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, spearheaded 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 all our contributors for their columns submitted to us for inclusion in the letters.

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The PanAmerican Society for Pigment Cell Research

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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

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

The PASPCR Newsletter is published three times a year (April, August and December) by the PanAmerican Society for Pigment Cell Research. All views are those of the authors. For further information or to submit articles, please use the e-mail address paspcr.newsletters@gmail.com.

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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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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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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 pigmentation 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!

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 pigmented 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 NUAK2 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.

**References:**


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INTRODUCING 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. Coincidently, 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

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 pigimentary 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.
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 oculoctuaneous 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.

References:

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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. Steketee. 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.

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
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.

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.
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