

PASPCR

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Newsletter



The **PASPCR Newsletter** is published three times a year and is intended to serve as a regular means of communication for the members of our Society. The PASPCR Newsletter is distributed via e-mail, in pdf format, on the first of April, August and December and it will continue to be posted on the web site of the Society.

Preparations for 17th PASPCR Meeting, spear-headed by Dr. Sancy Leachman, are progressing well. The meeting will be held in Park City, Utah, on September 19th-22nd, 2012. Further information on the meeting can be found on page 5 of this newsletter.

In this issue, we continue the “*Laboratory Updates*” section with a column by Dr. Vincent Hearing. Starting with this number, we introduce a new section under the Pigmentation Community Connections called “*Clinical Insights*”. This series debuts with a column written by physician-scientist Dr. David Adams.

We hope you enjoy this issue. We encourage you to send us your comments at our email address paspcr.newsletters@gmail.com. Let us know what you would like to see in the letters, suggest sections you think would be useful to include, and recommend any changes that you would like to see.

We also encourage you to let us know about meetings that you think would be of interest to members of the Society. If you attend a scientific meeting at which you heard about work which you think will be of interest to the membership of the PASPCR, please write a few paragraphs summarizing what was presented and share it with us. Also, keep us updated on any “*Members in the News*” so we can spread the word of your successes.

Also, if you know of training courses that would be of interest to the PASPCR members, please let us know and we will add them to a new section to our Calendar of Events.

This is **your Newsletter**, and we depend upon you to help us ensure it best serves the Society’s needs. We look forward to hearing your ideas and suggestions and to continue working together to compile the Newsletters for our Society.

The PASPCR Newsletter Editorial Team would like to thank to all our contributors for their columns submitted to us for inclusion in the letters.

PASPCR Newsletter Editorial Team

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The PASPCR Web Site can be found at:

<http://www.paspcr.org>

**The PanAmerican Society for
Pigment Cell Research**

C/O Andrzej T. Slominski, M.D., Ph.D.
University of Tennessee Health Science Center
Department of Pathology and Laboratory Medicine,
930 Madison Avenue, Room 525 (Clinical Office),
Memphis, TN 38163, U.S.A.

Officers:

Greg Barsh

President

Caroline Le Poole

President-elect

Andrzej Slominski

Secretary/Treasurer

Council Members:

Robert Cornell (2010-2012)
Gertrude-Emilia Costin (2012-2014)
Deborah Lang (2011-2013)
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Michael Marks (2010-2012)
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Richard Spritz (2010-2012)

IFPCS Representatives:

Andrzej Slominski (*Secretary*)
Greg Barsh (*Council Member*)
Frank Meyskens (*Council Member*)

CALENDAR OF EVENTS

2012

**The Annual Meeting of Society for Investigative Dermatology
& 75th Anniversary Celebration**

Date and place: May 9-12, Raleigh, NC, USA

Web-site: <http://www.sidnet.org/AnnualMeeting.aspx>

2012

17th ESPCR

Date and place: September 11-14, Geneva,
SWITZERLAND

Web-site: www.espcr.org/ESPCR2012

2012

17th PASPCR

Date and place: September 19-22, Park City, UT, USA

Web-site: <http://www.huntsmanccancer.org/paspcr2012>

2012

5th ASPCR

Date and place: November 3-4, New Delhi, INDIA

Web-site: <http://www.aspcr2012.com/home>

2012

24th JSPCR

Date and place: November 24-25, Nagahama, JAPAN

Web-site: <http://jspcr.jp/english/meeting.html>

2012

The 51st Annual Meeting of American Society for Cell Biology

Date and place: December 19-20, San Francisco, CA, USA

Web-site: <http://www.ascb.org>

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Publication Committee

Gertrude-Emilia Costin, Ph.D., M.B.A.

Editor

Institute for In Vitro Sciences, Inc. (IIVS)
30 W Watkins Mill Road #100
Gaithersburg, MD 20878, U.S.A.
(301) 947-6524
ecostin@iivs.org

Prashiela Manga, Ph.D.

Associate Editor

New York University School of Medicine
Department of Dermatology
Smilow Research Center
522 First Avenue, Room 401
New York, NY 10016, U.S.A.
(212) 263-9086
prashiela.manga@nyumc.org

William S. Oetting, Ph.D.

University of Minnesota
Department of Medicine - Genetics
MMC 485; 4-12 Moos Tower
515 Delaware Street S.E.
Minneapolis, MN 55455, U.S.A.
(612) 624-1139
oetti001@umn.edu

Andrzej T. Slominski, M.D., Ph.D.

University of Tennessee
Department of Pathology and Laboratory Medicine
930 Madison Avenue, Room 525 (Clinical Office)
Memphis, TN 38163, U.S.A.
(901) 448-3741
aslominski@uthsc.edu

CORPORATE SPONSORS

by Dr. Andrzej Slominski

The PASPCR would like to acknowledge and thank our Sponsors. The list below reflects contributions made during the year of 2011. In the past, financial gifts from our Sponsors have allowed our Society to increase benefits to the membership far out of proportion to the actual dues collected from members. We gratefully acknowledge the contributions for the XXIst IPCC made through PASPCR as follows:

Johnson & Johnson Consumer Companies
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University of British Columbia

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PASPCR PRESIDENT'S CORNER

Thanks to our intrepid newsletter editor and new council member for the opportunity (and encouragement) to provide a few thoughts about the Society, science, and the relationship between the two...

First, with regard to Society news, plans are well underway for this year's meeting in Park City, Utah, the state that gave us one of the more intelligent Republican candidates for the 2012 Presidential race (more on the Huntsman family below). I look forward to this meeting not only as an opportunity to enjoy the delightful environment in Deer Valley but, more importantly, to enrich and invigorate our research. Featuring speakers and subjects that expand our typical boundaries will certainly be fun, but can also help the Society grow in terms of both membership and scientific impact. Additional efforts are underway to provide financial support for a larger number of students and postdocs to attend and participate, which will help ensure that the Society continues to thrive. The 2012 meeting will be, in many ways, an experiment, and I hope what we learn can be applied to the 2013 meeting, whose location, local organizer, and dates should be announced in the near future.

What about science? As Frank Loesser wrote, "Baby, it's Cold Outside". Cold in terms of NIH funding levels, cold in terms of industrial partners who find themselves struggling to support their R&D groups, and cold in terms of an appreciation for basic research. Recent items in the media are particularly telling, even disturbing, but also inspiring. Published last fall and written by Shawn Otto, "Fool Me Twice" describes the "assault on science in America". Otto tries, although not all that successfully, to avoid partisanship; indeed, he places much of the blame on an irresponsible media and on politicians that line both sides of the aisle. Reviews for the book include comments suggesting that Otto is "preaching to the choir", and that the people most in need of the message are the least likely to hear it. I'm not so sure; Otto's book is a call to arms for science practitioners to also be science advocates, engaging our neighbors and our communities to explain what it is we do, and why we do it.

Speaking of which, Huda Akil, a neuroscientist at the University of Michigan, wrote an editorial at the end of last year about science in America. Her words resonated with me.

"...there is a more fundamental reason, I believe, to support science in this country and to keep on doing so even during tough times. A reason that the hat the world seems to recognize but we in America seem to be forgetting: Discovery is at the heart of what America is. It represents an attitude that rings American - a fundamental belief that when you seek, you discover, and when you discover, you transform. In this culture, unlike older cultures, truth is not fully defined by what is handed down. Truth is sought, and new knowledge is prized but held with the expectation that a greater depth of understanding is always around the corner. In America, more than in any other place I know, it is not only possible, but it seems essential, to know more and do better."

Huda's words are inspiring, not only because they speak to what can be truly exceptional about America, but also because they appeared in the Washington Times, where a readership perhaps less

likely to buy Otto's book will nevertheless be exposed to his message.

Which brings me back to the provincial sensitivities that develop from local culture, and politics. Having spent most of my professional life in West Coast academic institutions, I lived, as Bill Maher might say, in a very thick bubble, where reason and liberalism always prevailed, and the voices of Rupert Murdoch were muffled if not silenced. Don't get me wrong, that's a very comfortable bubble, but having spent a couple years living in Huntsville, Alabama, Murdoch-inspired voices come through, painfully loud and clear. In this context, I found it particularly sad when Jon Huntsman, Jr., dropped out of the 2012 Presidential race, and particularly inspiring, to read about Jon Huntsman, Sr. In Utah this fall, I hope all of us can appreciate what the Huntsman family has done for science and society, and that we can be inspired to advocate, and educate, outside our own bubbles.

Greg Barsh, M.D., Ph.D.
PASPCR President

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**LETTER FROM PASPCR
SECRETARY/TREASURER**

Dear PASPCR members,

I believe that everybody is ready to attend the next PASPCR Conference in Park City, Utah, September 19-22, 2012, chaired by Sancy Leachman. Sancy has been extremely successful in organizing the support, securing venues and her NIH Conference grant got extremely high score. We are encouraging trainees, postdocs and junior faculty to send applications for travel awards. To be eligible the applicant has to be the first and presenting author of the poster or oral presentation, and to be a member of the PASPCR in good standing at least 3 months prior the application. The priority in distributing travel awards will be given to students, trainees and postdocs. Still having residual funds from the PASPCR meeting in

Memphis in 2009 and from the PASPCR Meeting in Vancouver in 2010, we will be able to award the travel grants in amount of \$800. Depending on the funds, Dr. Leachman may also consider waving the registration fees for the awardees.

The applications for travel awards should be sent to the Secretary/Treasurer. Only applications fulfilling the above considerations with abstracts selected for either oral or poster presentations by regular review committees will be considered. The applications will be ranked based on priority scores obtained after reviews of abstracts and applicant's status with priority for student/postdoc/trainee vs junior faculty.

Although our Society has a comparatively good membership with a total of 105 members (decrease from 113 last year) including 19 students/fellows, 75 regular members, 2 joint SMR members, 4 IFPCS members and 5 honorary members, I ask past members of the Society to renew the membership in PASPCR for 2012. I am acknowledging here the donation \$100 from our honorary member Dr. Seymour Pomerantz towards our Society.

I also ask everybody to recruit new members. The dues have remained the same since 2008 and we offer very attractive rate for students, postdoctoral fellows and other trainees. It is important to grow, because our community is underrepresented in decision making panels at NIH. The reviewers outside of melanin pigmentation field (especially immunologists) usually are critical because of the lack of proper expertise. A strong Society will be able to educate others on the importance of melanin pigmentation in medicine and biology.

We greatly appreciate your support and participation in activities sponsored by the PASPCR and I wish you Happy Passover and Happy Easter!

Andrzej Slominski, M.D., Ph.D.
PASPCR Secretary/Treasurer and Secretary of the IFPCS

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17th PASPCR MEETING – PARK CITY, UT, USA

Dear Pigment Cell Colleagues and Friends,

The next Pan American Society of Pigment Cell Research Annual Meeting will be held in Park City (Deer Valley), Wednesday, September 19th through Saturday, September 22nd, 2012. The Society has received generous support from the St. Regis Deer Valley and All Resort Express to offset housing and transportation costs respectively.

The meeting promises to be not only scientifically and medically valuable, but also fun. On Wednesday afternoon, the meeting will kick off with melanoma and vitiligo patient support meetings, followed by a Keynote speaker and welcome reception at the St. Regis. All attendees are welcome to attend these events. The theme of the meeting will be genetics of pigmentation and melanoma. Thursday and Friday sessions will include animal models of melanoma and pigmentation, genetics and developmental biology, natural selection for pigmentary traits, environmental modulation of pigment phenotypes, translational research presentations and more. Saturday will be devoted to melanoma and will emphasize the role of genetics and genomics in the investigation, prediction and treatment of melanoma. Of course, selected oral abstracts will be chosen to complement the main plenary sessions. A Western Gala will be hosted at Huntsman Cancer Institute on Friday night and there will be opportunities to explore and enjoy the mountains of Park City throughout the meeting.

For more information on registration and abstract deadlines, hotel reservations, program, and recreation opportunities, please see our website at: <http://www.huntsmancancer.org/paspcr2012>.

Best wishes,

Sancy Leachman, M.D., Ph.D.
17th PASPCR Meeting Organizer

For more on what to do and see in Park City visit: <http://www.visitparkcity.com>

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PCMR JOURNAL CORNER

PCMR Journal Recent PubCast

A new pubcast has been recently released for Pigment Cell & Melanoma Research (**PCMR**), the scientific journal associated to IFPCS and SMR:

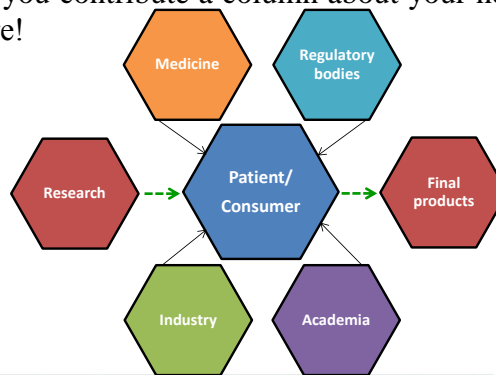
This video refers to the work of Dr. Caroline Le Poole's laboratory (Department of Pathology, Microbiology & Immunology, Oncology Institute, Loyola University, Maywood, IL, USA) on "HSP70i is a critical component of the immune response leading to vitiligo" by Jeffrey A. Mosenson, Andrew Zloza, Jared Klarquist, Allison J. Barfuss, Jose A. Guevara-Patino, I. Caroline Le Poole, Pigment cell & Melanoma Research 25(1), 88-98 (2012).

This Pubcast (video) is available at: <http://www.scivee.tv/node/39524>.

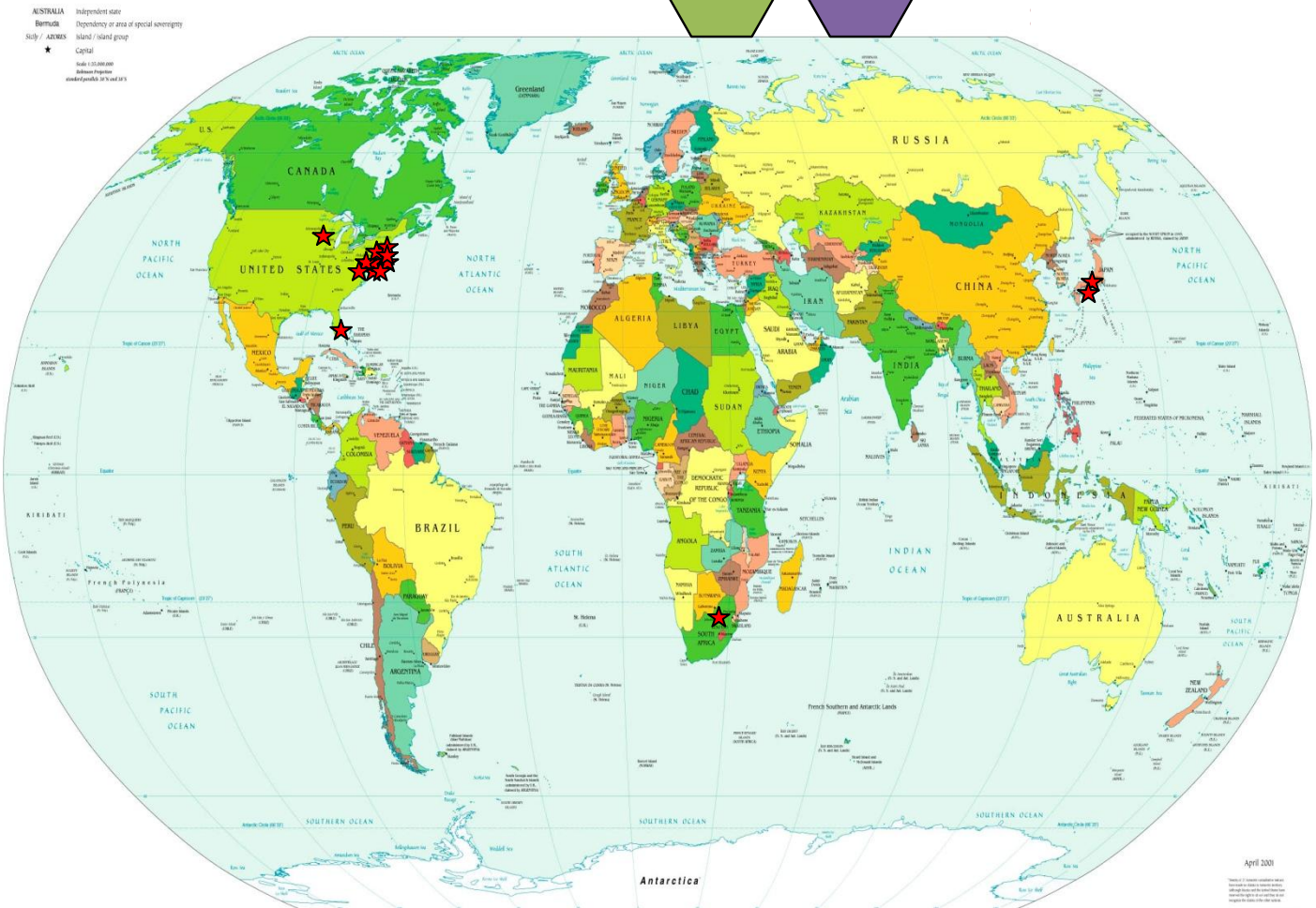
PIGMENTATION COMMUNITY CONNECTIONS

In this issue, we continue the “*Laboratory Updates*” with a column by Dr. Vincent Hearing. We hope that you will be inspired to take the opportunity to fill us in on what is happening in your lab or company. Volunteers would be greatly appreciated, just email us at paspcr.newsletters@gmail.com. As our Society has numerous friends around the world, last year we have transitioned the section “*Let me introduce...*” under the Pigmentation Community Connections. In this number we introduce a new series entitled “*Clinical Insights*” with a column by Dr. David Adams.

This initiative is part of our effort to keep the pigmentation community connected and to emphasize the importance of collaboration and communication between groups. We will keep adding stars on our world map below each time you contribute a column about your newest research projects. So, let’s go on a global research adventure!



Political Map of the World, April 2001



Courtesy: <http://www.mygeo.info/karten/802784.jpg>

LABORATORY UPDATES

by Dr. Vincent Hearing

My Research Group at the National Cancer Institute has been involved for quite some time now in studying the regulation of human skin pigmentation, the impact that has on photoprotection, the mechanisms whereby it is disrupted to cause pigmentary diseases, and how the information we obtain about melanocyte-specific proteins might be used to target melanomas.

As we all know, many factors regulate normal skin pigmentation, including environmental stresses such as UV, and we recently reported that underlying fibroblasts in the dermis play a major role in determining the level of constitutive pigmentation and other characteristics of the overlying epidermis, via their secreted factors. In the case of the hypopigmented epidermis in palmoplantar skin (on the soles of the feet and palms of the hands), this is due to the effects of secreted DKK1 which inhibits Wnt signaling (Yamaguchi et al., 2004) and in the case of different racial/ethnic skin colors it is due, at least in part, to secreted NRG1 which signals through ErbB receptors (Choi et al., 2010b). We recently published invited reviews in the *J Biol Chem* and in *FASEB J* in 2007 and in *BioFactors* in 2009 that are still relatively current on the topic and that summarize our work and those of other groups in the field (Costin and Hearing, 2007; Yamaguchi et al., 2007; Yamaguchi and Hearing, 2009). So let's consider where those 4 topics are heading in my laboratory:

Regulation of Skin Pigmentation – we are continuing our studies on the trafficking of melanosomal proteins and the transport of melanosomes within melanocytes (Valencia et al., 2007; Watabe et al., 2008). We have recently reported gene expression patterns of the skin following exposure to UVA and/or UVB and we are continuing our data mining efforts in those databases to identify important regulators that function during UV responses of the skin (Miyamura et al., 2007; Choi et al., 2010a; Miyamura et al., 2011) and/or following activation of the MC1R by MSH or its inhibition by ASP (Le Pape et al., 2009). We found that UVA- or UVB-induced tans are dramatically different and we are actively trying to identify the mechanism whereby

skin appears 'tanned' following exposure to UVA, yet no new melanin is synthesized (Wolber et al., 2008). One early regulator in the UV response, the transcription factor SOX9, was shown to play an important role in synergizing with MITF to elicit dramatic increases in the melanocytic machinery (Passeron et al., 2007).

Photoprotection – as noted above, while UVB stimulates the expression genes involved in the melanogenic cascade, UVA does not do that yet can produce a comparable tan (Miyamura et al., 2007). The UVA-induced tan provides no photoprotection and we are trying to further study how that occurs (Miyamura et al., 2011). The physiological impact of that is quite significant since commercial tanning parlors are now using UVA-rich lamps to produce tans, which they imply provides protection against subsequent UV exposure/damage, which it clearly does not. To help define the mechanisms involved and to produce a more sensitive and specific assay for melanin production and distribution in human skin, we are generating antibodies against the different types of melanins found in human skin (a preliminary report on that was presented at the recent IPCC). We also have identified a new skin response to UV exposure, termed long-lasting pigmentation (LLP), which can persist in the skin of some subjects for times measured in years (Brenner et al., 2009; Coelho et al., 2009). We are investigating why this occurs, and what cellular mechanisms are involved.

Pigmentary Diseases – Our studies on pigmentary diseases thus far have centered around oculocutaneous albinism (OCA); we reported that the 4 known forms of OCA result from trafficking issues of getting tyrosinase to melanosomes, and thereby melanin production and distribution are disrupted. Although the mechanism whereby mutations in TYR (OCA1) or TYRP1 (OCA3) cause tyrosinase to be captured in the ER and degraded in proteasomes was elucidated a few years ago, it remains unknown how mutations in P (OCA2) and SLC45A2 (OCA4), both expected to be transporters of some type, so effectively disrupt the trafficking of tyrosinase. We are continuing our efforts to determine their functions in collaboration with several groups here at NIH. We are also expanding our horizons somewhat to look at the converse situation, i.e. hyperpigmentary diseases. In collaboration with Beiersdorf, we have developed

models and approaches to use microarray analysis and related techniques to compare nonlesional control skin with hyperpigmented skin, such as occurs in age spots, post-inflammatory hyperpigmentation and of course UV-melanosis. To move beyond the scope of those individual studies, we are establishing a meta-analysis approach to combine all databases we have generated on pigmentary conditions of the skin, to determine common factors that might be significantly involved, but that couldn't be identified in any single database. We are hopeful that this approach will lead to the identification of a number of novel factors that regulate skin pigmentation that were not previously known.

Melanoma Targeting – The identification of SOX9 as an early responder to UV, its effects on the expression of MITF and p21, and its resulting stimulation of differentiation over growth, led us to investigate whether regulation of SOX9 function might be an effective approach to melanoma therapy (Passeron et al., 2009). The results showed great promise and we are trying to make that approach more effective. We also found that the kinase NUA2 is closely involved with increased risk of acral melanomas (Namiki et al., 2011a; Namiki et al., 2011b) and we are continuing our efforts to further detail the mechanism underlying that and how it might potentially be exploited to target melanomas. Finally, the enigmatic Pmel17 – the critical structural component of melanosome structure. Earlier studies by our group and others showed the complicated post-translational processing of Pmel17 that is required to traffic it to early melanosomes, yet >90% of the de novo protein is not targeted to melanosomes (Valencia et al., 2007) and a major question is, what if anything, is the bulk of Pmel17 doing? We hypothesize that it plays another role in skin pigmentation in addition to its structural role in melanosomes and we are looking into that possibility. An active area of interest is also the dual promoter shared by Pmel17 and CDK2, and how the balance in expression between those 2 genes is controlled in melanocytic cells to regulate growth versus differentiation.

The sum of these studies should provide important new insights into the pigmentary system, what goes right and what can go wrong.

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Contact:

Vincent J. Hearing, Ph.D.
Laboratory of Cell Biology
National Institutes of Health
Phone: 301-496-1564
E-mail: hearing@nih.gov

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INTRODUCING CLINICAL INSIGHTS

by Dr. Gertrude-Emilia Costin

In this issue, we introduce a new section under the Pigmentation Community Connections called “**Clinical Insights**”. This series aims to add a medical perspective with particular emphasis on global efforts towards safe and efficacious treatments for various pigmentary diseases and consumer cosmetic needs. This section debuts with a column written by Dr. David Adams who is a pediatrician and biochemical geneticist at the National Human Genome Research Institute. Dr. Adams studies rare inborn errors of metabolism and other rare genetic syndromes to

understand the disease process and identify potential treatments. Dr. Adams’ research interests include the biology of pigment-related disorders, lysosomal storage disorders and the use of exomic and genomic sequencing techniques applied to rare and new diseases.

CLINICAL INSIGHTS

by Dr. David Adams

I completed an MD PhD program at the University of Washington in 2000 before traveling across the country to the University of Maryland for a pediatrics residency. I had worked at a genome sequencing center during graduate school, right in the middle of the Human Genome Project. Even at the time of starting a genetics residency at the NIH in 2004, it seemed hard to imagine that genomic medicine would move forward as quickly as it has! During my genetics training, I worked in the lab of the NHGRI Clinical Director, Dr. William Gahl. He had been working on Hermansky Pudlak and Chediak Higashi disease before I arrived, and with the retirement of Dr. Richard King, there seemed to be an opportunity to work on oculocutaneous albinism using some of the resources available in the Gahl lab and the NIH intramural program. Coincidentally, we started the NIH Undiagnosed Diseases Program, an entirely separate project, which has been fun and hard and incredibly time consuming. I find that with the study of unknown diseases and pigmentation, I have finally found some sort of confluence for my background in bench research, genomics and medicine. In a way, every person who has OCA or other pigmentation disorder, and who does not have a definitive diagnosis, becomes their own medical mystery and an opportunity to combine cell biology and genomic laboratory techniques to find an answer.

Albinism is an inherited medical condition affecting approximately one in 20,000 persons worldwide. It is an important cause of inherited low vision. One study found that albinism was responsible for 15% of childhood-onset legal blindness [1]. Our laboratory, run by Dr. William Gahl, studies both syndromic, e.g. Hermansky-Pudlak Syndrome, and non-syndromic forms of albinism. My work, in

collaboration with Dr. Brian Brooks and other colleagues in the National Eye Institute and NIH Clinical Center, includes investigations into the diagnosis, natural history and potential therapy of isolated oculocutaneous albinism (OCA). (ClinicalTrials.gov identifier 00808106).

The diagnosis of OCA is based principally on clinically ascertained, consistent eye findings such as altered foveal development and iris transillumination. A molecular diagnosis is not required for treatment beyond genetic counseling, and is frequently not obtained in a clinical setting. Four genes are known to produce OCA: TYR (coding for tyrosinase, EC 1.14.18.1), OCA2, TYRP1 and SLC45A2. Each gene produces OCA in an autosomal recessive inheritance pattern. Among individuals who produce little to no eye, hair and skin pigment, two correctly-phased, disease-causing mutations are detected 80 to 85% of the time. Individuals with reduced, but not absent, pigmentation are less likely to produce a definitive molecular diagnosis. A variety of hypotheses have arisen regarding the “missing mutations” in OCA, but none have provided a definitive answer to the problem [2]. Examinations of gene promoter regions and assays designed to detect large deletions have found only occasional examples. It has long been expected that additional genes might cause OCA. We are beginning to use next-generation DNA sequencing techniques, e.g. exome sequencing, to look for variant in additional gene candidates. In addition, our OCA natural history study participants contribute a variety of specimens including a skin biopsy and blood for DNA isolation. Subsequent preparation of dermal melanocyte cultures is allowing us to correlate genetic background with the expression of TYR and other genes involved in melanogenesis.

The natural history information we learn from our study participants is applied to both direct clinical and basic biology study aims. A clinical problem in OCA is that affected persons and their families are often given minimal advice as to the prognosis and management of their condition. From the traditional medical perspective, OCA is considered to be a static condition with a variable visual phenotype. Therefore, simple advice about UV exposure and standard ophthalmologic follow-up are considered adequate treatments. Strategies actually employed by people with albinism, however, are highly variable. Some

parents use minimal intervention for their affected children, focusing on allowing them to fit in with their normally-sighted peers. Other parents teach their children Braille, use canes and guide dogs, and consider enrolling their children in schools for the blind. Except for a few children with relatively good visual acuity, there is poor correlation between parental practice and visual ability. Similarly, there are no practice standards for dermatologic follow-up of hypopigmented skin. Qualitative reviews of parent/patient experiences, plus objective data from careful ophthalmologic phenotyping, are being used to lay the groundwork for rational treatment recommendations.

Despite being considered a static condition, it is well known that vision for a person with OCA can change dramatically during the first few years of life. Visual plasticity and the extent of post-natal visual development is of great interest to us as a fundamental understanding needed to design rational treatment. Foveal hypoplasia is the fundamental cause of low vision in OCA. Once the fovea has stopped developing, there are likely to be significant limitations in the degree to which any treatment could be expected to influence long term visual outcome. We include young children in our natural history study and perform careful visual phenotyping in an attempt to understand the pattern and duration of visual development after birth. Such phenotyping remains challenging as the available tools for measuring visual acuity in young children are imperfect. Optical coherence tomography is an example of a technique that we, and others, are evaluating as a means of capturing quantitative or semi-quantitative data about foveal structural changes in infants over time.

Improving the lives of patients is the ultimate goal of medical research, and we are constantly searching for new avenues toward effective treatment. Mild improvement in vision has been reported with eye surgery for nystagmus [3], but essentially all current OCA treatment options are supportive. Work within the OCA field continues to search for better treatments, and a study underway at the University of Minnesota is evaluating whether the administration of L-DOPA will improve vision based on work suggesting that L-DOPA insufficiency may alter visual development [4]. (ClinicalTrials.gov identifier NCT01176435).

We recently collaborated on a paper published by the Brooks laboratory showing that the FDA approved drug NTBC (2-(2-nitro-4-fluoromethylbenzoyl)-1,3-cyclohexanedione or “Nitisinone”) improves skin and eye pigmentation both in a mouse model of OCA type 1B and cultured human melanocytes from a study participant with OCA type 1B [5]. NTBC is currently licensed for use in the disorder Hereditary Tyrosinemia, type I, a defect in the fumarylacetoacetate hydrolase (FAH, EC 3.7.1.2) enzyme. In Tyrosinemia type I, NTBC serves to block a proximal enzyme in the tyrosine catabolism pathway (4-Hydroxyphenylpyruvate dioxygenase, HPPD, EC 1.13.11.27) and prevents the accumulation of toxic intermediates caused by the distal FAH insufficiency. In our case, we are using a low dose of NTBC to take advantage of a side effect, namely the inhibition of tyrosine catabolism and resulting increase in plasma tyrosine. Low dose NTBC administration can increase plasma tyrosine levels into the millimolar range [6]. We hypothesize that, in our mice and cultured melanocytes, the high-concentration tyrosine helps to keep defective tyrosinase enzyme close to its Vmax by providing excess substrate and/or by acting as a chaperone that improves protein folding. We are writing a proof of principle clinical protocol for a small number of adult participants, and are in the process of refining our outcome measures. We plan to start recruiting later this year.

Albinism is an uncommon/rare inherited disease with important, lifelong health manifestation. We hope that by addressing basic-biological and clinical aspects of OCA we can make contributions to improve the lives of our patients and all people with OCA.

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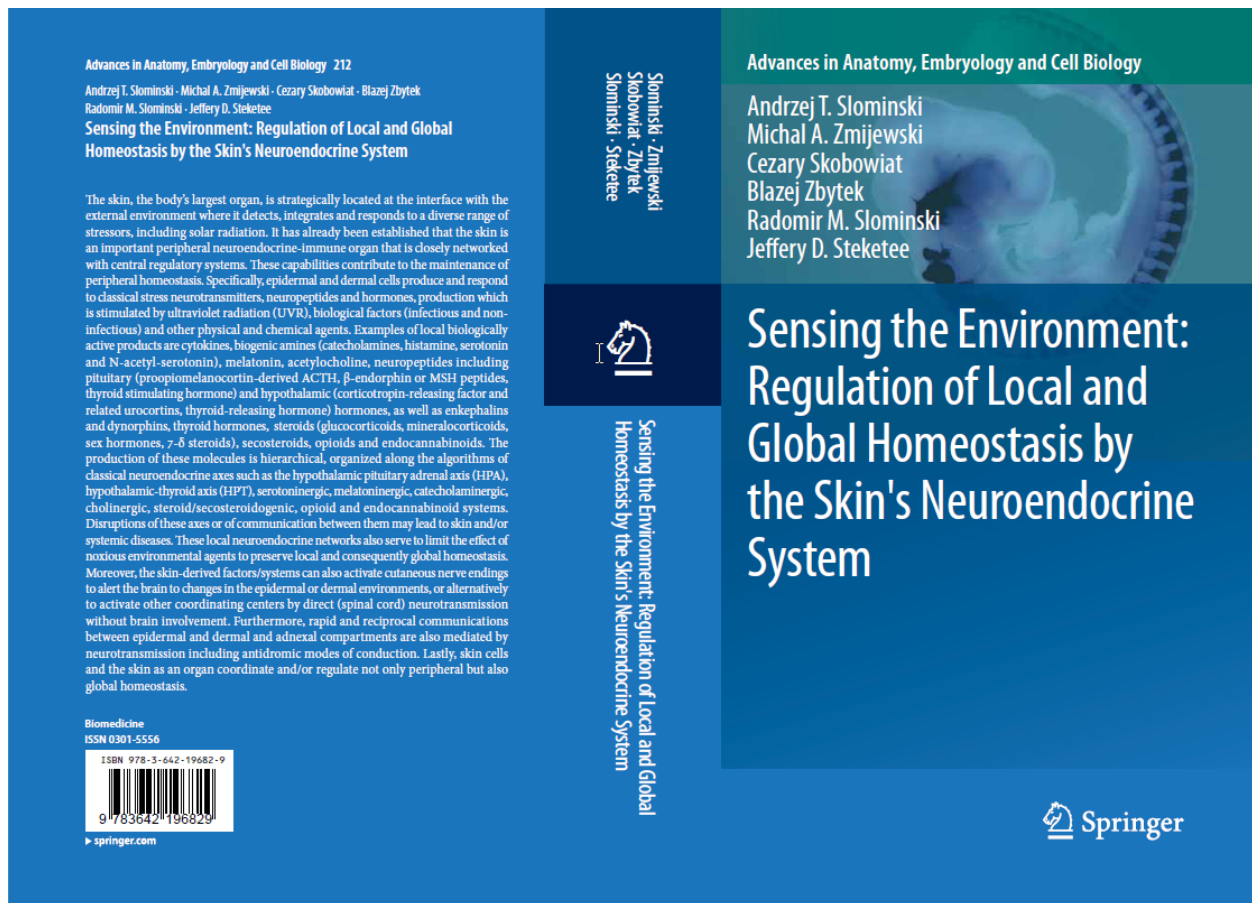
Contact:

E-mail: dadams1@mail.nih.gov

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MEMBERS IN THE NEWS

Several PASPCR members – **Drs. Andrzej T. Slominski, Cezary Skobowiat, Blazej Zbytek, and Radomir M. Slominski** – have recently authored the book titled “Sensing the Environment: Regulation of Local and Global Homeostasis by the Skin’s Neuroendocrine System”, with Dr. Michal A. Zmijewski and Dr. Jeffery D. Stekete. The book has been recently published by Springer-Verlag and it presents the most recent research establishing the skin as an important peripheral neuroendocrine organ, tightly linked to central axes of stress. Furthermore, the book details research results on the response of the epidermal cells to ultraviolet radiation and other biological factors.



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PASPCR member **Dr. James Grichnik** joins Dr. Harold S. Rabinovitz as Director of the University of Miami’s Miller School of Medicine “Dermatology Close-Up 2012: Melanoma and Other Neoplasms of the Skin” Course to be held on April 21st-22nd, 2012 at the Alexander All-Suite Oceanfront Beach Hotel in Miami Beach, FL. The course brings together thought-leaders in dermatology to discuss current methods of diagnosing skin cancers and a comprehensive review of both dermoscopy and reflectance confocal microscopy.

More info: <https://cmetracker.net/UMIACME/Files/Brochures/104192.pdf>

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POSITIONS WANTED/AVAILABLE

Postings for **Positions Wanted** will be open only to members of the PanAmerican Society for Pigment Cell Research (PASPCR) or its sister Societies (ASPCR, JSPCR and ESPCR). Postings for **Positions Available** will be open to all individuals and institutions so long as the position is related to pigment cell research. Please send postings to Dr. William Oetting at oetti001@umn.edu.

The postings will remain on the **Positions Wanted and Available** section of the PASPCR Newsletter and on the web page for 1 year, unless other arrangements are made. Please provide an expiration date for any submitted posting if less than 1 year. Final decisions will be made by the Publications Committee of the PASPCR.

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2012 PASPCR MEMBERSHIP LIST

Dear PASPCR members,
Thank you for supporting our Society and paying your dues in time!

Andrzej Slominski

The PASPCR Membership List is published in the April number of the PASPCR Newsletter. However, the membership is updated continuously and the names and addresses of new members and any changes in members' contacts are published during the year in the remaining two issues. Therefore, please inform the Secretary/Treasurer of any changes in your contact info that happen during the year so we could communicate them to the members through the Newsletter.

Abdel-Malek, Zalfa A. Ph.D.
University of Cincinnati
Department of Dermatology
231 Albert Sabin Way
Cincinnati, OH 45267-0592 U.S.A.
Phone: 513-558-6246
Fax: 513-558-0198
E-mail: abdelmza@uc.edu
Regular member

Ahmad, Nihal Ph.D.
University of Wisconsin
Department of Dermatology
1300 University Avenue, MSC 423
Madison, WI 53706 U.S.A.
Phone: 608-263-5359
Fax: 608-263-5523
E-mail: nahmad@wisc.edu
Regular member

Anderson, Michael G. Ph.D.
The University of Iowa
Bowen Science Bldg. 6-430
51 Newton Road, 6-430 BSB
Iowa City, IA 52242 U.S.A.
Phone: 313-335-7839
Fax: 313-335-7730
E-mail: michael-g-anderson@uiowa.edu
Regular member

Arnheiter, Heinz Ph.D.
NINDS/NIH
Bldg. 35, Room 2A201
35 Convent Drive MSC 3706
Bethesda, MD 20892 U.S.A.
Phone: 301-496-1645
Fax: 301-482-7686
E-mail: Ha3p@nih.gov
Regular member

Bagnara, Joseph T. Ph.D.
University of Arizona
Department of Cell Biology and Anatomy
1501 N. Campbell
Tucson, AZ 85724 U.S.A.
Phone: 520-621-7516
Fax: 520-621-9339
E-mail: bagnara@u.arizona.edu
Honorary member

Barsh, Greg S. M.D., Ph.D.
HudsonAlpha Institute for Biotechnology
601 Genome Way
Huntsville, AL 35806 U.S.A.
Phone: 650-723-5061
E-mail: gbarsh@hudsonalpha.org
Regular member

Belitsky, Jason M. Ph.D.
Oberlin College
Department of Chemistry and Biochemistry
119 Woodland Street
Oberlin, OH 44074 U.S.A.
Phone: 440-775-8303
Fax: 440-775-6682
E-mail: jason.Belitsky@Oberlin.edu
Regular member

Bhawan, Jag M.D.
Boston University School of Medicine
Department of Dermatology
609 Albany Street, Room J 309
Boston, MA 02118 U.S.A.
Phone: 617-638-5570
Fax: 617-638-5575
E-mail: jbhawan@bu.edu
Regular member

Bieniek, Radoslaw M.D.
GI Pathology
7661 Spirit Lake CV
Cordova, TN 38016 U.S.A.
Phone: 781-467-8632
Fax: 901-448-6979
E-mail: r.bieniek@d-path.com
Regular member

Bonte, Frederic Ph.D.
LVMH Recherche
185 Avenue De Verdun
St. Jean De Braye 45804 FRANCE
Phone: 33-0238-60-3388
Fax: 33-0238-60-3117
E-mail: fredericbonte@research.lvmh-pc.com
Regular member

Bosko, Carol Ph.D.
Unilever Research
Department of Skin Bioscience
40 Merritt Blvd.
Trumbull, CT 06410 U.S.A.
Phone: 203-381-5727
Fax: 203-381-5476
E-mail: carol.bosko@unilever.com
Regular member

Bowers, Roger R. Ph.D.
California State University, Los Angeles
Department of Biology
5151 State University Drive
Biological Sciences Building 333
Los Angeles, CA 90032 U.S.A.
Phone: 323-343-2081
Fax: 323-343-6451
E-mail: rbowers@calstatela.edu
Honorary member

Carlson, Andrew J. M.D.
Albany Medical College, MC-81
Department of Pathology
47 New Scotland Avenue; ML-81
Albany, NY 12208 U.S.A.
Phone: 518-262-6414
Fax: 518-262-6251
E-mail: carlsoa@mail.amc.edu
Regular member

Chen, Suzie Ph.D.
Rutgers University
The State University of New Jersey
Ernest Mario School of Pharmacy
Susan Lehman Cullman
Laboratory for Cancer Research
164 Frelinhuysen Road
Piscataway, NJ 08854 U.S.A.
Phone: 732-445-3400 (x227)
Fax: 732-445-0687
E-mail: suziec@rci.rutgers.edu
Regular member

Cheng, Tsing Ph.D.
New York University Medical Center
Department of Dermatology
Smilow Research Center, Room 401
New York, NY 10016 U.S.A.
Phone: 212-263-9077
E-mail: tsing.cheng@nyumc.org
Student member

Chintala, Sreenivasulu Ph.D.
Roswell Park Cancer Institute
Department of Cancer Biology
Elm and Carlton Street
Buffalo, NY 14263 U.S.A.
Phone: 716-845-3109
Fax: 716-845-4928
E-mail: sreenivasulu.chintala@roswellpark.org
Student member

Cornell, Robert A. Ph.D.
University of Iowa
Carver College of Medicine
Department of Anatomy and Cell Biology
1-400-D BSB/51 Newton Road
Iowa City, IA 52242 U.S.A.
Phone: 319-335-8908
Fax: 319-335-7198
E-mail: robert-cornell@uiowa.edu
Regular member

Costin, Gertrude-Emilia Ph.D., M.B.A.
Institute for In Vitro Sciences, Inc.
30 W Watkins Mill Road #100
Gaithersburg, MD 20878 U.S.A.

Phone: 301-947-6524
Fax: 301-947-6538
E-mail: ecostin@iivs.org
Regular member

de la Serna, Ivana L. Ph.D.
University of Toledo College of Medicine
Department of Biochemistry
and Cancer Biology
Mail Stop #1010, Health Science Campus
3000 Arlington Avenue
Toledo, OH 43614 U.S.A.
Phone: 419-383-4111
Fax: 419-383-6228
E-mail: ivana.delaserna@utoledo.edu
Regular member

Dellinger, Ryan W. Ph.D.
University of California, Irvine
Department of Medicine
101 The City Drive, Bldg. 23, Room 436B
Orange, CA 92868 U.S.A.
Phone: 714-456-7220
Fax: 714-456-8551
E-mail: rdelling@uci.edu
Regular member

Diwakar, Ganesh Ph.D.
Alticor Inc.
BIO-ASSAY, Room 50/2D
7575 Fulton St. East
Ada, MI 49355 U.S.A.
Phone: 616-787-5992
Fax: 616-787-4466
E-mail: ganesh.diwakar@amway.com
Regular member

D'Orazio, John A. M.D., Ph.D.
University of Kentucky College of Medicine
Pediatrics, Markey Cancer Center
Combs Research Building
800 Rose Street
Lexington, KY 40536 U.S.A.
Phone: 859-323-6238
Fax: 859-257-8940
E-mail: jdorazio@uky.edu
Regular member

Epstein, Howard A. Ph.D.
EMD Chemicals
Product Development
480 South Democrat Road
Gibbstown, NJ 08027 U.S.A.
Phone: 856-599-6625
E-mail: howard.epstein@emdchemicals.com
Regular member

Erf, Gisela F. Ph.D.
University of Arkansas
Center of Excellence for Poultry Science O-410
1260 W. Maple Street
Fayetteville, AR 72701 U.S.A.
Phone: 479-575-8664
Fax: 479-575-7139
E-mail: gferf@uark.edu
Regular member

Ganesan, Anand K. M.D., Ph.D.
University of California, Irvine
Department of Dermatology and Biological Chemistry
1324 Sprague Hall
Irvine, CA 92697-2400 U.S.A.
Phone: 949-824-2926
Fax: 949-924-7454
E-mail: aganesan@uci.edu
Regular member

Grando, Sergei A. M.D., Ph.D., D.Sc.
University of California, Irvine
Department of Dermatology
134 Sprague Hall, Zot 40406
Irvine, CA 92697 U.S.A.
Phone: 949-824-2713
Fax: 949-824-2993
E-mail: sgrando@uci.edu
Regular member

Grichnik, James M. M.D., Ph.D.
UMHC/Sylvester Comprehensive Cancer Center
Biomedical Research Building
1501 N.W. 10th Avenue, Room 912, Loc. D-1
Miami, FL 33136 U.S.A.
Phone: 305-243-6045
Fax: 305-243-6072
E-mail: grichnik@miami.edu
Regular member

Grimes, Pearl E. M.D.
The Vitiligo and Pigmentation
Institute of Southern California
5670 Wildshire Blvd., Suite 650
Los Angeles, CA 90036 U.S.A.
Phone: 323-467-4389
Fax: 323-467-4488
E-mail: pegrimesMD@aol.com
Regular member

Grim, Elizabeth Ph.D.
University of Texas
MD Anderson Cancer Center
Experimental Therapeutics
1515 Holcombe Blvd., Unit 362
Houston, TX 77030 U.S.A.
Phone: 713-792-3667

Fax: 713-792-2070
E-mail: egrimm@mdanderson.org
Regular member

Gunn, Teresa M. Ph.D.
McLaughlin Research Institute
1520 23rd Street South
Great Falls, MT 59405 U.S.A.
Phone: 406-454-6033
Fax: 406-454-6019
E-mail: tmg@mri.montana.edu
Regular member

Guo, Huazhang M.D., Ph.D.
300 S. Central Ave. #c39
Hartsdale, NY 10530 U.S.A.
E-mail: huazhang.guo@gmail.com
Student member

Hakozaki, Tomohiro Ph.D.
The Procter and Gamble Company
Beauty Technology Division
11810 East Miami River Road
Cincinnati OH 45252 U.S.A.
Phone: 513-627-1105
Fax: 513-627-0319
E-mail: hakozaki.t.1@pg.com
Regular member

Halaban, Ruth Ph.D.
Yale Medical School
Department of Dermatology
P.O. Box 208059
333 Cedar Street
New Haven, CT 06520-8059 U.S.A.
Phone: 203-785-4352
Fax: 203-785-7637
E-mail: ruth.halaban@yale.edu
Regular member

Hearing, Vincent J. Ph.D.
NCI, National Institutes of Health
Laboratory of Cell Biology
Building 37, Room 2132
9000 Rockville Pike
Bethesda, MD 20892 U.S.A.
Phone: 301-496-1564
Fax: 301-402-8787
E-mail: hearingv@nih.gov
Regular member

Herlyn, Meenhard D.V.M.
The Wistar Institute
Program of Molecular and Cellular Oncogenesis
3601 Spruce Street
Philadelphia, PA 19104-4268 U.S.A.
Phone: 215-898-3950

Fax: 215-898-0980
E-mail: herlynm@wistar.org
Regular member

Hornyak, Thomas J. M.D., Ph.D.
University of Maryland
School of Medicine
Department of Dermatology & Biochemistry
and Molecular Biology
108 N. Greene St., Room 308A
Baltimore, MD 21201 U.S.A.
Phone: 410-706-3725
E-mail: Thornyak@som.umaryland.edu
Regular member

Hou, Ling Ph.D.
Wenzhou Medical College
Developmental Cell Biology
and Disease Program
270 Xueyuan Road
Wenzhou, Zhejiang 325003 CHINA
Phone: 301-402-2036
E-mail: lhou88@gmail.com
Lhou88@yahoo.cn
Regular member

Huizing, Marjan Ph.D.
National Institutes of Health
NHGRI
10 Center Drive, Bldg. 10, Room 10C-103
Bethesda, MD 20892-1851 U.S.A.
Phone: 301-402-2797
Fax: 301-480-7825
E-mail: mhuizing@mail.nih.gov
Regular member

Indra, Arup K. Ph.D.
Oregon State University
College of Pharmacy
Department of Pharmaceutical Sciences
203 Pharmacy Building
1601 SW Jefferson Avenue
Corvallis, OR 97333 U.S.A.
Phone: 541-737-5775
Fax: 541-737-3999
E-mail: arup.indra@oregonstate.edu
Regular member

Ito, Shosuke Ph.D.
Fujita Health University
School of Health Sciences
Toyoake Aichi 470-1192 JAPAN
Phone: 81-562-93-2595
Fax: 81-562-93-4595
E-mail: sito@fujita-hu.ac.jp
Regular member

Jablonski, Nina G. Ph.D.
Pennsylvania State University
Department of Anthropology
409 Carpenter Building
University Park, PA 16802 U.S.A.
Phone: 814-867-0004
E-mail: ngj2@psu.edu
Regular member

Janjetovic, Zorica Ph.D.
University of Tennessee HSC
Department of Pathology & Laboratory Medicine
19S Manassas Ave., Room 258
Memphis TN 38153 U.S.A.
and
120 Sawyer Circle, Apt. 394
Memphis, TN 38103 U.S.A.
Phone: 901-448-6341
Fax: 901-448-6979
E-mail: zjanjeto@uthsc.edu
Student member

Kadekaro, Ana Luisa Ph.D.
University of Cincinnati
Department of Dermatology
231 Albert Sabin Way, Room 7409
PO Box 670592
Cincinnati, OH 45267-0592 U.S.A.
Phone: 513-558-6659
Fax: 513-558-0198
E-mail: kadekaal@uc.edu
Regular member

Kaelin, Christopher B. Ph.D.
HudsonAlpha Institute of Biotechnology
and Stanford University
Department of Genetics
B275 BM333 Always Buildingeckman Center
279 Campus Drive
Stanford, CA 94305 U.S.A.
Phone: 650-954-3993
Fax: 650-723-1399
E-mail: kaelin@stanford.edu
Regular member

Kim, Tae-Kang Ph.D.
University of Tennessee
Department of Pathology & Laboratory Medicine
19S Manassas Ave.
Memphis, TN 38163 U.S.A.
Phone: 901-448-6341
Fax: 901-448-3745
E-mail: tkim10@uthsc.edu
Student member

King, Richard A. M.D., Ph.D.
University of Minnesota

MMC 485 Mayo
420 Delaware Street, S.E.
Minneapolis, MN 55455 U.S.A.
Phone: 612-624-6657
Fax: 612-624-6645
E-mail: kingx002@umn.edu
Honorary member

Kobayashi, Nobuhiko M.D., Ph.D.
Nara Medical University School of Medicine
Department of Dermatology
Kashihara Nara 634-8522 JAPAN
Phone: 81-744-29-8891
Fax: 81-744-25-8511
E-mail: nobuk2@naramed-u.ac.jp
Regular member

Kovacic, Diane M.D.
University of Tennessee HSC
Department of Pathology
930 Madison Avenue, Suite 500
Memphis, TN 38163 U.S.A.
Phone: 201-723-4094
E-mail: dkovacic@optoline.net
Student member

Kubic, Jeniffer D. Ph.D.
University of Chicago
Department of Dermatology
5841 South Maryland Avenue
MC 5067 L518B
Chicago, IL 60637 U.S.A.
Phone: 773-702-6486
E-mail: jkubic@uchicago.edu
Student member

Lamoreux, M. Lynn Ph.D.
8255 Sandy Point Road
Bryan, TX 77807 U.S.A.
E-mail: mllamoreux@hotmail.com
Honorary member

Lang, Deborah Ph.D.
University of Chicago
Department of Dermatology
5841 South Maryland Avenue MC 5067 L518B
Chicago, IL 60637 U.S.A.
Phone: 773-702-6005
Fax: 773-702-8398
E-mail: dlang@medicine.bsd.uchicago.edu
Regular member

Le Poole, I. Caroline Ph.D.
Loyola University Medical Center
Department of Pathology
Cancer Center, Bldg. 112, Room 203
2160 South First Avenue

Maywood, IL 60153 U.S.A.

Phone: 708-327-2032

Fax: 708-327-3238

E-mail: ilepool@lumc.edu

Regular member

Leachman, Sancy A. M.D., Ph.D.

University of Utah, Huntsman Cancer Institute

Department of Dermatology

2000 Circle of Hope, Suite 5242

Salt Lake City, UT 84112 U.S.A.

Phone: 801-585-1810

Fax: 801-585-7477

E-mail: sancy.leachman@hci.utah.edu

Regular member

Lee, Michael M.D.

University of Maryland

125 South High Street

Baltimore, MD 21202 U.S.A.

Phone: 617-543-2476

E-mail: mlee4@umm.edu

Student member

Lee, Tim Ph.D.

BC Cancer Research Centre

Cancer Control Research Program

675 West 10th Avenue

Vancouver BC V5Z1L3 CANADA

Phone: 604-675-8053

Fax: 604-675-8180

E-mail: tlee@bccrc.ca

Regular member

Leming, Philip D. M.D.

Cincinnati Hematology-Oncology, Inc.

Department of Hematology / Oncology

7745 Hartford Hill Ln

Cincinnati, OH 45242 U.S.A.

Phone: 513-321-4333

Fax: 513-533-6033

E-mail: pianoblues@aol.com

Regular member

Lin, Connie B. Ph.D.

Johnson & Johnson CPPW

Skin Research Center

199 Grandview Road

Skillman, NJ 08558 U.S.A.

Phone: 908-874-1532

Fax: 908-874-1254

E-mail: blin1@its.jnj.com

Regular member

Lister, James A. Ph.D.

Virginia Commonwealth University

Department of Human Genetics

Sanger Hall 11-014

1101 E. Marshall Street

Richmond, VA 23298-0033 U.S.A.

Phone: 804-628-4518

Fax: 804-827-1124

E-mail: jalister@vcu.edu

Regular member

Liu, Feng Ph.D.

University of California, Irvine

Department of Medicine

101 The City Drive

Building 23, Room 436

Orange, CA 92868 U.S.A.

Phone: 714-456-8551

E-mail: liufe@uci.edu

Regular member

Loftus, Stacie K. Ph.D.

National Institutes of Health

National Human Genome Research Institute

Genetic Disease Research Branch

49 Convent Dr., Bldg. 49, Room 4A66

Bethesda, MD 20892-4472 U.S.A.

Phone: 301-594-1752

Fax: 301-402-2170

E-mail: sloftus@mail.nih.gov

Regular member

Loy, Chong J. Ph.D.

Johnson and Johnson Co.

51 Science Park Road, Aries #01-01

Singapore Science Park 2

Singapore 117586 SINGAPORE

Phone: 656-720-6307

Fax: 656-464-1382

E-mail: cloy@its.jnj.com

Regular member

Manga, Prashiela Ph.D.

New York University School of Medicine

Department of Dermatology

Smilow Research Center

522 First Avenue, Room 401

New York, NY 10016 U.S.A.

Phone: 212-263-9086

Fax: 212-263-5819

E-mail: prashiela.manga@nyumc.org

Regular member

Marks, Michael Ph.D.

University of Pennsylvania School of Medicine

Department of Pathology & Laboratory of Medicine

513 Stella-Chance Labs, 422 Curie Blvd./6100

Philadelphia, PA 19104-6100 U.S.A.

Phone: 215-898-3204

Fax: 215-573-4345

E-mail: marksm@mail.med.upenn.edu

Regular member

Mascarenhas, Joseph B. D. Ph.D.
University of Chicago
Department of Medicine,
Div. Dermatology
5841 South Maryland Avenue
Chicago, IL 60637 U.S.A.
Phone: 773-702-6486
E-mail: jmascare@medicine.bsd.uchicago.edu
Student member

Meadows, Gary G. Ph.D.
Washington State University
College of Pharmacy, Cancer Prevention
and Research Center
Box 646510
Pullman, WA 99164-6713 U.S.A.
Phone: 509-335-4753
Fax: 509-335-7559
E-mail: meadows@wsu.edu
Regular member

Menon, Gopinathan Ph.D.
Ashland Specialty Ingredients
Global R&D
Wayne, NJ 07470 U.S.A.
Phone: 973-872-4343
Fax: 973-628-3886
E-mail: gmenon@ashland.com
Regular member

Meyskens, Frank L. Jr. M.D.
University of California
Comprehensive Cancer Center
101 The City Drive S.
Building 56, Room 209
Orange, CA 92868 U.S.A.
Phone: 714-456-6310
Fax: 714-456-2240
E-mail: flmeyske@uci.edu
Regular member

Mintz, Beatrice Ph.D.
Fox Chase Cancer Center
333 Cottman Avenue
Philadelphia, PA 19111 U.S.A.
Phone: 215-728-2479
Fax: 215-728-3574
E-mail: beatrice.mintz@fcc.edu
Regular member

Morrison, Randall L. Ph.D.
McDaniel College
Department of Biology
2 College Hill
Westminster, MD 21157 U.S.A.
Phone: 410-857-2409
Fax: 410-386-4613

E-mail: rmorriso@mcDaniel.edu
Regular member

Nejati, Reza M. M.D.
University of Tennessee Health Science Center
Department of Pathology & Laboratory Medicine
930 Madison Ave.
Memphis, TN 38163 U.S.A.
Fax: 901-448-6979
E-mail: mnejati@uthsc.edu
Student member

Nordlund, James J. M.D.
1156 Riverside
Cincinnati, OH 45202 U.S.A.
Phone: 513-871-4168
Fax: 513-871-6214
E-mail: jjnordlund@fuse.net
Regular member

Oetting, William S. Ph.D.
University of Minnesota
Department of Medicine - Genetics
MMC 485; 4-12 Moos Tower
515 Delaware Street S.E.
Minneapolis, MN 55455-0392 U.S.A.
Phone: 612-624-1139
Fax: 612-624-6645
E-mail: oetti001@umn.edu
Regular member

Orlow, Seth J. M.D., Ph.D.
New York University Medical Center
Department of Dermatology
560 First Avenue, Room H-100
New York, NY 10016 U.S.A.
Phone: 212-263-5245
Fax: 212-263-8752
E-mail: seth.orlow@nyumc.org
Regular member

Panich, Uraivan, Ph.D.
Faculty of Medicine Siriraj Hospital,
Mahidol University
Department of Pharmacology
2 Prannok Rd.
Bangkoknoi, Bangkok 10700 THAILAND
Phone: 081-815-5925
Fax: 024-195-7576
E-mail: siuks@mahidol.ac.th
Regular member

Pavan, William J. Ph.D.
National Institutes of Health
National Center for Human Genome Research
Building 49, Room 4A82
9000 Rockville Pike

Bethesda, MD 20892-4472 U.S.A.
Phone: 301-496-7584
Fax: 301-402-2170
E-mail: bpavan@nhgri.nih.gov
Regular member

Pavicevic, Zoran M.D.
University of Tennessee
Health Science Center
Department of Neurosurgery
120 Sawyer Circle Apt. 394
Memphis, TN 38103 U.S.A.
Phone: 901-652-7374
Fax: 901-448-8468
E-mail: zpavice1@uthsc.edu
Student member

Pawelek, John M. Ph.D.
Yale Medical School
Department of Dermatology
333 Cedar Street
New Haven, CT 06520-8059 U.S.A.
Phone: 203-687-7318
Fax: 203-785-7637
E-mail: john.pawelek@yale.edu
Regular member

Pinczewski, Joel M.D., Ph.D.
University of Maryland
Department of Pathology
413 Woodhill Drive
Owings Mill, MD 21117 U.S.A.
Phone: 443-204-2847
E-mail: jp123ok@yahoo.com
Student member

Pomerantz, Seymour H. Ph.D.
59 Sereni Street, Apt. 8
Rehovot 76240 ISRAEL
Phone: 011-972-8-931-6541
Fax: 011-972-8-931-6541
E-mail: spomerantz@bezeqint.net
Honorary member

Potterf, S. Brian Ph.D.
Unilever R&D, Trumbull
Clinical Research
50 Commerce Drive
Trumbull, CT 06611 U.S.A.
Phone: 203-381-5795
Fax: 203-381-5790
E-mail: brian.potterf@unilever.com
Regular member

Qu, Di Ph.D.
Amway Corporation
Research and Development, 50-1B
7575 Fulton Street East

Ada, MI 49355-0001 U.S.A.
Phone: 616-787-4403
Fax: 616-787-4445
E-mail: di_qu@amway.com
Regular member

Reiter, Russel J. Ph.D.
University of Texas Health Science Center
Department of Cellular and Structural Biology
7703 Floyd Curl Drive
San Antonio, TX 78229 U.S.A.
Phone: 210-567-3859
Fax: 201-567-3803
E-mail: reiter@uthscsa.edu
Regular member

Rhodes, Arthur R. M.D., M.P.H.
Rush University Medical Center
Department of Dermatology
1653 W. Congress Pkwy
Annex Building Suite 220
Chicago, IL 60612 U.S.A.
Phone: 312-942-6096
Fax: 312-942-7778
E-mail: arthur_rhodes@rush.edu
Regular member

Ronai, Ze'ev Ph.D.
Sanford-Burnham Medical
Research Institute
10901 N. Torrey Pines Road
La Jolla CA 92037 U.S.A.
Phone: 858-646-3185
Fax: 815-366-8003
E-mail: ronai@sanfordburnham.org
Regular member

Scott, Glynis M.D.
University of Rochester
Dermatology / Pathology
601 Elmwood Avenue, Box 697
Rochester, NY 14642 U.S.A.
Phone: 585-275-8811
Fax: 585-273-1346
E-mail: glynis_scott@urmc.rochester.edu
Regular member

Seiberg, Miri Ph.D.
Johnson & Johnson CPPW
Skin Research Center
168 Herrontown Rd.
Princeton, NJ 08540 U.S.A.
Phone: 908-874-2325
Fax: 908-874-2323
E-mail: mnseiberg@gmail.com
Regular member

Setaluri, Vijayaradhi Ph.D.
University of Wisconsin
Department of Dermatology
1300 University Avenue, B25
Madison, WI 53706 U.S.A.
Phone: 608-263-5362
Fax: 608-263-5223
E-mail: setaluri@wisc.edu
Regular member

Silva de Castro, Caio C. M.D.
Pontificia Universidade Catolica do Parana
Department of Dermatology
Padre Anchieta
1846 cj 1014
Curitiba-Parana 80730-000 BRAZIL
Phone: 41-3566-2036
Fax: 41-3027-3186
E-mail: caio_castro@yahoo.com.br
Regular member

Skoboviat, Cezary Ph.D.
University of Tennessee Health Science Center
Department of Pathology & Laboratory Medicine
19S Manassas Avenue, Room 258
Memphis, TN 38163 U.S.A.
Phone: 901-448-6341
Fax: 901-448-6979
E-mail: cskobowi@uthsc.edu
Student member

Slominski, Andrzej T. M.D., Ph.D.
University of Tennessee
Department of Pathology and Laboratory Medicine
930 Madison Avenue, Room 525 (Clinical Office)
Memphis, TN 38163 U.S.A.
Phone: 901-448-3741
Fax: 901-448-6979
E-mail : aslominski@uthsc.edu
Regular member

Slominski, Radomir M.
Jagielonian University
9385 Old Plantation Cove
Germantown, TN 38139 U.S.A.
Phone: 901-758-6979
E-mail: radomir.slominski@gmail.com
Student member

Spritz, Richard A. M.D.
University of Colorado
School of Medicine
Anschutz Medical Campus
12800 E. 19th Avenue, MS 8300
Aurora, CO 80045 U.S.A.
Phone: 303-724-3107
Fax: 303-724-3100

E-mail: richard.spritz@ucdenver.edu
Regular member

Sugumaran, Manickam Ph.D.
University of Massachusetts at Boston
Department of Biology
100 Marrissey Boulevard
Boston, MA 02421 U.S.A.
Phone: 617-287-6598
Fax: 617-287-6650
E-mail: manickam.sugumaran@umb.edu
Regular member

Swope, Viki B. D.V.M.
University of Cincinnati
Dermatology Department
231 Albert Sabin Way
Cincinnati, OH 45267 U.S.A.
Phone: 513-558-3940
E-mail: viki.swope@uc.edu
Regular member

Tada, Akihiro Ph.D.
POLA Research & Development Laboratories
560 Kashio-cho, Totsuka-ku
Yokohama 244-0812 JAPAN
Phone: 81-45-826-7232
E-mail: akihiro@pola.co.jp
Regular member

Van Otterloo, Eric B.S.
University of Iowa
Department of Anatomy and Cell Biology
2270 Holiday Rd. #512
Iowa City, IA 52241 U.S.A.
Phone: 319-335-7725
Fax: 319-335-7198
E-mail: eric-vanotterloo@uiowa.edu
Student member

von Koschimbahr, Anne M. B.A.
University of Cincinnati
Dermatology Department
231 Albert Sabin Way
Cincinnati, OH 45267 U.S.A.
Phone: 513-558-6242
Fax: 513-558-0198
E-mail: vonkosam@mail.uc.edu
Student member

Van Raamskonk, Catherine D. Ph.D.
University of British Columbia
Department of Medical Genetics
2350 Health Science Center Mall
Vancouver BC V6T123 CANADA
Phone: 604-827-4224
Fax: 604-822-5348

E-mail: cvr@mail.ubc.ca
Regular member

Waugh, Jacob M. M.D.
Revance Therapeutics Inc.
7555 Gateway Blvd.
Newark, CA 94560 U.S.A.
Phone: 510-742-3405
Fax: 510-662-4816
E-mail: jwaugh@revance.com
Regular member

Xe, Lifang Ph.D.
University of California
UCI Cancer Center
Bldg. 23, Room 436
Orange, CA 92868 U.S.A.
Phone: 714-456-7242
Fax: 714-456-8551
E-mail: lifangx@uci.edu
Student member

Zbytek, Blazej M.D., Ph.D.
University of Tennessee
Department of Pathology &
Laboratory Medicine
930 Madison Ave.
Memphis, TN 38163 U.S.A.
Phone: 901-448-6173
Fax: 901-448-6979
E-mail: bzbytek@yahoo.com
bzbytek@uthsc.edu
Student member

Zhang, Jiong, M.D., Ph.D.
University of Tennessee HSC
Department of Pathology &
Laboratory Medicine
930 Madison Ave., 5th Floor
Memphis, TN 38163 U.S.A.
Phone: 901-448-0530
E-mail: pathdoc.zhang@gmail.com
Student member

Zhou, Youwen, M.D., Ph.D.
University of British Columbia
Department of Dermatology & Skin Science
835 W. 10th Avenue
Vancouver, BC V5Z4E8 CANADA
Phone: 604-875-4747
Fax: 604-873-9919
E-mail: Youwen.Zhou@vch.ca
Regular member

Yang, Sun Pharm.D., Ph.D., R.Ph.
University of California Irvine
Department of Pharmaceutical Sciences

Chao Family Cancer CTR
B200 Sprague Hall
Irvine, CA 92697 U.S.A.
Phone: 649-824-9321
E-mail: syang2@uci.edu
Regular member