	0.00
			0.00	Equipment Maintenance	0.00
			0.00	Books & Journals	0.00
			0.00	Overheads	0.00
			0.00	Equipment	0.00
4000000.00		6666000.00	13645531.00		20535143.00
9645531.00	Excess of expenditure over income	13869143.00	0.00	Closing Balance	
13645531.00		20535143.00	13645531.00		20535143.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD COE - II : DBT Project on " Centre of Excellence for Microbial Biology" P.I: Dr J Gowrishankar, Dr K Anupama, Dr Abhijit A Sardesai, Dr Ranjan Sen and Dr Shekar C Mande RECEIPTS AND PAYMENTS ACCOUNT FROM 01.04.2013 TO 31.03.2014					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	8969700.00	Opening Balance	12818181.00
7881000.00	Grant in aid	0.00	8093406.00	Salaries- Manpower	8847332.00
			1785000.00	Consumables	1415000.00
			41075.00	Contingencies	428925.00
			510000.00	Travel	72135.00
			1300000.00	Training & Workshop	0.00
			0.00	Equipment	0.00
7881000.00		0.00	20699181.00		23581573.00
12818181.00	Excess of expenditure over income	23581573.00	0.00	Closing Balance	0.00
20699181.00		23581573.00	20699181.00		23581573.00

फोटो गैलरी
Photo Gallery



Visit of Delegation from Queensland Institute of Medical Research, Berghofer on 17th February, 2014



Visit of delegation from Ministry of Science & Technology, Argentina on 17th February, 2014



Glimpses of CDFD Foundation Day 2014



Children's Day Activity



Dr J Gowrishankar and other staff at the 5K Run organised by CDFD



Flag hoisting on the occasion of Independence Day 2013



Celebration of Hindi Day on 14 September 2013



Renewal of MoU with NIMS



Training program on Medical Laboratory Management Systems & Internal Audit for Diagnostic Division of CDFD



Scientific presentation by Prof Steve Busby, University of Birmingham, Birmingham, UK



Mock Fire Drill