THE DOSSIER

The Digest on CCR Staff Scientists and Staff Clinicians: Information, Employment and Research

September 2014 Issue 17

From the Editor

Welcome to the September issue of The Dossier, a newsletter dedicated to the Staff Scientists and Staff Clinicians (SSSC) of the CCR!

This issue contains important messages from the Director's Office and a special article by James B. McMahon, Ph.D. Information on the SS Quadrennial Review is provided by Cynthia Masison, Ph.D., while program topics for the upcoming 3rd Biennial SSSC Professional Development Day are presented by Christophe Marchand, Ph.D.

We feature Debananda Das, Ph.D., in our SSSC Corner and Sikandar G. Khan, Ph.D., describes his collaboration with the CCR Genomics Core, headed by Kathleen D. Hartman. In addition, the published work of Sven Bilke, Ph.D., is highlighted in our Author's Corner.

We hope to continue to provide pertinent information to aid in the success of SSSCs. Please send your contributions, suggestions, and comments to budhua@mail.nih.gov.

Anuradha Budhu, Ph.D. (SS)
Editor-in-Chief
Laboratory of Human Carcinogenesis

In This Issue

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From the Office of the Director

Update on CCR's Major Opportunity Program

I'm pleased to update you on the Major Opportunity (MO) Program that was launched in the fall of 2012. This program was envisioned as a way to identify ambitious ideas that, if successful, will create paradigm shifts in fundamental issues in oncology and accelerate development of innovative and successful cancer treatment strategies. As such, the goal of these projects is to test novel cancer treatments in the clinical setting within five years.

The MO program represents a new funding approach in CCR. Projects are funded for a maximum of five years and are reviewed semi-annually by the CCR Science Board against specific milestones leading to the clinic. We understand that because these projects are ambitious, it may be that one -- or even all of them -- will not reach their milestones or be fully funded for five years. A second notable feature of the MO program is that in addition to being team-based, each project is challenged to create collaborative opportunities for CCR PIs, bridging the project's clinical goals with the work of basic and translational scientists.

You may remember that 28 proposals were submitted by CCR PI's and these were vetted at a CCR PI retreat in October, 2011. In the fall of 2012, two projects were funded:

"Matrix Drug Screening for Combination Therapies in Cancer" is led by Lou Staudt, M.D., Ph.D. (Lymphoid Malignancies Branch, CCR), Craig Thomas, Ph.D. (NCATS) and James Doroshow, M.D. (DCTD). This project seeks to identify synergistic drug combinations for the treatment of cancer using a quantitative, high-throughput screening approach. These studies are in progress and collaborators from throughout the CCR have been selected for FY14 and the first half of FY15.

"Targeting the Metabolic Basis of Cancer" is led by Marston Linehan, M.D. (Urologic Oncology Branch, CCR) and Murali Cherukuri, Ph.D. and James Mitchell, Ph.D. (Radiation Biology Branch, CCR). The goal of this project is to characterize metabolic changes associated with cancer, to develop and evaluate novel approaches for imaging and therapy in preclinical models and, based on these findings, to perform clinical trials targeting these metabolic pathways in patients. Collaborators from throughout the CCR are expected to be selected in FY15.

A third pilot project, "Cancer Chromatin Profiling," was identified as having the potential to grow into a Major Opportunity. This project, led by Gordon Hager, Ph.D. (Laboratory of Receptor Biology and Gene Expression, CCR), is aimed at using genome-scale chromatin profiling to characterize cancer state, progression, response to therapy and drug development. If the project advances to the MO stage, it will accept collaborators in the future.

I encourage participation in these projects. More information on the MO Program can be found at https://ccrod.cancer.gov/confluence/display/CCRMO/Overview.

Lee J. Helman, M.D.
Scientific Director for Clinical Research

The Quadrennial Review Corner

Preparing Your Staff Scientist Quadrennial Review Package

The Staff Scientist position was created at NIH to allow the hiring and retention of highly qualified scientists to support the long-term research of a PI. The first NCI Staff Scientist was hired in 1999 and since then the number has grown to 216. CCR leadership recognizes and supports the important role Staff Scientists play at NCI and Harold Varmus, M.D. and colleagues in a recent PNAS article (http://www.pnas.org/content/111/16/5773.full.pdf+html) endorsed the Staff Scientist position as an effective resource in our current funding climate.

The CCR Quadrenniel Review Process: Staff Scientist positions are time-limited and renewable. The NIH requires Staff Scientist positions to be reviewed every four years and this is accomplished through the CCR Quadrennial Review (Quad Review) process. CCR Staff Scientists are a diverse group composed of not only bench scientists but also facility heads and bioinformatics specialists. In response to the distinct roles of the Staff Scientist, the Quadrennial Review process has evolved by developing criteria that distinguish between Facility Head/Core Managers and PI laboratory-based Staff Scientists. These changes were made with input from the CCR Staff Scientist/Staff Clinician Organization. Quad Reviews form the basis for decisions about renewal and salary adjustments. Thus it is important for Staff Scientists to understand the process and the criteria used for these reviews.

What The Reviewers Evaluate: The Quad Review submission is composed of three parts: the recommending memo from the PI, the CV and two letters of recommendation from collaborators. The CCR Review Panel is asked to comment specifically on the Staff Scientist's accomplishments over the past four years in the areas of scientific productivity (publications, patents, clinical trials), scientific presentations (talks and posters), participation in the scientific community (interest groups, faculties, technology transfer, journal reviewer, grant reviewer, SSSC organization), collaborations, mentoring/teaching, awards/honors and continuing education. This information should be clearly addressed in both the recommending memo from the Staff Scientist's PI and in the CV.

The Reviewers only know what you tell them so be sure to give specific details about everything in your CV. For example, while it is important to list all the names of your mentees, the details of how your mentoring affected their careers in terms of poster presentations, papers, educational opportunities and where they have gone since working with you may have more of an impact. Pointing out specific roles that you have played in collaborations and as a participant in a special interest group allows the Reviewers to more fully appreciate your particular involvement.

This year 39 Staff Scientists underwent a Quad Review by a committee of the ten Promotion Review Panel members and three CCR Deputy Directors. Eighty-two percent of them received a rating between Outstanding and Excellent/Outstanding. Staff Scientists make substantial contributions to their labs, the NIH campus and the greater scientific community. Your Quadrennial Review package provides you an opportunity to highlight your individual contributions with a strong supporting memo from your PI and a detailed CV.

The forms and Quad Review information can be found on the CCR SSSC Organization website https://ccrod.cancer.gov/confluence/display/CCRSSSCArchive/Home under the Practical information and Quad Review 2010 links.

Cynthia Masison, Ph.D.
Scientific Program Analyst, Office of The Director

The PI Corner

Section Editor: Lakshmi Balagopalan, Ph.D. (*SS*)

The Molecular Targets Laboratory (MTL) is unique within CCR, both in its mission and how it is structured. Its mission is to leverage CCR basic science discoveries into bioassays and high throughput screens designed to identify novel modulators of targets and pathways associated with cancer and AIDs. The MTL is structured to be highly collaborative and requires a wide range of interdisciplinary skill sets including natural products chemistry, biochemistry, molecular biology, and assay development. In order to collaborate successfully with CCR scientists on a diverse range of molecularly-targeted projects, MTL staff must be highly flexible and function well in a team environment.

Such a multi-focused research environment is ideal for scientists with a Staff Scientist appointment. Unlike postdocs and tenure tracks where independent publications and investigator-initiated research are paramount, Staff Scientists can adapt to the needs of the lab by taking a more expanded research focus. In the long run, Staff Scientists are critical in building the foundations of the laboratory and ensuring its scientific productivity.

The MTL is extremely fortunate to have two of the highest reviewed Staff Scientists in the CCR; John Beutler, Ph.D., and Barry O'Keefe, Ph.D. Their career scientific contributions have made the distinction between Staff Scientists and Principal Investigators unimportant. Indeed, both are scientifically responsible for their own research sections within the MTL and have been reviewed at site visits as Principal Investigators. John and Barry have international reputations and they play leadership roles in overseeing numerous CCR collaborations. They have made substantial scientific contributions and are active members of several key NCI committees.

With the increasing emphasis within the CCR for translational research, labs must be able to apply new technologies and approaches to accommodate multi-laboratory collaborations. Staff Scientists play a critical role in this adaptation and the MTL is fortunate to have two of the very best.

James B. McMahon, Ph.D.
Head, Assay Development and Screening Section,
Laboratory Chief, Molecular Targets Laboratory

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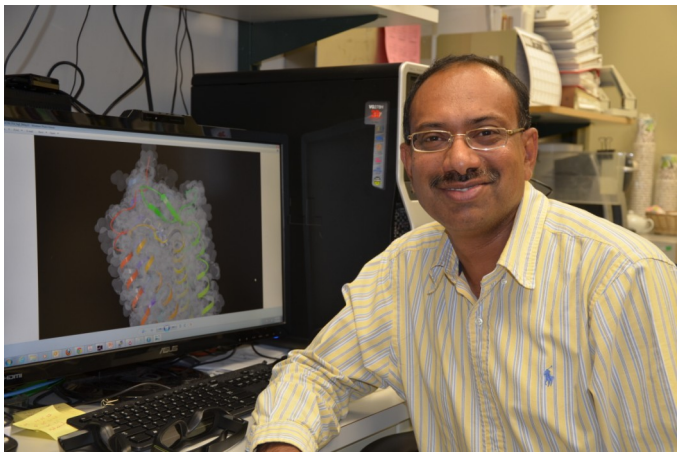
The SSSC Corner

Section Editor: Takashi Furukawa, Ph.D. (*SS*)



I am trained as a computational chemist and as a computational structural biologist. I use computational tools to elucidate the binding and interaction of small molecules with proteins. I came to NIH in the spring of 1999 for my postdoctoral work with Bernard Brooks, Ph.D., of NHLBI. After my postdoc, I worked for a company that makes molecular modeling software that is widely used in the academia and in the pharmaceutical industry. I joined the laboratory of Hiroaki Mitsuya, M.D., in the Experimental Retrovirology Section, HIV and AIDS Malignancy Branch in 2005. Most of my colleagues are virologists, and I collaborate with them to enhance our understanding of the structure to function relationships of the different cellular (CXCR4/CCR5) and viral (protease/reverse transcriptase, etc.) proteins involved in the entry and replication of the human immunodeficiency (HIV) virus.

Dr. Mitsuya and Kenji Maeda, M.D. had worked on the discovery and development of aplaviroc, a CCR5 inhibitor, with potent anti-HIV activity. However, the binding mode and the mechanism of action of aplaviroc were not known. By leveraging a host of biological data and computational tools, I built structural models of the interaction of aplaviroc and other inhibitors with CCR5. The models gave valuable insight into the mechanism of antiviral activity and helped in the design and synthesis of other novel CCR5 inhibitors.

As a group, we are actively engaged in the development of a newer generation of inhibitors that show efficacy against drug resistant HIV. Discovery of new drug molecules requires effective collaboration across multiple disciplines. Dr. Mitsuya collaborates with Arun Ghosh, Ph.D., of Purdue University for the synthesis of novel HIV-1 protease inhibitors. We work on the biological evaluation and structural interaction of these inhibitors. One of my most satisfying projects has been to discover, from computational screening of a large database, piperidinylethanamine derivatives as a completely new class of inhibitors of CXCR4 that demonstrate anti-HIV activity.

I work very closely with the biologists in the laboratory, with extramural chemists and enjoy the interdisciplinary nature of my projects. Dr. Mitsuya, having played a leading role in the discovery of four therapeutics used in the treatment of HIV (AZT, ddI, ddC, darunavir), and in the discovery of many other novel preclinical and clinical drug candidates, is an excellent mentor. My interest is in small molecule drug discovery, and I consider myself fortunate that I am in an environment that provides the freedom of academia with the applied research environment of a pharmaceutical company.

Debananda is pictured with his daughter Disha after both finished a 10K run on a cold and rainy day.

The SSSC Corner Con't

Outside of NIH, currently my favorite activity is to train for half and full marathons. I started training for these endurance events a couple of years ago. So far, I have run two full marathons and am training for the Marine Corps Marathon which takes place in late October. Running through the various trails and monuments in the DC area is very exciting and gives a very different perspective of the landscape. My kids are interested in tennis and other sports, but I might have inspired them to participate in running events too. My son, in elementary school, recently completed a 1K run. My daughter, in middle school, has completed two 10K runs this year. Her first 10K run was on a hilly course on a cold and rainy day last spring, and completing that was very satisfying.

Debananda Das, Ph.D. (SS)
Experimental Retrovirology Section
HIV and AIDS Malignancy Branch

The Professional Development Corner

The Third Biennial NCI SSSC Professional Development Day September 26, 2014, Room TE406/Joseph Fraumeni Conference Room, NCI Shady Grove, 8:30am-4:00pm

The SSSC Professional Development Committee is proud to announce its Third Biennial NCI SSSC Professional Development Day to be held in the Joseph Fraumeni Conference Room in NCI Shady Grove on September 26, 2014. Both the 2010 and 2012 Professional Development Days were great successes.

The 2014 event will deliver key information about the new grant application process in place at CCR followed by an interactive workshop about the professional networking tool LinkedIn. **It is recommended that all participants bring their laptops or mobile devices** for this LinkedIn hands-on training session. Following an interactive workshop by the Office of Workforce Planning and Development on Managing Up, a panel discussion focused on the critical issue of job stability and displacement of SSSCs will conclude the day. The panel is composed of two CCR deputy directors, one ARC deputy director, two former displaced SSSCs who successfully transitioned to other positions within the NCI and two currently displaced SSSC.

Please join us for this unique professional development opportunity and important discussions.

Christophe Marchand, Ph.D. (SS)
Laboratory of Molecular Pharmacology
Developmental Therapeutics Branch

The Author's Corner

Section Editor: Cristina Bergamaschi, Ph.D. (*SS*)

The Invisible Hand

A chromatin structure-based model accurately predicts DNA replication timing in human cells

Yevgeniy Gindin, Manuel S. Valenzuela, Paul S. Meltzer, Sven Bilke. Molecular Systems Biology 10:722 (2014)

Synthesizing a faithful and complete copy of genomic DNA within the time constraints set by the cell division cycle is central to the genomic integrity of dividing cells. Although the molecular process of copying DNA is well known, the global mechanisms responsible to ensure completeness and timeliness are still not completely understood.[1] Many researchers have worked on identifying a hierarchical molecular control mechanism that decides where and when to synthesize particular parts of the genome. Yet, these efforts have only been partially successful. In our recent paper,[2] we tried a different approach. We asked: what is the minimal control mechanism required to explain existing genome-wide replication timing data? This analysis led us to a mechanism radically different from the expected one, namely: there is none. At least, not an explicit one.

The so-called replication timing program is an experimentally accessible manifestation of global replication control. Eukaryotic DNA replicates in a specific, seemingly orchestrated temporal order. For each tissue, different genomic regions are synthesized at highly reproducible time points. The timing program shows such pronounced plasticity that it has been characterized as a "fingerprint" of cell fate decisions. Importantly, it shows characteristic aberrations in cancers. Replication timing correlates with local mutation rates and with the formation of genomic structural rearrangements. It is also tightly associated with the large-scale 3D genomic organization.

In metazoans, chromosome duplication starts at distinct DNA sequences termed replication origins. Our studies in collaboration with the NCI DNA Replication Group led by Mirit Aladjem, Ph.D., were the first to map genome-wide replication origins in human cells.[3] However, detailed mapping of origins in various tissues remains challenging and their currently relatively poor characterization has held back earlier efforts to fully understand the regulation of DNA replication. We used a computer modeling approach to circumvent this problem by performing hundreds of simulations, each implementing different hypotheses about the properties of human replication origins. The model that placed origins within DNase hypersensitive sites produced the best results. The accuracy of its predictions was astonishing, rivaling experimental repeats (Figure 1A). Pearson's correlation between the computer model and experiment was r=0.94, slightly below the r=0.95 obtained between experiments. The model correctly predicted timing plasticity (Figure 1B). It explains the recently discovered abnormal timing behavior around the TEL/AML-1 fusion often found in pediatric acute lymphoblastic leukaemia (Figure 1C). Even the length of the S-phase was correctly estimated at 8.1 hours.

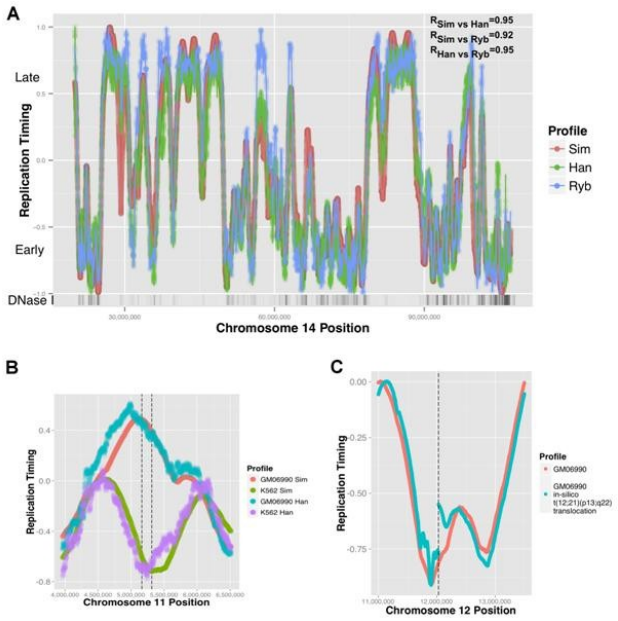


Figure 1. *(A) The replication timing program on chromsome 14 of GM06990 lymphoblastoid cells. The model predictions (red) agree well with experimental datasets (green, blue) from two different groups. (B) An example for characteristic timing differences between myelogeneous leukemia (K562 cell line) and lymphoblastoid cells (GM06990). The model predictions accurately reproduce these differences. (C) The predicted timing around an in-silico fusion gene reproduces the characteristic non-continuous timing behavior at the break.*

The Author's Corner Con't

Section Editor: Cristina Bergamaschi, Ph.D. (*SS*)

Replication origins fall into two broad categories, early and late firing. Like most researchers, our hypothesis was that this property determines global replication timing. To our surprise, the various choices made in modeling this aspect had no impact on the model's accuracy. We achieved optimal predictions even when we made no distinction at all between early and late origins. This rather subtle observation is probably the most consequential result of our work. The long sought mechanism believed to instruct origins about when to fire does not exist. If modulating the efficacy of initiation sites does not change the global timing, what then determines the global replication timing program? Random chance and the density of DNase hypersensitive sites. All origins have the same chance of initiating at any given time. But that probability is so low that it almost always takes a long time before replication actually initiates. In other words, on average, all origins fire late. Consequently, regions harboring isolated replication origins replicate late. In regions with a high density of origins, the chance of at least one getting "lucky" early on is much higher and the entire region replicates early. But, if all origins are late firing, why do experiments suggest consistent early firing origins? Origins in an early replicating region will be passively replicated most of the time, unless they are the lucky ones initiating early on.

It is common wisdom that correlation does not imply causation. But this is only true as long as there is no mechanistic process connecting one to the other. Our model provides such a mechanism, a reductionist representation of the kinetics of replication forks. It produced near perfect timing predictions from the location of DNase hypersensitive sites alone. We thus conclude that DNA accessibility is the dominant factor in selecting active replication origins.

Future work can benefit from our results in various ways. We now understand replication timing as a convenient probe of the regional chromatin state. Timing plasticity points to coordinated regional changes in the chromatin structure. Such changes are a layer of gene expression regulation that modulates DNA accessibility for transcription factors. Plastic timing during cell differentiation or cancer progression, therefore, suggests that early replicating genes are "important" in context of the cell phenotype. Furthermore, there is already a long list of genes implicated in replication timing decisions. Our results suggest that they are part of chromatin remodeling pathways. A careful analysis may uncover clues into their role in the regulation of regional chromatin states. Finally, the recently discovered association between replication timing and the 3D genomic structure opens a fascinating window into the question "What controls the large scale spatial organization of the genome"?

The Genetics Branch Chief and Head of the Molecular Genetics Section, Paul S. Meltzer, M.D., Ph.D., has a long standing interest in the role of DNA replication timing in the broader context of cancer. The project highlighted here originally grew out of the data analysis component of two collaborative projects.[3, 4]

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The Author's Corner Con't

Section Editor: Cristina Bergamaschi, Ph.D. (*SS*)

Staff Scientist Sven Bilke, Ph.D., developed the basic simulator with the aim to validate the analysis of early nascent strand chip experiments when he noticed the predictive power of his model. He worked with Yevgeniy Gindin, a graduate student in the lab, to optimize the model predictions. Upon completion of this project, Dr. Gindin received his Ph.D. based on this work. He has been nominated for the dissertation of the year award in the bioinformatics program at Boston University.

Sven Bilke, Ph.D. (SS)
Cancer Genetics Branch

Sven plays an important role in all the computational biology projects in Dr. Meltzer's lab. He enjoys training graduate students and new fellows in the lab. Sven works on his own line of research with a focus on mathematical oncology such as the study highlighted here. This work will undoubtedly impact the direction of Sven's future work and has already led to collaborations inside and outside NIH.

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1. Renard-Guillet C, Kanoh Y, Shirahige K, Masai H. Temporal and spatial regulation of eukaryotic DNA replication: from regulated initiation to genome-scale timing program. *Semin Cell Dev Biol* 2014; **30:** 110-120.
2. Gindin Y, Valenzuela MS, Aladjem MI, Meltzer PS, Bilke S. A chromatin structure-based model accurately predicts DNA replication timing in human cells. *Mol Syst Biol* 2014; **10:** 722.
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Section Editor: Anne Gegonne, Ph.D. (*SS*)

Trichothiodystrophy and xeroderma pigmentosum: The promise of exome sequencing in identifying novel candidate genes

Trichothiodystrophy (TTD) is a rare autosomal recessive multi-system disorder. TTD patients have a wide spectrum of clinical manifestations such as sulfur deficiency, brittle hair, recurrent infections, developmental abnormalities, and photosensitivity without skin cancer. Xeroderma pigmentosum (XP) is a rare autosomal recessive disease of defective DNA repair. XP patients are very sensitive to ultraviolet radiation (UVR); these patients can develop freckling in sun exposed skin before age 2, severe burns after minimal sun exposure (50% of patients), and skin cancers in children with a median age of less than 10 years. As compared to the general population, XP patients have a 10,000-fold increased risk of non-melanoma skin cancers and a 2000-fold increase in melanomas. Both TTD and XP patients are photosensitive. TTD patients have defects in some of the same genes as XP, such as *XPB* and *XPD,* but do not develop skin cancers. Interestingly, *XPB* and *XPD* genes encode proteins that play important role in nucleotide excision repair (NER) of UVR-induced DNA damage as well as transcription. We have been studying TTD and XP patients to get insight into the pathophysiology of these human diseases. The CCR Genomics Core in Building 37 led by Kathleen Hartman, was very critical in performing Sanger sequencing that helped in the diagnosis of TTD and XP patients. These studies reveal the significance of DNA repair in protection against cancer, neurological abnormalities, and development defects.

Some TTD patients have defects in NER/transcription genes (*XPB*, *XPD*, or *TTDA*) while others have mutation in *TTDN1*, a gene of unknown function. The majority of TTD patients with mutations in NER/ transcription genes are photosensitive but we diagnosed *XPD*-TTD patients who were non-photosensitive. TTD patients with mutations in *TTDN1 (C7ORF11)* were reported by others to be non-photosensitive. We diagnosed five TTD patients from four families with defects in the *TTDN1* gene: four had no photosensitivity and one had photosensitivity. Surprisingly, there were no significant differences in post-UVR cell survival of fibroblasts from non-photosensitive and photosensitive TTD patients. These results indicate that fibroblast cell killing is dissociated from clinical photosensitivity in the TTD patients.

Most XP patients have mutations in NER genes (*XPA*, *XPB*, *XPC*, *XPD*, *XPE*, *XPF,* or *XPG*) or in the error prone polymerase (pol eta). We have established cell lines from patients who have the clinical disease TTD or XP, but do not have mutations in these known genes. We are attempting to discover novel candidate genes that are involved in TTD or XP using whole-exome sequencing on genomic DNA from affected individuals and unaffected family members, including parents. The whole-exome sequencing was performed in the Genomics Laboratory within Advanced Technology Research Facility (ATFR) at Frederick National Laboratory for Cancer Research under the supervision of Daniel Soppet, Ph.D., and Bao Tran.

There are three major steps to identify the novel candidate genes. The first step is the exome capture using probes and sequencing employing high-throughput next-generation technologies. The second step is the analysis of the sequences that include approximately 1% to 2% of the whole-genome by using bioinformatics tools. The third step is the identification of candidate genes associated with the disease. We identified several genes in four TTD patients comprising three families in which the Mendelian inheritance is consistent. One TTD patient is homozygous for a mutation in GTF2E2, which encodes the beta subunit of the general transcription factor 2 E (TFIIE). We hypothesize that GTF2E2, whose function is closely linked to TFIIH, is involved in the pathophysiology of TTD. The use of exome sequencing seems to be very promising approach, which may reveal novel candidate genes involved in patients with rare inherited disorders such as TTD and XP.

The Core Corner Con't

Section Editor: Anne Gegonne, Ph.D. (*SS*)

The CCR Genomics Core

The CCR Genomics Core provides CCR Investigators with rapid processing of their DNA sequence samples and access to NanoString Digital Gene Expression technology and Miseq. The facility accepts sequencing samples from any Investigator in the NCI, and all of NIH for the NanoString. Our goal is to generate accurate results as rapidly and efficiently as possible. The electrophoreses is carried out using one of two ABI 3130XL capillary DNA sequencers or the one 3710, a 96 well capillary instrument, all located within the Core. These instruments are capable of reading up to 800 base pairs with 95% accuracy. A professional staff, assigned exclusively to the Core, makes it possible to generate this data almost as rapidly as could be expected with a dedicated machine in the Investigator's laboratory.

The Core has a NanoString Digital Gene Expression Analysis instrument. This is the only system capable of direct quantification of individual mRNAs in a biological sample without the use of enzymes or amplification. NanoString designs and builds each CodeSet and then investigators bring their samples to the core to be processed. In 2013, we added MiSeq to the services offered by the Core. Individuals meet with a committee to discuss the best experimental design.

The Core also houses an Agilent Microarray Scanner, an Agilent 2100 Bioanalyzer and recently added the Fluidigm C1 in FY14. A Bio-Rad Digital Droplet PCR QX200 as well as the BioNano Genomic Irys, a single molecule imager, will be added by the end of the year. The CCR Genomics Core intends to continue to provide high quality rapid DNA sequencing, as well as embrace new and advanced technologies that may not otherwise be available in individual laboratories. The specialized staff is dedicated to assisting CCR Investigators in achieving their research goals.

Sikandar G. Khan, Ph.D. (SS)
Dermatology Branch

Kathleen D. Hartman
Head, CCR Genomics Core

Announcements

Attend!

The Third Biennial NCI SSSC Professional Development Day
September 26, 2014
Room TE406/Joseph Fraumeni Conference Room
NCI Shady Grove, 8:30am-4:00pm

Time	Topic
8:30am	**Welcome remarks**
8:45am – 9:45am	**Current Dos and Don'ts of Grant Submissions** *Eric Hale, Associate Director, NCI Office of Clinical and Preclinical Development Resources*
9:45 am– 10:00am	**Break**
10:00am – 12:00pm	**LinkedIn Optimization for Busy Professionals; an Interactive Workshop (Bring your laptop, tablet or smartphone)** *Kelly Leonard, Business Development Leader, Taylor Leonard Corp.*
12:00pm – 1:00pm	**Lunch on your own**
1:00pm – 2:15pm	**Managing Up Interactive Workshop** *Shannon Connolly, MSW, MPA, Office of Workforce Planning and Development*
2:15pm – 2:30pm	**Break**
2:30pm – 4:00pm	**Panel Discussion with NCI Leadership and Staff Scientists: Staff Scientist/Staff Clinician Displacement** *Rena Rodriguez, CCR Administrative Resource Center Deputy Director* *Larry Samelson, CCR Deputy Director* *Jeff Strathern, CCR Deputy Director*

Looking for Editorial Experience?

The Dossier is looking for SS or SC to participate as Section Editors. If interested, please contact Anuradha Budhu at budhua@mail.nih.gov

A Call for Content

We need your input! Send your articles or suggestions with subject title "The Dossier" to budhua@mail.nih.gov

This newsletter is an avenue for you to express your ideas and thoughts on being a Staff Scientist or Staff Clinician at CCR and to make pertinent announcements.

Your contribution is very important to the success of The Dossier. Please send us your commentary, announcements, and suggestions for topics/subject matter and we will do our utmost to include your material in upcoming issues.

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