THE DOSSIER

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

March 2017 Issue 27

From the Editor

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

This issue contains important messages from the Director's Office by Tom Misteli, Ph.D., and Mary Custer, and a special article by Mirit I. Aladjem, Ph.D. We feature the collaboration between Michael J. Kruhlak Ph.D., and Philipp Oberdoerffor, Ph.D., in the Core Corner, while the published work of Erin Tran, Ph.D., is highlighted in our Author's Corner. Co-Chairs Balamurgan Kuppusamy, Ph.D., and Siddhartha Datta, Ph.D., discuss the agenda for the 2017 SSSC Retreat, while Swati Choksi, Ph.D., provides an update on the SSSC Social Networking Committee.

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

*The CCR Director regularly invites senior staff members as guest columnists to expertly inform the SSSC community on diverse aspects of the CCR.

The CCR Animal Resource Program*

The use of laboratory research animals in biomedical research is integral to the mission of the CCR. In vivo experiments are key to helping us understand cancer mechanisms and they contribute to the improvement of human health particularly through their use in development of treatments and interventions in preclinical studies. The CCR Animal Resource Program (ARP) provides our scientists with laboratory animal resources to enable high-quality cutting-edge basic and preclinical research and offers assistance to CCR researchers in the planning, development and coordination of their research animal program requirements.

The overall function of the ARP is to direct the technical and scientific planning and management of CCR's animal research program. Because animal space is a precious commodity, one of the main activities of the ARP is to coordinate the use of animal holding space within the various NCI animal facilities on both the Bethesda and Frederick campuses, NIH central animal facilities and shared animal facilities. The ARP also oversees the use of all centrally funded animal resources and provides assistance with cost effective options for animal technical support, off-site animal contracts and animal-related Interagency Agreements. Beyond merely supporting existing colonies and protocols, the ARP also facilitates the creation and use of novel genetically-engineered mouse models by offering funding support for a complete array of technologies aimed at the successful generation of transgenic and gene-targeted mice, their expansion and cryopreservation. While the vast majority of CCR animal resources involves mice, other animal models including non-human primates, zebrafish, amphibians, ungulates, canines, etc., used for cancer and HIV/AIDS research, are also supported by ARP. The ARP plays an important role in CCR strategic and resource planning in that it determines and anticipates the current and future research animal resource needs of the CCR community, gauges the feasibility of fulfilling such needs, and facilitates their realization. Another key function of the ARP is in training and education. We help new investigators and those new to animal research navigate the training and regulatory requirements, offer training in the use of mouse research models in cancer research, hands-on training sessions for specialized techniques and equipment, and educate researchers on other available training tools and resources for individual needs. An important cornerstone of the success of CCRs animal program is its partnership with outstanding contract staff who provide animal husbandry and veterinary care, technical support, diagnostic and program support across all the NCI/NIH facilities we utilize.

An important event for the animal program that you will be hearing more about in the coming months is the Association for Assessment and Accreditation of Laboratory Animal Care (AAALAC) International Site Visit that will be conducted across the NIH on the Bethesda campus on June 12-16, 2017 and on the Frederick campus in the fall of 2017. AAALAC International is a private, nonprofit organization that promotes the humane treatment of animals in science through voluntary accreditation and assessment programs. AAALAC accreditation is extremely important as it demonstrates that NCI has a commitment to a high-quality animal program where humane animal care is valued, that our users are responsible and well trained, and that our animal program is operating based on federal laws and guidelines.

A typical animal program evaluation during the AAALAC visit includes a review of the written program description, tour of facilities, meetings with the animal care staff, animal users and Animal Care and Use Committee (ACUC), review of protocols and SOPs, and tour of PI laboratories. As we get closer to the site visit date this year, you can expect to see presentations for the research staff on what to expect and how to prepare. Reminder emails on expectations will also be provided.

In short, the ARP supports all aspects of our basic science program and is a critical piece in our ability to develop and test preclinical models of a variety of

From the Office of the Director Con't

cancers and validation of promising molecular targets. The ARP is committed to helping the CCR research community advance biomedical research and minimize barriers to accomplishing research goals involving animal studies. For questions and additional information about the CCR ARP and the available resources, visit https://ccr.cancer.gov/about/animal-resource-program or contact Mary Custer, ARP Office Head.

* This article was co-authored by *Mary Custer*, Head, ARP Office.

Tom Misteli, Ph.D.
Director, CCR

The SSSC Social Networking Committee

The Social Networking Committee was started a few years ago. The purpose of this committee is to provide the SSSC community with a platform to come together in an informal setting to exchange views and experiences, both professional and personal. As we all have heard time and time again, networking is key to professional development. The Social Networking Committee holds a Coffee/Tea Break on the first Wednesday every other month from 3-4 p.m. at the Starbucks in Building 35. People gather and relax for a bit and get to know each other, but more importantly, there is a wonderful exchange of ideas. I have chaired the Social Networking Committee for the past few years and have now handed over the position to Even Walseng, Ph.D., from the Experimental Immunology Branch, NCI. Even plans to continue holding the Coffee/Tea Break at the time/place noted above. I am sure he will bring his own flavor to this committee and depending on the interest, I know he has some new ideas to bring forth. I wish him the best of luck and hope to see more of you at the Coffee/Tea Break!

Swati Choksi, Ph.D. (SS)
Laboratory of Genitourinary Cancer Pathogenesis

The Author's Corner

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

Mapping of Ebolavirus Neutralization by Monoclonal Antibodies in the ZMapp Cocktail Using Cryo-Electron Tomography and Studies of Cellular Entry

Tran EEH, Nelson EA, Bonagiri P, Simmons JA, Shoemaker CJ, Schmaljohn CS, Kobinger GP, Zeitlin L, Subramaniam S, White JM. *J Virol. 2016. 90(17):7618-7627.*

In 2014, an outbreak of hemorrhagic fever in West Africa, caused by the Ebola virus, resulted in more than 11,000 deaths (1) and underscored the need for a vaccine or therapeutic that can prevent outbreaks like this from occurring in the future. The ZMapp cocktail is one such therapeutic candidate. This cocktail consists of three monoclonal antibodies that target the Ebola glycoprotein (GP) surface spike. Studies with non-human primates showed 100 percent survival even when ZMapp was administered five days after challenge with the virus (2), and the cocktail was subsequently administered to infected patients on a compassionate care basis during the West African outbreak (3).

One of the major focuses of the Laboratory of Cell Biology, Biophysics Section, headed by Sriram Subramaniam, Ph.D., is the development of cryo-electron tomography (cryo-ET) and subvolume averaging for use in determination of viral glycoprotein structures. The use of these techniques with whole, intact viruses enables us to address important questions about the mechanisms of viral entry. For example, our work with HIV-1 (4) led to fundamental insights into the structural basis by which broadly neutralizing antibodies bind and block entry and to unexpected findings on the structural plasticity of trimeric envelope glycoproteins. We are currently applying these techniques to studies of HIV, influenza and Ebola viruses (see https://electron.nci.nih.gov/).

Previous work showed that the ZMapp antibodies can bind to a soluble version of the Ebola GP (5). However, no structural information existed about the specific Ebola strain at the source of the West African outbreak, EBOV-Makona. In collaboration with Judith White, Ph.D., (University of Virginia School of Medicine) and Connie Schmaljohn, Ph.D., (United States Army Medical Research Institute of Infectious Diseases), we aimed to characterize GP structures from EBOV-Makona virus-like particles using intact, full-length ZMapp antibodies and to decipher, using cellular entry assays, the point at which these antibodies inhibit Ebola virus infection.

Using cryo-ET and subvolume averaging, we characterized the binding locations of the full-length antibodies that comprise the ZMapp cocktail on the surface of membrane-bound EBOV-Makona GP (Fig. 1A). In this case, cryo-ET began with a sample of EBOV-Makona VLPs on a copper grid. After blotting away excess liquid, the sample was quickly plunged into a pool of liquid ethane maintained at around -180°C, freezing the VLPs in a near-native state. The frozen sample was then imaged in an electron microscope. By tilting the grid during imaging, we obtained a series of images containing views of the VLPs in different orientations. These images were combined computationally to produce a three-dimensional image, called a tomogram. Images of individual GP proteins on the surface of VLPs were then averaged to produce representative GP structures.

Cryo-ET structural analysis showed that, compared to an unbound GP structure, antibody-bound maps displayed density extending either from the top, for antibody c13c6 (Fig. 1A, top row), or from the base of the GP ectodomain in overlapping epitopes for antibodies c2G4 and c4G7 (Fig. 1A, bottom row). These results agree with the binding locations on a soluble version of a different Ebola strain, EBOV-Mayinga (5), and suggest that neither the presence of a membrane nor the close proximity of neighboring GP proteins inhibit the ability of the ZMapp antibodies to bind to GP, as no unbound classes of GP spikes were resolved for any of the antibody complexes.

Imaging VLPs rather than soluble protein allowed us to observe the ZMapp antibody-GP complexes on the membrane surface in a state similar to that found during an infection. GP spikes coated the surface of the VLPs (Fig. 1B, blue box indicates a single GP on the VLP surface), and we observed clearly defined and well-separated trimeric structures on the top and bottom surfaces of the VLPs that are presumed to correspond to top-views of GP spike proteins (Fig. 1B, red box). Tomographic slices of EBOV-Makona VLPs that had been incubated with either of the base-binding ZMapp antibodies, however, showed thin

lines of density running between the trimeric GP spikes. We measured center-to-center spacing of GP spikes in tomographic slices and used these data to model a viral surface based on the dimensions of our unbound GP structure and the estimated size of an antibody molecule (PDB ID: 1IGT). In this model, the edge-to-edge distance between GP spikes at the site of the base-targeting antibody epitope was approximately 11.5 nm, indicating that the spacing is likely to allow an antibody molecule, which is estimated to span approximately 11.5-14 nm (6), to crosslink neighboring glycoproteins (Fig. 1C).

In addition to the structural insights gained by cryo-ET, cellular entry assays showed that the base-binding antibodies inhibit entry into the cytoplasm without blocking endolysosome entry or fusion to the NPC1 cellular receptor. From these results, we conclude that the base-binding antibodies in the ZMapp antibody cocktail are likely to inhibit fusion of the viral and cellular membranes by preventing the necessary conformational changes in NPC1-bound GP. Additionally, the potential for the base-binding antibodies to crosslink neighboring spikes may result in an amplification of the neutralizing effect.

Ideally, we would like to continue this work by using cryo-ET to visualize specific conformational changes that occur to the GP spike during infection and to better understand how these changes are prevented by the binding of neutralizing antibodies, such as those found in the ZMapp antibody cocktail.

Erin Tran, Ph.D. (SS)
Biophysics Section
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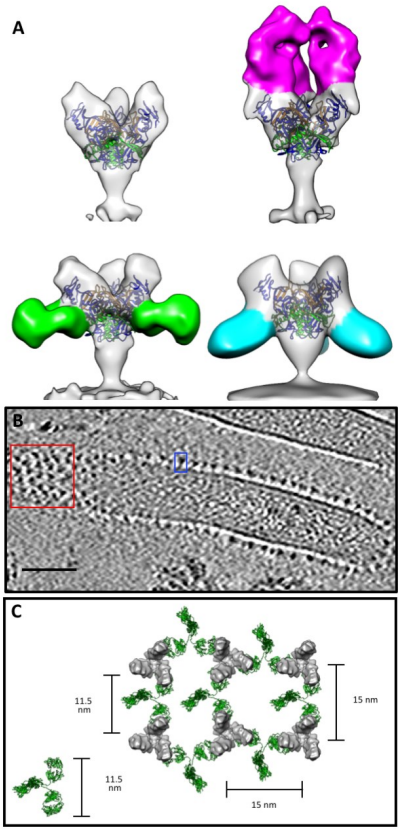

Figure 1. Structures of ZMapp antibodies bound to Ebola glycoprotein. (A) Isosurface representations of tomographic density maps are shown for EBOV-Makona GP in an unbound state (top left) or bound to ZMapp antibodies, c13c6 (top right, magenta), c4G7 (bottom left, green) or c2G4 (bottom right, cyan). (B) A tomographic slice through an EBOV-Makona virus-like particle shows GP spikes covering the membrane surface (blue box shows a single GP spike). Top views of trimeric GP spikes are seen on a portion of the membrane surface, as indicated by the red box. Scale bar is 50 nm. (C) A model of the viral surface shows the average measured spacing of GP spikes.

References:

1.Centers for Disease Control and Prevention website: https://www.cdc.gov/vhf/ebola/outbreaks/2014-west-africa/case-counts.html

2. Qiu X, *et al*. 2014. Reversion of advanced Ebola virus disease in nonhuman primates with ZMapp. Nature. **514**:47

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Erin Tran, Ph.D., is a Staff Scientist in the Biophysics Section of the Laboratory of Cell Biology in the Center for Cancer Research. Her role as a Staff Scientist includes leading tomography research within the lab, including the study highlighted in this article. Additionally, she helps to train postdoctoral fellows and students in technical and scientific aspects of the lab, such as sample preparation, data collection, data processing and analysis.

DNA Break Associated Chromatin Reorganization is an Important Modulator of BRCA1-dependent Genome Maintenance

The response to double-strand breaks (DSBs) in DNA occurs in a highly organized and conserved chromatin structural environment. Remodeling chromatin within proximity of the DNA breaks facilitates the accessibility of DNA damage response mediator proteins to the DNA lesions and initiates the signaling of the DNA damage response. Chromatin modifications associated with the DNA lesion are thought to modulate the recruitment of competing DNA damage response mediators which, ultimately, influence which DNA repair pathway is invoked. During the DNA damage response, 53BP1 is a negative regulator of DNA end resection and promotes non-homologous end joining (NHEJ), whereas BRCA1 antagonizes 53BP1 to facilitate end resection and homologous recombination (HR) in the presence of a sister chromatid. Consequently, 53BP1 is thought to be responsible for the HR defects in BRCA1-deficient cells and the resulting increase in cancer susceptibility. Thereby, the choice between DNA repair pathways has important implications for the integrity of the genome.

It is known that chromatin-associated proteins are recruited to damaged DNA and that chromatin structure is actively remodeled in the presence of DNA breaks. It was less clear, however, whether the post-translational modifications and reorganization of chromatin were able to regulate the signaling attributes of the DNA damage response and influence the DNA repair pathway used to repair the DNA lesion. Philipp Oberdoerffer, Ph.D., in the Laboratory of Receptor Biology and Gene Expression, NCI, set out to test the hypothesis that orchestrated chromatin remodeling regulates DNA double-strand break repair pathway choice. His laboratory first employed a chromatin-focused high-throughput small hairpin RNA (shRNA)-based RNA interference (RNAi) screen for mediators of HR. Their screen implicated two chromatin associated proteins, the histone variant macroH2A1 and the histone methyltransferase PRDM2. With the use of reporter cell lines, the involvement of macroH2A1 and PRDM2 as mediators of HR was confirmed. Moreover, NHEJ remained unaffected in the absence of either protein. PRDM2 establishes dimethylation of histone H3 on lysine 9 (H3K9me2), a histone mark associated with silent chromatin, whereas macroH2A1 is also frequently associated with chromatin silencing. These results indicated that a repressive or condensed chromatin structure may be involved in supporting BRCA1-dependent homology directed repair of DSBs.

It remained unclear whether the macroH2A1 and PRDM2 chromatin-associated proteins were recruited to DNA breaks, and whether they were actively involved in the reorganization of DSB-proximal chromatin. To test this, Dr. Oberdoerffer established a collaboration with Staff Scientist Michael Kruhlak, Ph.D. who runs the Experimental Immunology Branch (EIB) Light Microscopy and Digital Imaging core facility in NCI. Dr. Kruhlak had established a number of confocal microscopy-based imaging assays that allow the monitoring of the recruitment of DNA damage response mediators to DNA breaks in live cells, as well as a more specialized assay to measure the changes in chromatin structure in real time upon the introduction of DNA breaks by laser microirradiation. Taking advantage of these assays, the presence of macroH2A1 and PRDM2 at DNA breaks was confirmed ,and it further established that the two proteins act epistatically, with macroH2A1 mediating recruitment of PRDM2 (Fig 1.). After PRDM2 recruitment, the H3K9me2 histone modification increased specifically in the chromatin containing DNA breaks, providing more experimental evidence that a repressive or condensed chromatin environment is part of the DNA damage response. It became even clearer that these results were part of the DNA damage response when further experiments revealed that the recruitment of both macroH2A1 and PRDM2, as well as the accumulation of the H3K9me2 mark, were dependent on ATM, the apical kinase involved in DNA damage response mediators to DNA breaks in live cells, as well

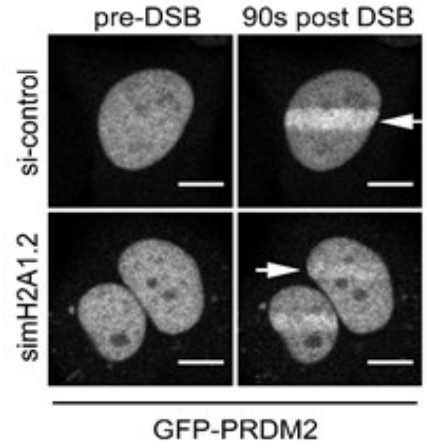

Figure 1. **Recruitment of PRDM2 tagged with GFP in control and macroH2A1.2 knockdown cells to sites of DNA breaks introduced by laser microirradiation.** Arrows denote stripe regions containing DNA breaks. Scale bars, 10 mm. Adapted from Khurana et al., 2014 *Cell Reports* 8:1049-62.

as a more specialized assay to measure the changes in chromatin structure in real time upon the introduction of DNA breaks by laser microirradiation. Taking advantage of these assays, the presence of macroH2A1 and PRDM2 at DNA breaks was confirmed and it further established that the two proteins act epistatically, with macroH2A1 mediating recruitment of PRDM2. After PRDM2 recruitment, the H3K9me2 histone modification increased specifically in the chromatin containing DNA breaks, providing more experimental evidence that a repressive or condensed chromatin environment is part of the DNA damage response. It became even clearer that these results were part of the DNA damage response when further experiments revealed that the recruitment of both macroH2A1 and PRDM2, as well as the accumulation of the H3K9me2 mark, were dependent on ATM, the apical kinase involved in DNA damage response signaling. When the chromatin structure was measured after introducing DNA breaks, it was found to first remodel to an open, more accessible state and then recondense to a more repressive state. The beginning of the recondensation phase coincided with the recruitment of PRDM2 and the establishment of the repressive histone mark H3K9me2. Importantly, the depletion of macroH2A1 or PRDM2 by shRNA and/or siRNA resulted in the impaired recruitment of BRCA1 but not 53BP1 to the DNA breaks, thereby supporting the cell reporter experiments where depletion of macroH2A1 or PRDM2 led to impaired HR (Fig 2). Collectively, the experimental results support the hypothesis that dynamic chromatin remodeling is involved in determining the DNA repair pathway choice, and Dr. Oberdoerffer's lab was able to uncover a repressive chromatin module (macroH2A1/PRDM2) that links the recondensation of DSB-proximal chromatin to BRCA1-dependent genome maintenance. The work was ultimately published in *Cell Reports* Khurana et al., 2014; 8:1049-62.

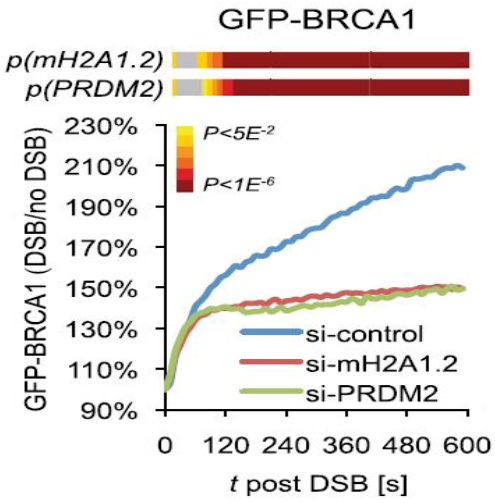

Figure 2. **Recruitment kinetics of GFP-BRCA1 to laser microirradiation induced DNA breaks in control (blue), macroH2A1.2 knockdown (red) and PRDM2 knockdown (green) cells.** The heat map indicates the p-value for each knockdown cell type over time compared to the control. Adapted from Khurana et al., 2014 *Cell Reports* 8:1049-62.

The Core Corner Con't

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

The collaboration between Dr. Oberdoerffer, a young tenure track investigator, and Dr. Kruhlak, a Staff Scientist, was successful by taking advantage of resources available in the EIB Microscopy core facility, which has a number of light microscopes available for imaging biological samples. For Dr. Oberdoerffer's work in particular, a laser scanning confocal microscope was used to introduce DNA breaks in live cells by laser microirradiation, and then monitor the recruitment of DNA damage response mediators tagged with fluorescent proteins with high resolution. Quantitative image analysis of the recruitment kinetics of the chromatin-associated proteins involved in the DNA damage response allowed Dr. Oberdoerffer to determine whether these proteins were involved in directing BRCA1-mediated genome maintenance.

Michael J. Kruhlak, Ph.D. (SS)
Facility Head,
Experimental Immunology Branch
Manager, Digital Imaging and Microscopy Facility

Philipp Oberdoerffer, Ph.D.
Head, Epigenetics of DNA Repair and Aging Section
Investigator, Laboratory of Receptor Biology
and Gene Expression

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The 13th Annual SSSC Retreat

This year's Annual CCR and DCEG Staff Scientist/ Staff Clinicians (SSSC) Retreat will be held on April 21, 2017, at the NCI Shady Grove campus. The theme of this year's retreat is "Drug Resistance and Sensitivity" a crucial aspect in the development of effective treatments for cancer, AIDS and other infectious diseases. The morning session of the retreat will begin with opening remarks by Stephen Chanock, M.D., Director, DCEG and Michael Gottesman, M.D., Ph.D., NCI, who will deliver a keynote lecture covering the main theme of the retreat. This will be followed by talks from four internationally known scientists (Matthew Holderfield, Ph.D., Stephen Hughes, Ph.D., Joel Morris, Ph.D., and Andre Nussenzweig, Ph.D., NCI) highlighting various aspects of current research on drug resistance and sensitivity. This session will conclude with an interactive panel discussion involving the four invited experts. During lunch, we plan on having small break-out sessions with the invited speakers, panelists and meeting attendees to foster informal discussion. In the afternoon, there will be two poster sessions showcasing research performed by NCI SSSC, followed by an oral abstract symposium (selected from submitted abstracts that are related to the theme of the retreat) by NCI SSSC. Finally, closing remarks will be provided by Douglas Lowy, M.D., Acting Director, NCI.

Throughout the retreat there will be an emphasis placed not only on the mechanisms underlying drug resistance and sensitivity in cancer, AIDS and other infectious diseases, but also on novel strategies to overcome drug resistance. This retreat will bring together diverse researchers from the fields of cancer and other infectious diseases, providing an opportunity to discuss recent developments. Sign up early to claim your spot as lunch meetings and guest registrations will be filled based on the registration. To register, learn more about the program and to submit an abstract, please visit: https://ncifrederick.cancer.gov/events/ssscretreat2017/default.asp

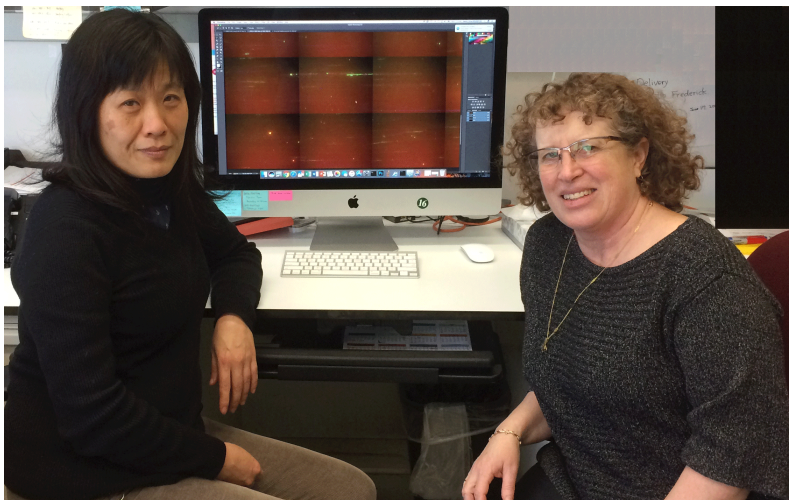
The 13th Annual SSSC Retreat
Program Agenda

8.00 a.m.	Registration and poster setup
8.30 a.m.	Welcome and Opening remarks, Stephen Chanock, M.D., Director, DCEG.
8.40 a.m.	Keynote lecture, Michael Gottesman, M.D., Deputy Director for Intramural Research, NCI.
9.30-10.50 a.m.	Presentation from the distinguished panelists: Matthew Holderfield, Ph.D., NCI. (9.30-9.50 a.m.) Stephen Hughes, Ph.D., NCI. (9.50-10.10 a.m.) Joel Morris, Ph.D., NCI. (10.10-10.30 a.m.) Andre Nussenzweig, Ph.D., NCI. (10.30-10.20 a.m.)
10.50 a.m.	Break
11.00 a.m.	Panel discussion
12.00-1.00 p.m.	Brown bag lunch with the speakers
1.00-1.45 p.m.	Poster session I
1.45-2.30 p.m.	Poster session II
2.30-3.30 p.m.	Oral abstract symposium: selected from submitted abstracts (related to the theme of the retreat) (3 or 4 presentations,12-15 min. each with 3-5 min. question session)
3.30 p.m.	Best poster awards/ Outstanding SSSC Mentor Award presentation
3.45 p.m.	Closing remarks, Doug Lowy, M.D., Acting Director, NCI.
4.00 p.m.	Group picture and adjournment.

Balamurugan Kuppusamy, Ph.D. (SS) and Siddhartha Datta, Ph.D., (SS)
2017 SSSC Retreat Co-Chairs

The PI Corner

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



Pictured are Mirit I. Aladjem (right) and Haiqing Fu (left).

My research group studies cell cycle regulation, asking how cells coordinate the duplication of their genome in concordance with the gene expression program and in the face of internal and external stress. Painting a detailed picture of the chromosome replication in human cells is very challenging, requiring us to develop, implement and continuously maintain novel tools to view and measure DNA synthesis on a single-fiber level and combine these measurements with whole-genome sequencing data. Because my small research group, like those headed by many colleagues at the NCI, consists mainly of trainees, a highly trained Staff Scientists is the lynchpin of this essential, long-term development effort.

Haiqing Fu, Ph.D., our Staff Scientist, provides us with the critical skills required to facilitate teamwork while promoting a continuous investment in novel experimental approaches. Haiqing helped introduce single-molecule analyses of DNA replication dynamics at the NIH, created a consistent and reliable workflow for replication measurements and applied these analyses to answer interesting and important questions. The years spent on technology development have paid off, allowing us to tackle research questions ranging from identifying proteins that interact with chromatin to regulate DNA replication to characterizing novel interactions between the DNA replication and the DNA repair machinery. Haiqing is leading these projects in the lab while collaborating with other groups and helping our trainees use the tools she had developed to ask their own questions about replication dynamics.

As a PI, I am confident assigning projects involving the implementation of new experimental approaches to a staff scientist, knowing that a highly trained staff scientist would be able to spend the time required for consistent and reliable method development despite the risk involved. We are now working on a new question, trying to understand why some cells re-replicate a part of their genome in response to external stimuli that interfere with proper chromatin assembly. Although this novel and risky project requires the development of an entirely new experimental approach, the stability and flexibility of the staff scientist role in the lab combined with Haiqing's considerable talents will be our best chance to tackle this challenge.

Mirit I. Aladjem, Ph.D.
Senior Investigator, Developmental Therapeutics Branch
Head, DNA Replication Group

Please share this newsletter with your colleagues and visit the SSSC website at sssc.nci.nih.gov.

Announcements

Congratulations!

2017 Director's Innovation Award Winners

Austin Duffy, M.D., (SC) Thoracic and Gastrointestinal Oncology Branch

Information on the Innovation Award Program, including a listing of prior year recipients can be found at http://ccrintra.cancer.gov/news/innovation-awards.asp

Attend!

The 2017 SSSC Retreat

Friday, April 21, 2017, NCI Shady Grove

https://ncifrederick.cancer.gov/events/ssscretreat2017/default.asp

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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SSSC Retreat
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Siddartha Datta, Ph.D.

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