THE DOSSIER

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

September 2013 Issue 14

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 Gordon L. Hager, Ph.D. In our new Opinion Corner, Tom Misteli, Ph.D., discusses journal impact factors and we highlight the work of Natalia Kommissarova, Ph.D., using the Sequencing Facility, directed by Bao Tran.

We also introduce our new Section Editor, Cristina Beramaschi, Ph.D., who will oversee our new Author's Corner, which will highlight the recent and high impact published work of our SSSCs. We also feature John J. DioGiovanna, M.D., in our SSSC 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

SSSC Co-Chairs

Adam Metwalli, M.D., Clinical Co-Chair, Urologic Oncology Branch, metwallia@nih.gov

Connie Sommers, Ph.D., Bethesda Co-Chair, Laboratory of Cellular and Molecular Biology, sommersc@mail.nih.gov

Sergey Tarasov, Ph.D., Frederick Co-Chair, Structural Biophysics Laboratory, tarasovs@mail.nih.gov

From the Office of the Director

Continuing Efforts to Strengthen our Clinical Program

I hope many of you have been following our efforts over the past several years to ensure that we are doing important clinical trials at CCR as quickly as possible. I want CCR to serve as a national model for clinical trials development and conduct. Over this time we have developed and instituted several useful new tools including "real-time" metrics of the protocol development process; consolidated resource "dashboards" to assess individual protocol cost; and a method for assessing strategic alignment of proposed protocols. We also created a protocol support office and initiated the CCR community-driven Major Opportunities program, funding three outstanding projects now under way.

The declining budgets we face make it even more imperative that we continue to seek ways to improve the effectiveness of our clinical program. I am asking us all, as a community dedicated to curing cancer, to work together to focus on the priorities most likely to reach that goal and to continually seek ways to be efficient with the publicly funded resources with which we have been trusted. Our community is made up of many talented, internationally-recognized researchers and I firmly believe we have an important role in the National Cancer Program and can continue to make important discoveries that improve the lives of patients.

To learn more about how we are continuing to seek to improve the clinical program so that it remains vital and efficient, I invite interested Staff Clinicians and Staff Scientists to attend the upcoming "Clinical All Hands" meeting on September 11, 2013, in the Lipsett Auditorium, Building 10, at 2:00 PM. Note that the meeting will be video cast to NCI-Frederick, building 549, conference room B. At the meeting, I will discuss a proposal to reorganize part of the clinical program around areas of strength and to retool our process for the scientific review of proposed protocols. Please join us and take an active role as part of our clinical research community as we work together to make a real difference in the fight against cancer and HIV.

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

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

The Opinion Corner

Eliminating the Impact of the Impact Factor

The Impact Factor is the most popular numerical measure of a scientist's work. Despite many well-documented flaws, the Impact Factor is commonly used in recruitment, appointment, and funding decisions. A diverse group of stakeholders is now making a concerted effort to combat misuse of the Impact Factor and is calling for the development of more accurate measures to assess research. The group has issued the *San Francisco Declaration for Research Assessment*. You too can join the campaign.

It is in the nature of us scientists to measure things—even things that are difficult to quantify such as an individual scientist's performance and impact. A commonly used metric to describe scientific impact is the journal Impact Factor (IF). The IF is a journal-specific number that is calculated as the ratio of total citations a journal receives over the preceding two years divided by the total number of citable articles published during that time. Each paper in a given journal then is described not by its own citation tally but rather by the journal-wide Impact Factor.

The IF is pervasive in the scientific community. Scientists refer to it casually in conversation to convince colleagues of the importance of their own papers, or they wonder how a paper ended up in "a journal with such a high Impact Factor." Students and postdocs want to publish only in "high Impact Factor" journals, and the IF is frequently used in recruitment, tenure, and granting decisions when a candidate's past publication performance is assessed.

The IF was never meant to be used in that way! It was introduced in the early 1960s to aid librarians in stocking their shelves with the journals that were most important to their constituents. It was not intended to assess the research quality or impact of a single paper, let alone an individual scientist's performance.

Numerous flaws in the IF have been pointed out over the years. Some of the more troublesome shortcomings are: a journal's IF can be driven by a few, extremely highly cited articles, yet all articles published in a given journal, even those that are never cited, are presumed to have the same IF; the IF does not say anything about an individual article, yet conclusions about a particular paper are often drawn; the IF can be manipulated by journals in many ways, for example by publishing more review articles, which are generally more highly cited, thus distorting the perceived impact of the journal's primary research articles; and the IF is sensitive to the nature of the scientific content and the size of a given field, with smaller communities naturally generating fewer citations.

Fortunately, awareness of the many flaws of the IF has grown over the last few years. Now, a group of prominent journal editors and publishers of scholarly journals, as well as representatives from major funding agencies and research institutions, is speaking up as one voice to highlight the limitations of the IF and to call for a concerted effort to improve the ways scientific output is assessed by funding agencies, academic institutions, and scientists themselves. The group has developed a set of specific recommendations and published them in the *San Francisco Declaration on Research Assessment*. The Declaration bears the signatures of about 200 institutions and individuals and is available at http://www.ascb.org/SFdeclaration.html for public signature by any party who wants to express its support.

The key points of the declaration are simple, yet profound. The central recommendation calls for the elimination of the use of the IF, and all other journal-level metrics, in funding, appointment, award, and promotion decisions. We need to return to a culture where these often life-changing decisions are made by careful, in-depth consideration of a candidate's work and future potential rather than merely adding up numerical values. This effort will require that funding agencies and institutions explicitly define, and adhere to, criteria they will use for evaluation of scientific productivity.

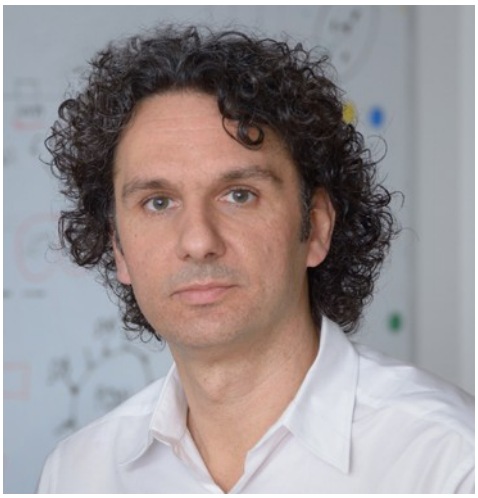
A second broad recommendation is to refrain from using publications and citation as the primary indicators of impact. Scientists produce much more than just publications. All research outputs—minable datasets, software, equipment and technology development, contributions to large-scale collaborative efforts, and reagents made available to the community—should be considered when assessing a scientist's contributions. In addition, an individual's

influence on policy and on scientific or clinical practice should be included in any evaluation.

Although initiated by a group of editors and publishers, the declaration is also self-critical and challenges publishers not to use the IF for promotional purposes. This includes removing mention of the IF from their websites and refraining from using it in advertising materials. In addition, rather than promoting a single metric, publishers are urged to provide a range of publication metrics, which will allow readers to more accurately assess the strengths and weaknesses of a given article or journal. Given that most journals are nowadays electronically published, extraction of a diverse set of publication metrics is easily feasible.

A final important recommendation is to call on scientists to do their part in eliminating inappropriate use of the IF. Active scientists should refrain from buying into the IF frenzy. When serving as a member of a recruitment or tenure committee, scientists should not consider IF-based information in their decisions. More importantly, we must teach our students and postdocs about the limitations of the IF and not promote the notion that only work in high Impact Factor journals is worth reading and building on for future research. Importantly, scientists must challenge others when faced with inappropriate use or interpretation of journal-based metrics, be it on formal committees or in casual conversation with colleagues.

The IF was created to assess a journal as a whole. But it is now often inappropriately used to assess the quality of individual articles and scientists. We scientists are not entirely innocent in bringing about the misuse of the IF. We like to measure, we like to compete, and we like numbers. The IF was a tempting way to satisfy all those inclinations despite its inappropriateness and its flaws in assessing individual impact. Scientists often express disdain for the IF, but most play along, because everyone else does. The *San Francisco Declaration on Research Assessment* is a chance to break this Catch-22. Make your voice heard to eliminate the impact of the Impact Factor by signing the *San Francisco Declaration on Research Assessment*.



Tom Misteli, Ph.D.
Head, Cell Biology and Genomes Group
Laboratory of Receptor Biology and Gene Expression
Editor-in-Chief, The Journal of Cell Biology

Originally published in *The Journal of Cell Biology,* May 20, 2013.

Meet our New Section Editor

Dr. Cristina Bergamaschi received her Ph.D. in Molecular Medicine specializing in Immunology from the University of Milan, Italy, in 2008, and she has worked in the Human Retrovirus Section (Vaccine Branch) headed by George N. Pavlakis, M.D., Ph.D., at NCI since 2005. In 2012, she was hired as Staff Scientist in the Human Retrovirus Pathogenesis Section (Vaccine Branch) headed by Barbara K. Felber, Ph.D., at NCI.

Dr. Felber's laboratory focuses on the posttranscriptional mechanism of gene regulation and on the development of novel vaccine strategies against HIV and cancer. Within the lab, Dr. Bergamaschi's main project investigates the molecular biology of cytokines and their role in lymphocyte homeostasis.

We encourage SSSCs to share their recently published work in the new SSSC Corner of *The Dossier*, The Author's Corner. The Author's Corner aims to highlight recent findings from SSSCs, with high impact for the scientific community. If you work has been recently published and you would like to see it considered to be highlighted in *The Dossier*, please email: bergamac@mail.nih.gov, with subject title "The SSSC Author's Corner".

The PI Corner

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

The revolution in genome biology has transformed research in causes and treatments for cancer. Few areas of the CCR research portfolio are untouched by the innovations developed almost daily in this rapidly developing technology. While major opportunities in cancer research are presented by these advances, traditional personnel and staffing structures are not well designed to move quickly in these areas. Few postdoctoral fellows trained in current cell and molecular biology programs are equipped to perform the complex data mining analyses needed for large scale genome wide datasets. The Staff Scientist appointment has provided an important mechanism to address the growing needs in this field. Two Staff Scientists in the Laboratory of Receptor Biology and Gene Expression have been essential to the research program now well developed in our group. Mia Sung and Songjoon Baek are both Ph.D. mathematicians with strong skill sets in informatics. These scientists are increasingly recognized within the international community for their development of novel algorithms to analyze chromatin transitions in mammalian cells, as well as new approaches to address large scale nuclear architecture with the multiple chromosome conformation capture methodologies increasing utilized in cancer research. The Staff Scientist appointment will continue for the near-term future to form the backbone of our efforts in studies that address the organization of the cancer genome.

Gordon L. Hager, Ph.D.
Head, Hormone Action and Oncogenesis Section,
Chief, Laboratory of Receptor Biology
and Gene Expression

The Author's Corner

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

53BP1 Mediates Productive and Mutagenic DNA Repair Through Distinct Phosphoprotein Interactions.

Callen E, Di Virgilio M, Kruhlak MJ, Nieto-Soler M, Wong N, Chen HT, Faryabi RB, Polato F, Santos M, Starnes LM, Wesemann DR, Lee JE, Tubbs A, Sleckman BP, Daniel JA, Ge K, Alt FW, Fernandez-Capetillo O, Nussenzweig MC, Nussenzweig A. *Cell. 2013 Jun 6;153(6):1266-80.*

Our cells are constantly exposed to a number of sources of DNA damage. Both endogenous metabolic processes as well as exogenous agents can cause a plethora of lesions. Among these, DNA double strand breaks (DSBs) are one of the most lethal for our genetic material. The proper repair of these lesions is critical to avoid the propagation of mutations that can lead to cancer.

DSBs are repaired by two major pathways: Homologous Recombination (HR) or Non-Homologous End Joining (NHEJ). Although there is still a lot to learn and understand about how these processes work, we know that the timing of their activity largely differs and is highly regulated. HR acts primarily during the S and G2 phases of the cell cycle, when a sister chromatid is available. This guarantees the "error-free" repair of the lesion using the sister chromatid as a template. On the contrary, NHEJ acts by re-ligating DNA ends mainly during the G1 phase of the cell cycle. In DNA-end resection, an evolutionarily conserved process that generates long stretches of 3' single-stranded DNA, a critical choice of DSB repair pathway is made, as it commits cells to HR, while suppressing NHEJ. Thus, both mechanisms need to be tightly regulated and coordinated so that they do not interfere with each other.

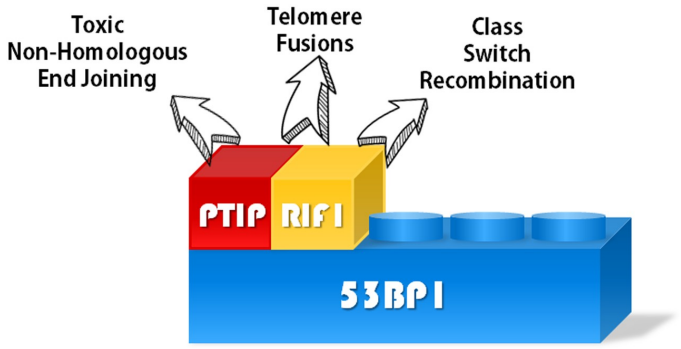


Figure 1. PTIP and RIF1 separate 53BP1 functions in productive and mutagenic DNA repair.

The DNA damage response protein, 53BP1, is a p53-binding protein that undergoes phosphorylation by the Ataxia-Telangiectasia Mutated (ATM) kinase and accumulates at the DNA break site forming nuclear foci, in response to damage. This factor has been described as one of the most critical determinants of the DSB repair pathway choice, promoting NHEJ and blocking end resection. One of the most surprising consequences of this feature was previously uncovered by the groups led by Andre Nussenzweig, Ph.D., and by his collaborator Toren Finkel, M.D., Ph.D. In Bunting *et al.* and Cao *et al.*, the authors showed that 53BP1 is responsible for the aberrant rearrangements that take place in BRCA1-deficient cells by NHEJ. In the absence of 53BP1, repair by HR is restored and the phenotypes associated with BRCA1 deficiency are reverted. Most strikingly, BRCA1 deficient mice are no longer susceptible to cancer when 53BP1 is absent. However, it was not yet clear how 53BP1 carries out its functions in DNA repair.

The Author's Corner Con't

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

Recently, Rif1 was identified by several groups as a downstream regulator of 53BP1, however its absence did not result in a fully restored HR pathway. This suggested that other factors acting downstream of 53BP1 could be implicated in the reversal of BRCA1 deficiency. In a recent study (*Callen et al.; http://www.ncbi.nlm.nih.gov/pubmed/23727112*), led by Andre Nussenzweig, Ph.D., and developed by Staff Scientist Elsa Callen, Ph.D., at the Laboratory of Genome Integrity (LGI), PTIP was identified as the phospho53BP1-binding factor required for 53BP1-mediated inhibition of HR in BRCA1-deficient cells. The independent and differential binding of Rif1 and PTIP to phosphorylated 53BP1 allows for the establishment of a separation of functions of 53BP1 in DSB repair (Figure 1). This exciting discovery opens new avenues for therapeutic strategies. For example, by specifically targeting the PTIP/53BP1 interaction, one could restore DNA repair in BRCA1-deficient cells without compromising other processes. Future studies will be needed to dissect how the 53BP1 effector proteins RIF1 and BRCA1 carry out their anti-recombination functions, understand the spatial/temporal relationship between these factors, and determine if there are other effectors involved in these DNA repair reactions.

Elsa Callen, Ph.D. (SS)
Laboratory of Genome Integrity

Elsa plays a pivotal role in the maintenance and organization of the lab. She is highly devoted to the training of new postdoctoral fellows and she actively participates and supports several ongoing projects in the lab. In addition, Elsa carries on her own line of research within the lab, the results of which are highlighted in the aforementioned study. Undoubtedly, this investigation has had a major impact in the direction of the current studies that Elsa is in charge of, and has opened more lines of research both at LGI as well as collaborations with other groups and clinicians inside and outside NIH.

References:

1. *Bunting, S. F., Callen, E., Wong, N., Chen, H. T., Polato, F., Gunn, A., Bothmer, A., Feldhahn, N., Fernandez-Capetillo, O., Cao, L., Xu, X., Deng, C. X., Finkel, T., Nussenzweig, M., Stark, J. M., and Nussenzweig, A. 53BP1 inhibits homologous recombination in Brca1-deficient cells by blocking resection of DNA breaks (2010) Cell 141, 243-254.*

2. *Cao, L., Xu, X., Bunting, S. F., Liu, J., Wang, R. H., Cao, L. L., Wu, J.J., Peng, T. N., Chen, J., Nussenzweig, A., Deng, C. X., and Finkel, T. A selective requirement for 53BP1 in the biological response to genomic instability induced by Brca1 deficiency (2009) Mol Cell 35, 534-541.*

Identification and Functional Studies of Non-Coding RNAs Regulating the Expression of MHC Class I Genes in Mice using RNA-seq.

The major histocompatibility complex (MHC) encodes a group of cell surface glycoproteins that play an important role in the immune system and autoimmunity. One of the two major classes of MHC molecules, class I, presents cytosolic antigens to cytotoxic T lymphocytes, which selectively kill the cells presenting antigens of a foreign (viral or tumor) origin. MHC class I molecules are expressed in all nucleated cells, but the level of their expression is tissue specific and is also regulated dynamically by extracellular signals (hormones and cytokines). The MHC class I genes constitute a multigene family, with extensive polymorphism. Such polygenism and polymorphism greatly extends the range of peptides that can be presented to T cells.

Complex regulation makes MHC class I an excellent model to uncover principles governing eukaryotic transcription, which is the main focus of the laboratory of Dinah Singer, Ph.D., at the Experimental Immunology Branch, NCI. Transfection techniques, transgenic animal models, in vitro transcription assays, chromatin immunoprecipitation and qRT-PCR data revealed that expression of MHC class I genes is regulated: (1) by binding of regulatory factors to the known elements of core promoter, (2) by the usage of alternative transcription start sites, (3) by controlling preiniation complex assembly and release into elongation via synchronized activity of transcription factors, and (4) epigenetically via histone modifications.

Recent studies revealed that a great fraction of eukaryotic genomes is transcribed into long (over 200 bases) and short (under 200 bases) noncoding RNAs (ncRNAs). Important gene regulatory functions were described for some of these RNAs. Preliminary results obtained in Dr. Singer's lab suggested that antisense transcription occurs around the promoter and in the body of MHC class I genes, and that an MHC class I promoter is bidirectional. A number of scientists in lab concentrated on characterizing the mechanisms that give rise to ncRNA originating from MHC class I cluster and revealing the regulatory role of this ncRNA.

We decided to determine the comprehensive transcriptional profile of the MHC class I cluster using RNA-sequencing (RNA-seq) technology. We chose RNA-seq technology over microarray technology, because it provides a better resolution (up to single base), larger dynamic range to quantify differences in RNA level (>8,000 fold vs a few hundred fold), and requires less RNA. We performed the sequencing at the NCI Sequencing Facility at the Advanced Technology Research Facility (ATRF), and the CCR-IFX Bioinformatics Core Facility provided help with data analysis.

The potential difficulty in analyzing MHC class I transcriptome lies in the high similarity of the sequences of these polymorphic genes. The second difficulty we faced was the need to detect low level ncRNAs. To distinguish the polymorphic transcripts and to detect rare transcripts, very deep sequencing was required, adequate library preparation was extremely important, and the bioinformatics analysis had to be adjusted accordingly. Both facilities were very helpful and responsive to our requests.

RNA-seq analysis of the RNA extracted from mouse spleen showed that in many genes the transcripts were restricted to the open reading frames. However, measurable RNA was also detected both upstream and downstream of many genes, including MHC class I genes. We will continue our collaboration with the NCI Sequencing Facility to determine the tissue specific profile of coding and ncRNA at the MHC cluster in the tissues that express MHC class I at lower levels, such as kidney and brain, and from mouse embryonic fibroblasts. We will also detect how ncRNA level and profiles change in response to extracellular signals, such as interferon gamma. In the future, we will modulate levels of these ncRNAs either by depleting them with RNA sponges or by overexpressing them from a vector. We will follow the alterations in the transcriptional profile of the MHC cluster caused by these modulations. The use of RNA-seq will allow us to later expand our analysis of the effect of these ncRNAs to other regions of the genome. We believe that this systematic approach will uncover a novel type of MHC class I regulation though ncRNA.

The Core Corner Con't

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

The Sequencing Facility:

The recent introduction of DNA sequencing instruments capable of producing millions of DNA sequence reads in a single run is rapidly changing the landscape of genetics and cancer biology. This technology is providing the ability to answer complex questions with unimaginable speed. The Sequencing Facility (SF) is a second and third generation high-throughput sequencing core established by the CCR, located at the Frederick National Lab: ATRF. The SF offers sequencing services on both the Illumina and Pacific Biosciences' platforms. These two platforms have complementary strengths and can be used separately or in a combined approach to answer many genomics questions. The established Illumina platform, with access to three state-of-the-art HiSeq 2000s, two GA IIx sequencers and one Miseq sequencer, has been in production at the SF since 2009. The newer Pacific Biosciences platform, the PacBio RS, was acquired by the SF prior to its commercial release as a part of their Limited Pre-Release program, giving SF an advantage in developing expertise in this platform early in its development. Both offer unique advantages for different sequencing applications, including whole genome sequencing, exome and transcriptome sequencing, targeted amplicon resequencing, ChIP-seq, base modification detection, and sequencing complex repeats, secondary structures, and AT and GC-rich sections of DNA. SF scientists provide consulting throughout the design and execution of your project to ensure the most effective experiments are being conducted to help you efficiently address your research needs. For more information about the SF, please visit http://ncifrederick.cancer.gov/atp/genetics-and-genomics/sequencing-facility/.

Natalia Komissarova, Ph.D. (SS)
Experimental Immunology Branch

Bao Tran
Laboratory Director
Sequencing Facility
Advanced Technology Research Facility

The SSSC Corner

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

Since arriving in 1980, I have seen many changes in the NIH, in my research directions, and in positions I have had at NCI or NIAMS. As a dermatologist, my interest has been in clinical, basic and translational research, which I believe are a continuum. Initially, I worked with Gary Peck, M.D., developing retinoid therapy for skin disorders, and skin cancer treatment and chemoprevention. Gary led this ground- breaking work here at NIH, and these novel and powerful drugs changed the face of dermatology by providing treatment for very severe inherited skin disorders (genodermatoses) and severe, disfiguring nodulocystic acne. I was interested in the mechanisms of retinoid action and collaborated with Gerald Chader, Ph.D., in NEI. I spent part of my time working in his lab studying retinoid receptors, which they had characterized in the eye. It was exciting for me to find and describe these receptors in skin. Much of my work centered on how retinoids could be helpful (efficacy) and problematic (toxicity). Understanding drug toxicity is necessary to know when to use drugs and how to follow for adverse events, and this is essential to being able to use drugs safely. Retinoids are now widely used not only for skin disorders and skin cancer chemoprevention, but also for the treatment of a range of malignancies.

Our work with retinoids led us to care for many patients with rare inherited disorders, which allowed us to develop considerable expertise in these conditions. In the late 1980's the great excitement was in positional cloning, mainly by genetic linkage analysis. One of the greatest resources at NIH is the diverse group of bright, enthusiastic, hard-working potential collaborators. I was extremely fortunate to develop a close working relationship with geneticist Sherri Bale, Ph.D., and molecular biologist John Compton, Ph.D. We forged a collaborative group targeting the genes underlying these rare inherited disorders. We carefully chose diseases based on our ability to acquire the necessary resources, and traveled on field trips across the U.S., and sometimes as far away as Cairo, Egypt, to study families. In doing so, we found the genetic basis of several genodermatoses. Since we were unable to test for these genes here at NIH, Sherri and John left NIH to start the successful genetic testing company Genedx. Throughout this time, I also worked with Kenneth Kraemer, M.D., on characterizing the clinical and molecular basis of DNA repair disorders (xeroderma pigmentosum and trichothiodystrophy) and their underlying pathophysiology. For 13 years, I split my time between Brown University in Providence, where I was Director of Dermatopharmacology and Professor of Dermatology and the NIH. Throughout that time, I came back to NIH every other week (yes, for 13 years) for an intensive research week. In 2010, I returned to NIH full time as a Staff Clinician focusing on DNA repair diseases and Dermatology consultation service.

John is pictured with his two Yorkie terriers, Moose (left) and Bear (right).

Outside of NIH, I enjoy traveling and puttering around in my house and garden in Bethesda with Julian, my partner of 23 years, and Moose and Bear, two Yorkie terriers who control most of the non work aspects of my day. I like restoring and repairing, preferably old things, and I particularly relish when something I have "fixed" actually works!

John J. DiGiovanna , M.D. (SC)
Dermatology Branch

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 regarding 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 manner and we will do our utmost to include your material in upcoming issues.

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