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 18th PASPCR Meeting, spear-headed by Dr. Vijayasaradhi Setaluri, are progressing well. The meeting will be held in Madison, Wisconsin, from September 8th to September 11th, 2013. Further information on the meeting can be found on page 6 of this newsletter.

In this issue, we continue the "***Laboratory Updates***" section with a column by Dr. John D'Orazio, and the "***Let me Introduce…***" section with a column by Dr. Marco d'Ischia. We also continue the "***Clinical Insights***" section with a column by Dr. Stanca Birlea and Dr. David Norris. Back in this number is the section "**Hot off the Presses**" that hosts columns by Drs. Feng Liu and Tatiana Krasieva.

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. 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 in our Calendar of Events.

Also, keep us updated on any "***Members in the News***" so we can spread the word of your successes.

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.

PASPCR Newsletter Editorial Team

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The PASCR Web-Site can be found at:
http://www.paspcr.org

The PanAmerican Society for Pigment Cell Research

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CALENDAR OF EVENTS

2013
The International Investigative Dermatology (IID) Meeting
Date and place: May 8-11, Edinburgh, Scotland, UK
Web-site: http://www.iid2013.org/welcome/

2013
The 18th ESPCR Meeting
Date and place: September 9-12, Lisbon, PORTUGAL
Web-site: http://www.espcr.org/ESPCR2013/

2013
The Pigment Cell Development Workshop
Date and place: May 6-8, Edinburgh, UK
Web-site: http://devbio.hgu.mrc.ac.uk

2013
The 1st IID Satellite Symposium on Dermatoendocrinology
Date and place: May 7, Edinburgh, UK
Web-site: http://iid2013.org/scientific_program/

2013
The ASPCR-ASDR Meeting
Date and place: May 17-19, Sydney, AUSTRALIA
Web-site: http://www.aspcr.org/

2013
The 18th PASPCR Meeting
Date and place: September 8-11, Madison, WI, USA
Web-site: www.paspcr.org/

2013
The Annual Meeting of American Society for Cell Biology
Date and place: December 14-18, New Orleans, LA, USA
Web-site: http://www.ascb.org/

2014
The 22nd IPCC
Date and place: September 4-7, Singapore, SINGAPORE
Web-site: http://www.ipcc2014.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.

Publication Committee

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 2012. 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 17th PASPCR Meeting made through PASPCR as follows (in alphabetical order):

All Resort Transportation

Department of Dermatology at the University of Utah

Huntsman Cancer Institute at the University of Utah

Johnson & Johnson Consumer Companies (Aaron B. Lerner Lecture)

Merck

Morphotek

Myriad Genetics

Prometheus

St. Regis Hotel

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PASPCR PRESIDENT'S CORNER

Spring is here. Or, at least it was, a few weeks ago. Seduced by a couple of warm weekends in early March, and held captive by the school calendar of a 15 year-old daughter, earlier this week we joined the yearly migration that is something of a tradition for Tennessee valley residents, driving 300 miles South to the Gulf Coast, for what was billed as a relaxing time at "the beach". Pigmentary variation was everywhere, from pastel colors of the houses and the sunsets to fluorescent greens and blues of the great egrets' breeding colors. But the ambient temperature was a balmy 45 degrees (Fahrenheit), and no amount of sunscreen or Jimmy Buffett music could ameliorate the sense of communal betrayal…what happened to Spring?

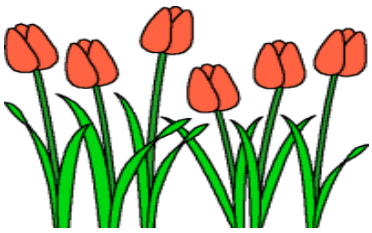
A sense of betrayal also underlies several recent science-related, societal, and personal events, and I will take this newsletter opportunity to share my reaction to some of those experiences, interspersed with a few bright notes. First, from the perspective of a citizen scientist, the only thing worse than the current sorry state of US science funding is the sorrier state of our US politicians, who are so dysfunctional that they barely managed a continuing resolution to keep the doors open—at the post office, the Washington monument, and, just barely, the NIH—earlier this week. What should we do? (a) Wait until 2014, and hope that the House turns over; (b) Demand that our elected representatives (regardless of party affiliation) stop seeing compromise as a dirty word; (c) Apply for postdoc positions in Switzerland. Thirty years ago, the third option might not have been in jest, but today, I will embrace both (a) and (b), and encourage Society members to do the same. Bright note: in the midst of 7% pay

lines and mandated budget reductions for funded grants, our own Vijay Setaluri has managed to get a terrific score for his R13 application to support our own meeting this Fall. Glückwünsche, Vijay!

Our 2013 meeting in Madison, Wisconsin, also provides an appropriate segue for another cloud with silver linings. One of several topics considered by the organizing committee over the last few months has to do with how, when, and why the abstracts and the proceedings of the meetings will be publicized, distributed, and archived. This is a perfect opportunity for collaboration between a scientific society and a science publisher, but science publishing is also a business, which sets an unfortunate stage for a potential betrayal of motivation. Bright note: the relationship between the IFPCS and Wiley has served both parties well, but will only continue to do so as long as the interests of the Societies are represented by a strong and thoughtful leader. As a member of the IFPCS council, I have been party (and occasionally contributed) to extensive discussions over the last few months in which the interests of the Societies are ably represented by President Picardo. Continuate così nobile, Mauro!

I will close on a personal and sad note…the unexpected loss of a dear friend earlier this year. David Cox, 66 years old, died suddenly from apparent heart disease on January 21, 2013. Dave and I followed somewhat similar paths, entranced by the awesome power of genetics, never sure whether we were physicians or scientists, and sharing a love for music, nature, and our families, who are related by choice and by spirit. Betrayed in this case by time (not enough of it), Dave's passing was a reminder that there are bright notes everywhere; we just need to look. Dave's passion for genetics and medicine is easy to see in an informal interview that is part of the Cold Spring Harbor Oral History collection: http://library.cshl.edu/oralhistory/interview/genome-research/involvement-genomics/cox-involvement-in-genomics/.

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



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LETTER FROM THE PASPCR SECRETARY/TREASURER

Dear PASPCR members,

Preparations for the XVIIIth PASPCR Conference in Madison, Wisconsin are under way under outstanding leadership by Dr. Vijay Setaluri and members of the Organizing Committees. In my opinion the program will attract many investigators from different fields. The key note speaker is Dr. Hector F. De Luca who contributed enormously to the field of vitamin D. His name was submitted at least twice to be considered by Nobel Prize Committee. Therefore, we would like to congratulate Vijay for inviting Dr. De Luca to this important conference. The team in Wisconsin is very well organized, and is already securing the proper outside financial support. The conference grant submitted to the NIH

got an outstanding score and will likely be funded. I also obtained a note from Johnson & Johnson that the application for the grant to support A.B. Lerner lectureship will be awarded. Therefore, please reserve following dates: September 8-11 to attend our meeting.

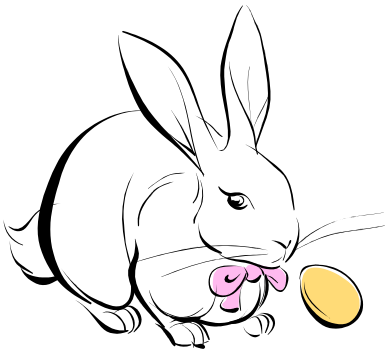
Concerning travel awards, there are two conflicting opinions. One presented by me is that the applicant should be a member of the PASPCR in good standing when submitting the application. This is the continuation of the previous policy realized since the meeting in Memphis. Second, voiced by the President and President-elect is that non-members can also apply for the travel grant on the same conditions as the members in good standing. These issues will be discussed and decided by the PASPCR Council shortly. I believe, however, that the priority in awarding grants to student members of the Society and requirement of being a first and presenting author will be maintained. If you have any opinion on this matter you can voice it prior the decisions are made.

I am also pleased to announce that financially we are very strong, which is good news for relatively small Society, and therefore, we are not changing the dues which remain the same since the Memphis meeting. Also, we subsidize students, since their dues only partially cover costs of electronic subscription. If anybody experiences problems accessing the journal (it has to be done through the IFPC website) or did not receive the printed journal for which she or he has paid, please let me know. I will refer such matter to the IFPC.

I would also like to provide some statistics on the membership. The total number of PASPCR members in good standing is 93, which includes 19 student members and 2 members of Japanese and 1 member of the European Societies. This is approximately 20-25% decrease in membership. Therefore, please recruit more investigators into the Society or ask previous members to renew. The bigger we are the stronger we are and the higher our impact on science and NIH funding is.

I thank you again for your support of our Society and I wish you Happy Easter and Happy Passover!

Andrzej Slominski, MD, PhD
Professor of Pathology and Medicine
Secretary/Treasurer of the PASCPR and Secretary of the IFPCS



18th PASPCR MEETING – MADISON, WI, USA

18th Meeting of the PanAmerican Society for Pigment Cell Research (PASPCR)

September 8-11, 2013
University of Wisconsin-Madison
Madison, WI, USA

Advances in Melanocyte and Melanoma Biology

Symposia Topics

Cell & Molecular Biology of Normal & Malignant Melanocytes

UV light: Role in Melanin Pigmentation and Melanoma

Melanocyte Development & Melanoma Cancer Stem Cells

Immune Responses to Normal & Malignant Melanocytes

Bench-to-Bedside & Back

Keynote Speaker

Hector F. DeLuca, Ph.D., University of Wisconsin

For more information on PASPCR, please visit our website at:

http://paspcr.med.umn.edu/

The conference site is currently under construction, please check back at a later date for additional information.

National Committee:

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

Andrzej Slominski, M.D., Ph.D.
Secretary/Treasurer

Caroline Le Poole, Ph.D.
President-elect

Hasan Mukhtar. Ph.D.
Member

Local Organizing Committee:

Vijayasaradhi Setaluri, Ph.D.
Chair

Mark Albertini, M.D.
Co-Chair

Nihal Ahmad, Ph.D.

Murray Brilliant, Ph.D.

B. Jack Longley, M.D.

Vladimir Spiegelman, M.D., Ph.D.

WISCONSIN
UNIVERSITY OF WISCONSIN-MADISON

DEPARTMENT OF DERMATOLOGY

Carbone Cancer Center

Dear Pigment Cell Colleagues and Friends,

The 18th Annual Meeting of the PASPCR will be held September 8-11, 2013 on the campus of University of Wisconsin-Madison, Madison, WI with active support and sponsorship of the UW Department of Dermatology and the UW Carbone Comprehensive Cancer Center.

The overarching theme of this year's meeting is **"Advances in Melanocyte and Melanoma Biology".** Since the molecular cloning of mammalian tyrosinase over 25 years ago and identification of MITF, the master regulator of melanocyte two decades ago, the field of pigment cell biology has experienced an explosion of knowledge and continues to attract many talented researchers with a wide range of expertise. This year's meeting is designed to bring together senior and young researchers to highlight the latest advances in in 5 selected areas 1) basic cell & molecular biology, 2) melanocyte development, 3) UV induced pigmentary changes, 4) immune response to melanocyte antigens and 5) opportunities and challenges in translating these advances to benefit patients with pigmentary disorders with special emphasis on vitiligo and cutaneous melanoma.

The keynote lecture will be delivered by Dr. Hector DeLuca, a pioneer in Vitamin D research. The list of invited speakers include: Dr. David Fisher, Dr. Ze'ev Ronai, Dr. Ruth Halaban, Dr. Jeff Sosman, Dr. Elsa Quintana, Dr. Jose Guvera-Patiño, and Dr. Catherine van Raamsdonk.

Objectives of this year's program are to:

- Review the latest developments in basic and clinical investigations in pigmentary biology and disease
- Identify areas for future collaborative and interdisciplinary research that focus on improving human health with regard to melanoma, vitiligo and other pigmentary disorders
- Facilitate collaborative relationships among researchers with diverse expertise
- Identify the opportunities and address the challenges in translating the basic discoveries to benefit patients with pigmentation diseases such as cutaneous melanoma and vitiligo

Distinctive features of this year's program include:

- A dedicated session to highlight advances and current challenges in bench-to-bedside translation
- Opportunities for young investigators to meet with experts in an informal and exclusive setting to discuss and understand opportunities and challenges in pigment cell research
- Special travel grants to support young investigators
- Publication of the program, abstracts, and meeting report online and/or by Pigment Cell and Melanoma
- Research

The University of Wisconsin-Madison was founded in 1848, and has long been recognized as one of America's great universities. Located near the Wisconsin state capitol building, its campus spans 936 acres along the southern shore of Lake Mendota. A public, land-grant institution, UW-Madison was ranked 4th among all higher education institutions in the United States for research and development in 2011.

The Fluno Center, the venue of the meeting is centrally located on the UW-Madison campus and offers superb views of the surrounding university and city. The Fluno Center combines collaborative classroom technology with the ambiance of a comfortable, relaxed residential setting. The interior captures the simplicity and elegance of the Arts and Crafts era, featuring prairie style furnishings and original lithographs of designs by Frank Lloyd Wright, an American architect with strong roots in the Madison area, recognized as "the greatest American architect of all time."

The local organizing committee looks forward to welcome you to Madison and your active participation in the conference.

Vijayasaradhi Setaluri, PhD
Professor of Dermatology, University of Wisconsin-Madison

Local organizing committee:

Vijayasaradhi Setaluri, PhD, (Chair) Professor of Dermatology, University of Wisconsin-Madison
Mark Albertini, MD, (Co-chair) Associate Professor of Medicine, University of Wisconsin-Madison
Murray Brilliant, PhD, Senior Scientist, Marshfield Clinic Research Foundation
Nihal Ahmad, PhD, Professor of Dermatology, University of Wisconsin-Madison
Vladimir Spiegelman, MD, PhD, Professor of Dermatology, University of Wisconsin-Madison
B. Jack Longley, MD, Professor of Dermatology, University of Wisconsin-Madison

KEY DATES:

Abstract Submission & Early Registration: **July 1, 2013**

For further details, please visit: **www.PASPCR.org**

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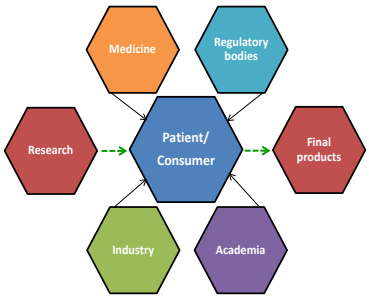
PCMR JOURNAL CORNER

PCMR Journal Recent PubCast

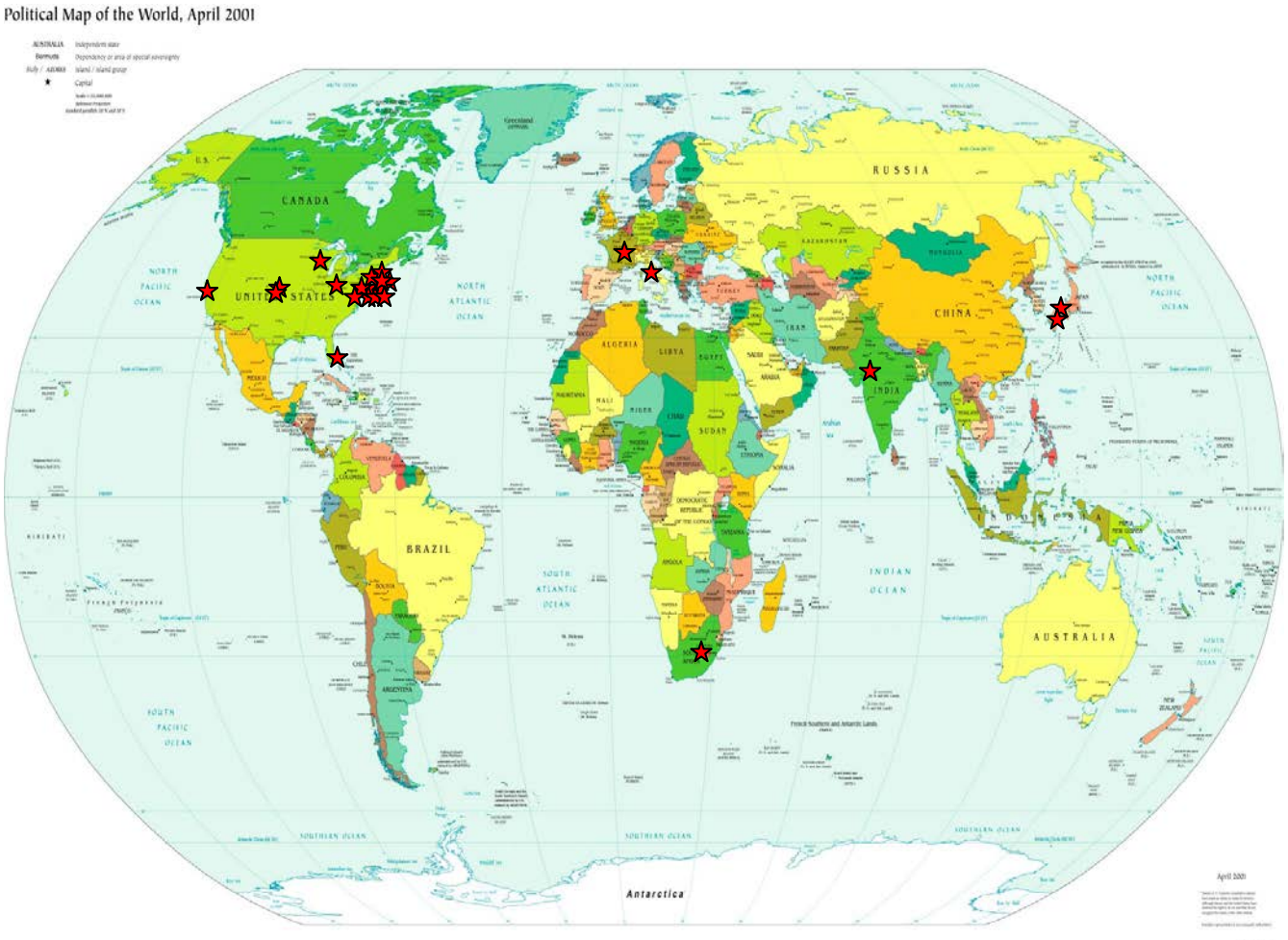
A new PubCast has been recently released for Pigment Cell & Melanoma Research (**PCMR**), the scientific journal associated to IFPCS and SMR. This video refers to the work of Dr. Arup K. Indra's Laboratory (Department of Pharmaceutical Sciences, Oregon State University, Corvallis, OR, USA) on "Endothelin-1 is a transcriptional target of p53 in epidermal keratinocytes and regulates ultraviolet-induced melanocyte homeostasis" by Stephen Hyter, Daniel J. Coleman, Gitali Ganguli-Indra, Gary F. Merrill, Steven Ma, Masashi Yanagisawa, Arup K. Indra – Pigment Cell & Melanoma Research 26(2), 247-258 (2013). This PubCast (video) is available at: http://www.scivee.tv/journalnode/56891.

PIGMENTATION COMMUNITY CONNECTIONS



In this issue, we continue the "***Laboratory Updates***" with a column by Dr. John D'Orazio, the "***Let me Introduce…***" section with a column by Dr. Marco D'Ischia, and the "***Clinical Insights***" with a column by Dr. Stanca Birlea and Dr. David Norris. 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.

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!



LABORATORY UPDATES

by Dr. John D'Orazio

Over 70,000 people are now diagnosed and 9,000 die from melanoma in the United States annually. Despite the wonderful advances in targeted therapy and immunotherapy realized in melanoma treatment over the last several years, the long-term prognosis for patients with advanced-stage melanoma remains pessimistic. Prompted by these dire statistics, my lab at the University of Kentucky Markey Cancer Center focuses on how skin interacts with the ultraviolet radiation found in sunlight. Our overall goal is to develop safe approaches to prevent melanoma development in high-risk populations and to minimize the deleterious consequences of UV exposure to the skin. Of particular interest to us is the melanocortin-1 receptor (MC1R) allele, which has emerged as a major genetic risk factor of melanoma and UV sensitivity. The MC1R gene encodes a G_s-protein coupled receptor expressed on the surface of the melanocytes that relays survival and differentiation signals to melanocytes upon interaction with its cognate ligand alpha melanocyte stimulating hormone (α-MSH) through activation of adenylyl cyclase and generation of the second messenger cAMP. The MC1R gene is highly polymorphic with variant alleles linked with red hair, fair skin, poor UV tanning, and elevated melanoma risk (Valverde, Healy et al. 1996; Abdel-Malek, Suzuki et al. 1999; Landi, Bauer et al. 2006). MC1R activation after UV exposure results in adaptive melanization (tanning) to provide enhanced protection against cutaneous UV penetration and damage. Defective MC1R signaling results in inadequate melanization of the skin, which allows UV-induced DNA changes to accumulate that can lead to mutations and carcinogenesis. In humans, loss-of-function MC1R polymorphisms are associated with UV sensitivity and a melanoma-prone phenotype. When MC1R signaling is defective, melanocytes preferentially produce pheomelanin rather than eumelanin species. Pheomelanin is a much poorer blocker of UV photons and may actually contribute to oxidative damage and mutagenesis, as has recently been suggested by data from Devarati Mitra and coworkers in the laboratory of my mentor, David Fisher (Mitra, Luo et al. 2012).

In addition to its role in skin melanization, MC1R may influence melanoma development by non-pigmentary pathways as well (Robinson, Dixon et al. 2010). Building on the work of many laboratories, we are attempting to determine molecular links between MC1R signaling and genome maintenance pathways in melanocytes. The last decade has seen compelling evidence of MC1R-cAMP-mediated signaling in promoting the early repair response to UV-induced DNA damage (Hauser, Kadekaro et al. 2006; Song, Mosby et al. 2009; Kadekaro, Chen et al. 2012; Wong, Ainger et al. 2012). Such a DNA repair function for MC1R was initially suggested by the observation that RHC variants had a reduced ability to remove UV-induced photolesions independent of the cellular melanin content. We and others have observed an impaired UV-induced DNA damage response in the setting of defective MC1R function. Of particular interest, MC1R agonists and cAMP stimulants enhance melanocytic nucleotide excision repair (NER), a major pathway responsible for removing UV-induced DNA damage and protecting cells from UV-induced malignant degeneration. Pharmacologic manipulation of the MC1R signaling pathway therefore holds great promise as a melanoma-preventive strategy. Determining the molecular mechanisms linking MC1R signaling and NER function remains a critically unanswered question, one of which our lab is currently investigating. My postdoctoral work in David Fisher's lab found that topical application of the adenylate cylase activating drug forskolin restored melanotic pigmentation in an animal model of the MC1R-defective fair-skinned human and that this "sunless tanning" was potently protective against UV damage and carcinogenesis in the skin (D'Orazio, Nobuhisa et al. 2006). We rely on this animal model to study

the many effects of cAMP stimulation in the skin, including a robust acceleration of the skin's ability to clear mutagenic UV photolesions.

Another area of interest for the lab is determining the contribution of epidermal thickness in aiding protection against UV-associated carcinogenesis. Recently, we utilized a humanized skin mouse model that maintains interfollicular epidermal melanocytes, and identified that activation of adenylyl cyclase was associated with expansion of epidermal thickness irrespective of melanization or the presence of epidermal melanocytes. Interestingly, cAMP-directed epidermal thickening was mediated through increased keratinocyte proliferation, possibly through secreted factor(s) including KGF from cutaneous fibroblasts. Even in the absence of pigmentation, the observed epidermal thickening significantly diminished the amount of UV-A and UV-B that passed through whole skin and reduced the amount of UV-B-associated epidermal sunburn cells, suggesting that this strategy might afford some UV protection even in amelanotic (albino) individuals (Scott, Christian et al. 2012).

My lab is privileged to be part of an exciting and dynamic "MC1R community", which together has provided new insights into the mechanistic relationships between UV exposure and skin cancer. However, the overall picture of MC1R's influence on carcinogenesis is not yet complete, and we expect that future studies of the MC1R-cAMP pathway may yet hold more surprises. In particular, we feel that pharmacologic manipulation of cAMP levels in the skin (Abdel-Malek, Ruwe et al. 2009; Passeron, Namiki et al. 2009; Abdel-Malek 2010; Kadekaro, Chen et al. 2012) represents a promising and novel approach to alter UV sensitivity and cancer risk in MC1R-deficient and melanoma susceptible individuals.

John D'Orazio's lab members (L to R):
John D'Orazio, Alexandra Amaro-Ortiz, Perry Christian, Christina Wicker and Stuart Jarrett.

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LET ME INTRODUCE…

by Dr. Marco d'Ischia

The EuMelaNet Group: Promoting Interdisciplinary and Translational Research on Melanins and Melanogenesis.

Melanins to the fore in biomedical and materials sciences

No doubt, pigment cells are endowed with unique and fascinating properties which make them an unrivalled research field to make new exciting discoveries and foster new projects that bring together investigators from diverse disciplines to study important biomedical problems. There is likewise no doubt that much of the pigment cell uniqueness is due to the pigment itself as a most distinguishing trait that has attracted the attention of scientists and clinicians since time immemorial. However, if we take a retrospective look at the way pigment cell research has evolved throughout the past decades, we will notice that the focus has been gradually shifted off from the pigment. Whereas early meetings and symposia used to feature scientific programs largely devoted to melanin structure, properties and biological functions, as rooted in the pathway of melanogenesis, in more recent years these themes have been somewhat abandoned, leaving a number of fundamental issues still unsettled. Paradoxically, an opposite trend has been apparent outside the pigment cell community, where melanins and

melanogenesis have been increasingly appreciated as a source of novel research opportunities. The burst of interest in polydopamine as biocompatible, highly adhesive and multifunctional biopolymer is a paradigmatic example of how melanin-related materials may provide remarkable solutions to scientific and technological problems, being amenable to an extraordinary variety of applications. So, why not stimulate the interdisciplinary study of melanins as a source of inspiration for human well-being, from health to technology?

The birth and mission of the EuMelaNet group

Prompted by these considerations, during the ESPCR meeting in Muenster in 2009 a number of colleagues and I decided to take a concrete and effective action not only to promote a renaissance of basic melanin research within our Societies but also, and especially, to raise the attention of scientific communities and industrial companies that operate outside of, or at the interface with, pigment cell biology. Starting from informal discussions, the idea of a special interest group pursuing these goals rapidly materialized into the establishment of EuMelaNet, thanks also to the strong and encouraging support of Lionel Larue, Alain Taïeb, Lluis Montoliu, Mauro Picardo and all the members of the ESPCR Council. The name of the group was selected in order to underscore both its European birthplace and the unexplored potential of eumelanins for application. The stated mission of EuMelaNet is detailed in the website (www.espcr.org/eumelanet/) and is summarized in Figure 1.

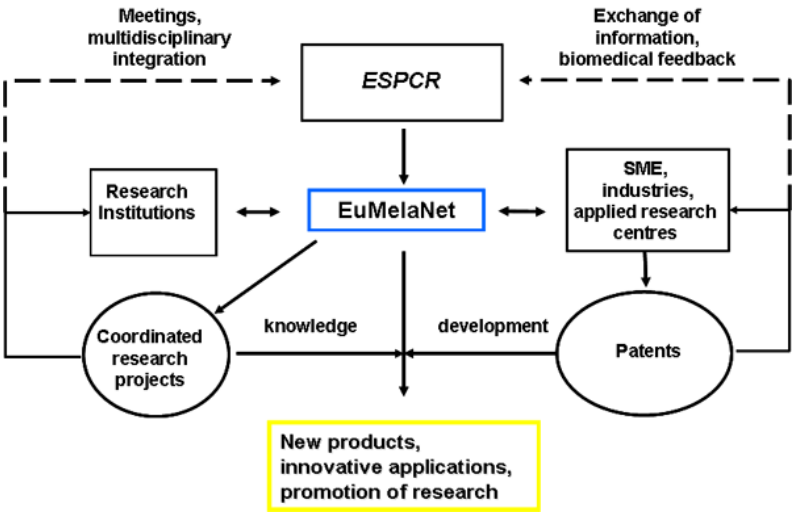

Figure 1

The group seeks to promote basic research on melanins as multifunctional biomaterials; to bridge the gaps between basic and applied research, to 'translate' fundamental research results into applications; to ensure coordination of research and to identify new opportunities to develop new melanin-based technologies; to promote the integration of multidisciplinary research teams aimed at technology-oriented research.

To pursue these goals, several activities will be envisaged which include the organization of meetings and colloquia, standardization of products and methods, supply of chemicals and standards, training courses for industry personnel, and coordination of research programs.

Current activities

The first official meeting of the EuMelaNet Special Interest group took place during the XXI International Pigment Cell Conference held in Bordeaux in 2011, on the occasion of Concurrent Session 5 chaired by Marco d'Ischia, Josè-Carlos Garcìa-Borron and Shosuke Ito. This session was entitled "Methods in Melanin Research" and was formatted as a round table centered around the launching project of the EuMelaNet group, that is, the definition of a set of recommended protocols and procedures to be followed for studies of melanins and melanogenesis. Eventually it was agreed that the presentations debated at CS5 were worthy of further assessment and elaboration by the group to generate a consensus paper on standardization of methods, which should be directed to guide pigment cell researchers into the experimental details of melanin-relating protocols with an easy-to-do approach for non-chemists. A draft of the paper is now ready and at the point of being submitted.

In line with the general mission of promoting melanin research at interdisciplinary levels, members of the EuMelaNet special interest group and their colleagues have been actively participating in various meetings directed to technological and biomedical applications.

Future perspectives

What's next? Certainly, as mentioned before, one of the most promising applications of melanin-type materials is in the field of organoelectronics. The integration of biopolymers into hybrid electronics is one of the emerging issues in view of the realization of fully bio-compatible devices. A highly attractive field of application of melanins is also in relation to the development of biological interfaces, a most intriguing area of research encompassing biology, biotechnology, diagnostics, and medicine, and involving (bio)chemists, physical chemists, applied physicists, analytical chemists and bioengineers. The current quest for biocompatible and biodegradable materials with advanced mechanical and electrical capabilities would prompt exploitation of melanins as the centerpiece for medical devices, for example in electronically active absorbable implants. Photoprotection and radioprotection are also important areas where melanins may be fruitfully utilized. In this rapidly changing scenario of interdisciplinary and multidisciplinary research, the EuMelaNet group may play an important role as a liaison between scientific communities and companies, in providing connections, the basic know-how and guidelines to applied scientists.

The way toward a full appreciation of melanins' potential for applications in biomedicine and technology is still long. It is hoped however that the commitment of the EuMelaNet members, with the encouraging support of the ESPCR and sister Societies like the PanAmerican Society, will soon materialize into novel projects of high technological and biomedical impact. Synergism with other scientific and technologically-oriented communities will be very important. Pursuit of the EuMelaNet mission will be useful not only for the advancement of technology but also for a reappraisal of the biological roles and functions of melanins.

We therefore invite all interested members of the Pigment Cell Societies to join EuMelaNet, exchange views and ideas, promote new initiatives, and contribute to exploit the immense potential of melanins as safe functional biomaterials for technology and health.

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CLINICAL INSIGHTS

by Dr. Stanca Birlea, M.D., Ph.D. and Dr. David Norris, M.D.

IDIOPATHIC GUTTATE HYPOMELANOSYS - A COMMON ESTHETIC PROBLEM

Following previous columns included in the Clinical Insights section of the PASPCR Newsletters, we are sharing our experience and thoughts about a less common pigmentary skin condition with unique profile and relatively limited treatment possibilities: idiopathic guttate hypomelanosis (IGH). While not as explored as other hypomelanotic disorders (i.e., vitiligo), IGH is seen in clinic and requires careful diagnosis and attention to patients, being cosmetically troublesome in dark skin phototypes.

Skin photoaging caused by the effects of cumulative prolonged sun exposure and intrinsic aging is one of the top concerns coming to the attention of dermatologists around the world. It has a polymorphic clinical picture, with progressive evolution, which follows four stages, according to Glogau classification (Ortonne, 2001). The early stages (Glogau I and II) are characterized by a continuous stimulation of pigmentary system, with increase in number of melanocytes and melanin production, and are clinically most often manifested as solar lentigines. With advanced age, dyschromic changes occur (Glogau III), in the form of a mottled appearance; this appearance is the result of the increased, alternating with decreased DOPA-positivity of melanocytes in the chronically sun-exposed skin, and of an irregular distribution of melanosomes within epidermal keratinocytes. In Glogau IV the skin becomes yellow-gray and wrinkled.

IGH is the most common hypomelanotic disorder of photoaged skin and has the following distinct features: a high frequency worldwide, increasing with advanced age, unaesthetic clinical aspect, lack of spontaneous resolution and of therapeutic response, and irreversible evolution after the onset. IGE is characterized by acquired multiple small, discrete, round or oval, porcelain-white 1-5 mm hypopigmented or achromic macules distributed on the sun-exposed surfaces (Figure 1). The most common sun exposed sites are the forearms and legs, the lesions being usually associated with other skin changes of chronological aging and photodamage (wrinkles, solar lentigines, cutaneous xerosis, freckles, actinic keratoses) (Shin et al, 2011). The face, chest and upper back are inexplicably not involved in early IGH. The terminal hairs within the lesions often remain pigmented. Previous reports showed that IGH is most commonly a complaint of middle-aged, light-skinned women, although its presence in dark skin phototypes is not uncommon. IGH is increasingly seen in both sexes; reports showed that, of affected individuals, only 28% were <30 years old, while >70% were of >40 years old (Falabella et al, 1986; Shin et al, 2011). The higher frequency reported in women, is supposedly the result of an increased subjective perception of cosmetic disfigurement in women versus men (Kim et al, 2010).

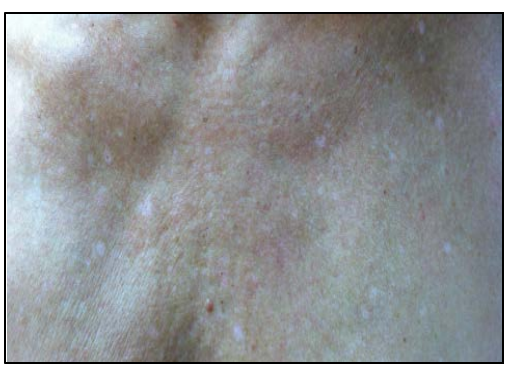

Figure 1. Idiopathic guttate hypomelanosis: small non-scaly porcelain-white macules scattered on the chest.

IGH diagnosis is usually clinical. A Wood light examination could be helpful in revealing suspected cases and the DOPA staining can be useful in showing decreased DOPA-positive melanocytes. In uncertain cases, a skin biopsy can confirm the diagnosis.

Our department has a vast experience with cases of vitiligo and other pigmentary disorders, including IGH. We and others observed in clinical practice that early stages of vitiligo can mimic IGH. In more advanced stages the differences between the two entities can be distinguished on clinical exam: vitiligo patches have well defined borders which enlarge gradually and lack the typical porcelain aspect of IGH. IGH rarely repigment after treatment or spontaneously, and the perifollicular repigmentation pattern present in vitiligo is not observed in IGH. Although a reduced number of melanocytes is present in the IGH epidermis, they are completely eliminated in vitiligo. It is noteworthy that many patients with vitiligo and IGH relate the onset of the depigmentation to sun exposure, even without a typical sunburn. We searched retrospectively the clinical records of a group of 425 new patients who addressed to our dermatology outpatient clinic between March 2012 and March 2013; of them, eleven women (nine African-Americans and two Caucasian, with ages between 43-77 years), had, as one of their concerns, white spots on the shins (with or without the affect of other sun exposed areas), that were diagnosed as IGH. All cases had other associated photodamage changes. In three of them IGH associated vitiligo, the latter showing some improvement with the topical treatments (corticosteroids, calcineurin inhibitors, vitamin D analogs) with or without phototherapy; none of the eleven patients showed improvement of the IGH lesions, most of them choosing only the daily application of a sunscreen.

Other groups reported a variety of treatments, including cryotherapy, superficial dermabrasion, topical steroids, and topical retinoids, and in general described modest therapeutic responses. Minigrafts of normal skin implanted in IGH lesions did not modify the achromic defects, whereas intralesional triamcinolone injections with or without grafts improved the appearance of these lesions (Falabella et al, 1986). IGH lesions clinically improved after 4 months application of topical tretinoin with almost complete recovery of skin glyphics and partial repigmentation, and restoration of skin elasticity (Pagnoni et al, 1999). However, the cosmetic outcome was not satisfactory, because of the lightening effect of tretinoin in the surrounding normal skin. Considering the lack of a satisfactory treatment, prevention measures (sun protection) should begin in early childhood and extend throughout life. Avoiding sun tanning is an important adjunct to prevention, as tanning accentuates the process and intensifies the pigmentary contrast.

The unique profile of IGH warrants more research into the mechanisms responsible for this hypomelanotic condition. The typical histopathological features of the white macules are a flattening of the dermal-epidermal junction, moderate to marked reduction in the number of DOPA-, c-KIT- and TRP1-positive melanocytes in the basal layer (Ortonne and Perrot, 1980; Wallace et al, 1998). Ultrastructurally, some of the melanocytes have normal melanogenic activity and contain numerous melanosomes while others contain only immature melanosomes and present some degenerative changes (attenuated dendrites, dilatation of the endoplasmic reticulum, swelling of the mitochondria) (Vachiramon, 2001; Kim et al, 2010). The melanosome content of the epidermal keratinocytes is overall decreased.

The pathogenesis of IGH remains unknown, although several plausible explanations were advanced:

a. The hypothesis of age-related somatic mutations
b. Possible inhibition of tyrosinase activity due to a reduction in catalase
c. The decrease in melanocyte number as a result of early aging of the skin (as it is most commonly seen in elderly people)
d. The triggering effect of microtrauma (although the supposition remains controversial) (Shin et al, 2001; Kim et al, 2010)

e. Genetic non-environmental triggers (frequent clustering of cases within families, compared with unaffected controls) (Falabella et al, 1986); a small case-control study of candidate genes in renal transplant patients with IGH showed some association with HLA-DQ3 and lack of association with HLA-DR8 (Arrunategui et al, 2002)
f. Direct cytotoxicity from chronic exposure to UV radiation which causes the loss of melanocytes. This is supported by arguments like: 1. Occurrence of the IGH lesions on the sun-exposed areas on patients with a personal history of chronic exposure; 2. IGH-like lesions are often developed after PUVA or NB-UVB phototherapy (Friedland et al 2010). Both short intense UV exposure, but also cumulative exposures were incriminated. It is noteworthy that, according to studies in mice, pigment cells can be killed by large doses of PUVA, being susceptible to UV injury (Friedland et al 2010).

Considering its cosmetically unacceptable clinical picture causing patients' distress, this insufficiently explored disease needs more attention, that can be focused on two directions:

1. A more in-depth basic research, to bring light into the yet undiscovered mechanisms.

2. Since there is no definite treatment for this condition, the preventive measures are particularly essential. With the known relationship between sun exposure and IGH, we strongly encourage the clinician to educate population on sun avoidance and sun protection with appropriate clothing and sunscreen with broad spectrum starting at early ages.

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HOT OFF THE PRESSES

In this number we invited the lead authors of two recent publications - Drs. Feng Liu and Tatiana Krasieva - to share with us the main findings reported in Pigment Cell Melanoma Research and Journal of Biomedical Optics, respectively.

Melanoma and NMSC comparison: UV or not UV?

by Dr. Feng Liu

Publication: Liu F, Bessonova L, Taylor TH, Ziogas A, Meyskens FL Jr., Anton-Culver H. A unique gender difference in early onset melanoma implies that in addition to ultraviolet light exposure other causative factors are important. Pigment Cell Melanoma Res 2013, 26(1):128-135.

What led to the study: This study originated from a critique that I received for my career development award application from NCI. I submitted a molecular epidemiology grant with Drs. Frank Meyskens, Hoda Anton-Culver and Argyrios Ziogas as my mentors. One of the major criticisms was that I did not have an epidemiology background. To obtain such training, I began my work with nationally renowned epidemiologist Dr. Anton-Culver who suggested that I should examine melanoma databases. I found that incidence rate was higher for young women than for young men in the US SEER17 database. To address whether this is caused by UV radiation, my mentors suggested using NMSC incidence rate as a UV-causative control for melanoma.

Summary of results: By comparing the age- and gender-specific incidence rates between melanoma and non-melanoma skin cancer (NMSC) in the Nordic Cancer Registry database, we have discovered that young women are at higher risk for melanoma, but not for NMSC, than young men. The peak difference of melanoma incidence rate between genders is at age 20-24 years (for women RR = 2.33, 95% CI is 2.09 to 2.61), where no difference was observed for incidence rate for NMSC for this age group (RR = 0.86, 95% CI is 0.64 to 1.17). In the old age groups (>50 years), the RR pattern is similar for melanoma and NMSC, suggesting a shared UV radiation impact.

Impact to the scientific community: While it has been known that young women are more susceptible to melanoma than young men, the comparison of age- and gender-specific incidence rates between melanoma and NMSC was not performed before, largely because data for NMSC was not reported for most countries including the USA. Our study suggests that in early onset melanoma, UV radiation may play a less important role while etiological factors such as gender-related factors including hormones may be more important.

Impact to the public: Numerous epidemiology studies of hormone impact (including hormone replacement therapy, oral contraceptive use and pregnancy) on melanoma incidence rate have been carried out and provided strong evidence that these events do not increase melanoma incidence rate in women. Hence, the precise mechanism leading to high melanoma incidence rate in young women is still not understood, and needs further research at various levels. Understanding these mechanisms is very important for effective prevention, and may also result in differential treatment approaches.

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Two-photon excited fluorescence provides a clear fingerprint identification of keratin, eumelanin, and pheomelanin in a single, label-free image. This could potentially be used for rapid characterization and imaging of melanoma *in-vitro* and *in-vivo*.
by Dr. Tatiana Krasieva

Publication: Krasieva TB, Stringari C, Liu F, Sun CH, Kong Y, Balu M, Meyskens FL, Gratton E, Tromberg BJ. Two-photon excited fluorescence lifetime imaging and spectroscopy of melanins in vitro and in vivo. J Biomed Opt 2013, 18(3): 31107.

Recently we have published an article in the Journal of Biomedical Optics (Krasieva et al., 2012) about a new method for distinguishing and measuring human eumelanin and pheomelanin in cells and tissue. The method is based on two-photon excited fluorescence (TPEF) using short-pulse near infrared (NIR) lasers. It is rapid, label-free, and non-destructive.

Currently, the total melanin content (eumelanin plus pheomelanin) in biological specimens is typically determined by measuring light absorption at 450 to 475 nm after alkaline degradation (Kadekaro et al., 2003). Due to a lack of understanding of the structures of eumelanin and pheomelanin, it has not been possible to directly measure these two types of melanins. Ito and Fujita developed an HPLC-based method to indirectly measure eumelanin and pheomelanin after a series of degradation and oxidation steps (S. Ito, Fujita, K., 1985; S. Ito, Wakamatsu K., 1994). Because of the complicated processing steps and amplification constant used in this method, a small error in the HPLC measurement can result in a large error in the final reading.

Changes in the amounts of cellular eumelanin and pheomelanin have been associated with carcinogenesis. Thus rapid, non-degrading methods enabling researchers to distinguish between those two melanins would be highly desirable. Researchers from Duke University have developed a promising pump-probe imaging approach (Matthews, Piletic, Selim, Simpson, & Warren, 2011; Matthews, Wilson, et al., 2011) for separating pheomelanin from eumelanin and have demonstrated its performance in excised human pigmented lesions and *in-vivo* in a murine model. Other optical approaches rely on reflectance spectroscopy (Marchesini, Bono, & Carrara, 2009) and absorption spectroscopy (Zonios, 2008).

There is a limited amount of published data on the fluorescence of naturally occurring melanins. *In-vivo* excitation of fluorescence using visible light is impractical due to low quantum yields, poor light penetration in tissue, and the strong, broadband melanin absorption which can lead to self-quenching and photodamage. NIR two-photon excitation is advantageous due to lower scattering, enhanced skin penetration, and reduced photodamage. We examined and characterized naturally occurring keratin, eumelanin and pheomelanin in human hair of different colors (red, brown and gray) using TPEF. We found that the pheomelanin emission peaks at approximately 615 to 625 nm and eumelanin exhibits a broad maximum at 640 to 680 nm. Based on these data we defined an optical melanin index (OMI) as the ratio of fluorescence intensities at 645 and 615 nm. The measured OMI for the MNT-1 melanoma cell line is 1.6±0.22 while the Mc1R gene knockdown lines MNT-46 and MNT-62 show substantially greater pheomelanin production (OMI 0.5±0.05 and 0.17±0.03, respectively). The measured values were in good agreement with chemistry-based melanin extraction methods.

In-vitro measurements were obtained from melanoma cells and artificial tissue constructs, and *in-vivo* from healthy volunteers. For this is a microscopy-based method, it provides us with both spectral properties (ability to separate the melanins) and spatial information (providing sub-micron resolved images). It is a label-free method relying on native properties of cells and tissues. So, in order to better separate melanin

fluorescence from other intrinsic fluorophores, we performed fluorescence lifetime imaging microscopy of *in-vitro* specimens. The relative concentrations of keratin, eumelanin, and pheomelanin components were mapped with the sub-cellular accuracy. Our results suggest that a noninvasive TPEF index based on spectra and lifetime could potentially be used for rapid melanin ratio characterization both *in-vitro* and *in-vivo.*

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MEMBERS IN THE NEWS

The group of scientists led by **PASPCR President-elect, Dr. Caroline le Poole**, recently published their findings on a modified HSP70 that holds promise in treatment of vitiligo. The authors showed that in a mouse model of depigmentation, modified inducible heat shock protein 70 (HSP0i) prevented T cell-mediated depigmentation. The mutant protein was able to reverse the autoimmune response involved in vitiligo. Some of the effects observed in mice were also reported in human skin specimens.

The citation for this breakthrough manuscript is:

J. A. Mosenson, A. Zloza, J. D. Nieland, E. Garrett-Mayer, J. M. Eby, E, J. Huelsmann, P. Kumar, C. J. Denman, A. T. Lacek, F. J. Kohlhapp, A. Alamiri, T. Hughes, S. D. Bines, H. L. Kaufman, A. Overbeck, S. Mehrotra, C. Hernandez, M. I. Nishimura, J. A. Guevara-Patino, I. C. Le Poole, Mutant HSP70 Reverses Autoimmune Depigmentation in Vitiligo. *Sci. Transl. Med.* **5**, 147ra28 (2013).
http://www.ncbi.nlm.nih.gov/pubmed/23447019.

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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 paspcr.newsletters@gmail.com.

*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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Position Available

A **postdoctoral fellowship** position is ***immediately available*** in the laboratory of
Zalfa Abdel-Malek, Ph.D.
Department of Dermatology
University of Cincinnati College of Medicine, Cincinnati, Ohio

The focus of Dr. Abdel-Malek's research is the photobiological responses of human melanocytes, modulation of these responses by melanocyte stimulating hormone and the melanocortin 1 receptor, and the signaling pathways activated by UV-induced paracrine and autocrine factors. These projects are funded primarily by NIH R01 grants. Since the position of postdoctoral fellow is supported by NIH T32 training grant, the candidate should be an American citizen or a permanent resident of the U.S.A.

Minimum requirements for the position are a Ph.D. in Biology, with expertise in cell and molecular biology techniques, including tissue culture, imunocytochemistry, Western blotting, and PCR. The candidate should be willing to work collaboratively as a team member.

Interested candidates must immediately contact Zalfa Abdel-Malek by email at abdelmza@ucmail.uc.edu.

2013 PASPCR MEMBERSHIP LIST

Dear PASPCR members,

Thank you for supporting our Society and paying your dues in time! I am pleased to announce that the first person who renewed for 2013 is Dr. Tae-Jin Yoon from Gyeongsang National University Hospital, and the first person who joined the Society for 2013 is Dr. David Norris from University of Colorado.

Andrzej Slominski

The PASPCR Membership List is published in the April issue 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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