

PASPCR

September-December 2007
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Newsletter



Introduction...

by *Bill Oetting*

The 14th Annual Meeting of the PASPCR, held in Chicago, on September 13 - 16, was a great success. Caroline Le Poole and Vijay Setaluri, and the members of the Organizing, Scientific Review and Poster Evaluation Committees put together an informative and enjoyable meeting. A meeting report can be found on pages 9 to 14 of the *PASPCR Newsletter* as well as on the PASPCR web site. You can also see photographs taken at the annual meeting.

New articles are being added to the PASPCR Commentary Page. These articles contain the latest in pigment related research and thoughts on pigment cell biology and physiology. The link can be found at the PASPCR home page. If you would like to see a particular topic included, or wish to write one yourself, please contact John Pawelek at john.pawelek@yale.edu.

The *PASPCR Newsletter* is published quarterly and is intended to serve as a means of communication for the members of our Society. You are invited to contribute articles, or other information you feel will be of interest to members of the PASPCR. If you attend a scientific meeting and have heard results 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. Any information on upcoming

meetings of interest will be added to the "Calendar of Events". This is your newsletter, and we depend upon you to help us make sure it best serves the Society's needs. Contributions and comments can be sent to me, preferably by E-mail, to bill@lenti.med.umn.edu.

The PASPCR Web Site is the major, up-to-date source of current information for the PASPCR membership and for individuals who are interested in the PASPCR. If there is additional information that you would like to see on the Web site, or you would like to include information of past PASPCR activities, please let me know and I will add them.

The IFPCS web site can now be reached by using the domain name **ifpcs.org**. The domain name **ipcc.info** will take you to the IPCC web site, providing you the most up to date information on the International Pigment Cell Conference which will be held on May 7 - 12, 2008 in Sapporo, Japan.

The PASPCR Web Site can be found at:

<http://www.paspcr.org>

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Zalfa Abdel-Malek,
President, IFPCS
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Calendar of Events:

2008 XXth International Pigment Cell Conference and Vth International Melanoma Research Congress
Held on May 7-12, 2008.
Contact: Kowichi Jimbow
E-mail: Go to web page for contact information
Web site: www.ipcc.info

2008 Melanoma 2008: 18th Annual Cutaneous Malignancy Update, to be held at the Omni San Diego Hotel, 675 L Street, San Diego, CA 92101
Held on January 19-20
Contact: Carlin Admirand
Email: med.edu@scrippshealth.org
Tel.: (858) 587-4404

2008 International Investigative Dermatology (Joint Meeting of the ESDR, SID and JSID) to be held in Kyoto, Japan.
Held on May 14-17
Contact: ESDR Office
E-mail: office@esdr.org
Web site: www.esdr.ch

2009 XVth Meeting of the ESPCR to be held in Münster, Germany
Held on September 20-23,
Contact: Dr Markus Böhm
Email: bohmm@uni-muenster.de

If you know of future meetings that you feel would be of interest to the PASPCR membership, please let us know.

The *PASPCR Newsletter* is published quarterly by the PanAmerican Society for Pigment Cell Research. All views are those of the authors. For further information or to submit articles, please contact members of the Publications Committee.

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Corporate Sponsors

by *Raymond E. Boissy*

The PASPCR would like to acknowledge and thank our Corporate Sponsors; the list below reflects contributions over the past 2 years. Financial gifts from these sponsors have allowed our Society to increase benefits to the membership far out of proportion to the actual dues collected from members. Monies contributed by these sponsors have been used over the years to support various PASPCR functions including our Young Investigator Award program, meeting travel stipends, annual meeting expenses and this Newsletter.

GOLD Corporate Patrons

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Mary Kay Inc.
POLA Chemical Co.

New Members

by *Raymond E. Boissy*

The PASPCR would like to welcome a new member to the Society:

Nannan Chen

Johnson & Johnson
Skillman, NJ

Claude Saliou

Johnson & Johnson
Skillman, NJ

Isabel Santana

Unilever
Trumbull, CT

New Members (continued)

Abdel Belmadani

Northwestern University Feinberg School of Medicine
Chicago, IL

James Lister

Virginia Commonwealth University Medical Center
Richmond, VA

Manpreet Randhawa

George Mason University
Manassas, VA

Rong Hu

Albany Medical College
Albany, NY

Tae-Jin Yoon

Gyeongsang National University Hospital
Jinju, Korea

Li Ni Komatsu

New York University
New York, NY

James Fichtelman

Vero Beach High School
Vero Beach, FL

Jon Fichtelman

Holy Cross Hospital
Fort Lauderdale, FL

Sluochama Devi

Univ Wisconsin – Madison
Madison, WI

Stacie Loftus

National Human Genome Research Institute
Bethesda, MD

Nina Jablonski

Pennsylvania State University
University Park, PA

Youwen Zhou

University of British Columbia
Vancouver, BC

Dear Members and Friends of the PASPCR:

It is astonishing to me that this is my final newsletter column as president of this wonderful society of pigment cell scientists and clinicians. I have enjoyed myself thoroughly through the past 6 years, first as president-elect and then as president, and I will miss it. Nonetheless, I am happy to announce that at the stroke of midnight, December 31, 2007 we will be transferred to the capable hands of Frank Meyskens as president and Greg Barsh as president-elect. And I am not the only turn-over at PASPCR. Ray Boissy, our steadfast Secretary-Treasurer for countless years is carefully handing over his duties (and there are many) to Andrzej Slominski. We heartily congratulate Ray on his being awarded the title of "PASPCR Secretary-Treasurer Emeritus" which he received at the last meeting through a presidential decree and a plaque. And finally, Bill Oetting is retiring from his long-held post of Newsletter Editor. We thank Bill enormously for his consistent, excellent contributions to this task. The newsletter has been so helpful in keeping us connected and Bill has been the master behind it for years and years. Fortunately, Bill is not leaving completely and will keep connected to us by continuing to manage our website. Again we thank you Bill! And seemingly with unending good fortune, we have not one but two new volunteers to take over the newsletter: Emilia Costin and Prashiela Manga. So there are big changes afoot! But it bodes well for the Society that such capable people are available to take over the leadership. That is a real test of strength in all democratic organizations and happily we have met it well. I am also pleased to mention that I will continue to organize the PASPCR Commentary feature that has been coming out more-or-less monthly for two years now. If anyone wishes to contribute, please contact me (john.pawelek@yale.edu). The format is 1000-1500 words tracing your scientific path to your current research interests. The length of the path doesn't matter, so you can write a commentary even should you be early in your career.

Finally, I would like to mention how much I personally enjoyed the 2007 conference in Chicago and I thank all the organizers of that event, particularly Caroline LePoole and Vijay Setaluri. The quality of the presentations was very high and enjoyed by all of the many attendees. I was especially pleased that we had three plenary talks and many separate discussions about the impacts of human skin color in society. As an outgrowth of this, Frank Meyskens has asked me to head a committee to fashion a website statement on the complicated impacts of skin color in society. Should anyone have suggestions for this statement or wish to join the committee, please contact me.

Best wishes to all!

John Pawelek
(still) *President, PASPCR*

PASPCR Award recipients

Here are the individuals who were presented awards for their research at the 14th Annual Meeting of the PASPCR.

PASPCR Poster Awards

Julio C. Valencia, Li Ni-Komatsu and Pei-Chih Lee

PASPCR Career Achievement Award

Meenhard Herlyn

PASPCR Special Lectureship

Beatrice Mintz

Chairman's Award

Elizabeth Hearing and Vincent J. Hearing

Honorary Member

Richard King

Congratulations!

Further memories of Aaron Lerner
by Seymour Pomerantz

I was recently looking through the March, 2007 issue of the PASPCR Newsletter and saw the appreciations of Aaron Lerner. I realized that I had some additional items to add about Aaron's kindness to a young investigator.

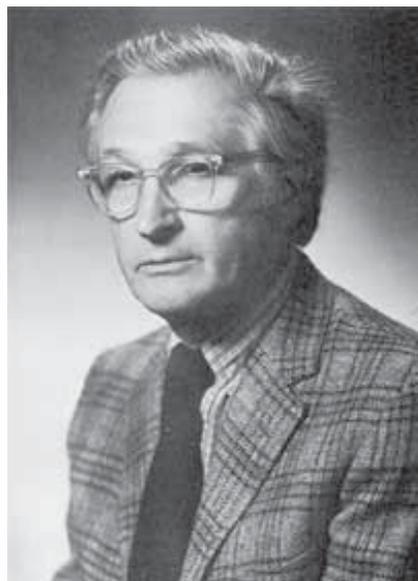
In 1957 I was hired by the late Richard Stoughton to do biochemical research in a new skin lab that he was setting up at Western Reserve University School of Medicine. For the first two years I investigated carbohydrate metabolism and the Krebs cycle in the skin of newborn rats.

In the autumn of 1959 I was looking for another problem related to skin and I discovered the papers of Aaron with Thomas Fitzpatrick on tyrosinase in homogenates of mouse melanoma. This immediately attracted me and I thought that this would be an interesting area in which only a few papers on the mammalian enzyme had been published. In December of that year I wrote Aaron and asked his advice about how to approach the problem of solubilization of the enzyme. He responded immediately and invited me to come to New Haven for a visit. The visit was arranged for some time in January, 1960. Aaron gave me a lot of time and we discussed many aspects of the problem. He suggested that I use hamster melanoma rather than the Cloudman mouse melanoma because the mouse tumor often became amelanotic. He said that he was propagating a hamster tumor that had been discovered as a spontaneous tumor by Harry Greene of the Yale Pathology Department and that when I was ready to start work that I should contact him again and he would send me the tumor. He was quick to assure me that I would not be competing with him because he was not directly interested in the purification of the enzyme. True to his word, after I received notice of the award of an NIH grant on the subject, he sent

me two hamsters with tumors. I propagated this tumor for about twenty years.

Later on when I became interested in MSH and its effects on tyrosinase Aaron generously sent me a fair amount of a partially purified preparation of beta-MSH which I purified further. On other occasions Aaron invited me to meetings which were held at Yale.

At the time of my first contacts with Aaron he was already the head of an important Dermatology Department in a leading university and I was just beginning a career as an independent investigator. Yet he took the time to see me, furnish valuable advice, and then gave me the tissue I needed to enable my work to get off the ground. We were fortunate to have this generous and wise scientist as a colleague.



Aaron Lerner

Positions Wanted / Available

Postings for **Positions Available** will be open to all individuals and institutions so long as the position is related to pigment cell research. Postings for **Positions Wanted** will be open only to members of the PanAmerican Society for Pigment Cell Research or its sister societies (JSPCR and ESPCR). Send postings to Bill Oetting at bill@lenti.med.umn.edu. Please provide an expiration date for any submitted postings. Final decisions will be made by the Publications Committee of the PASPCR.

Positions Wanted

Postdoctoral Position Wanted

Postdoctoral level position wanted for a Scientist with more than 3 years of postdoctoral experience with expertise in primary melanocyte culture, cell signaling in melanocytes, developing transgenic mouse models. Available to join immediately. Please respond at daizeus@yahoo.com.

Positions Available

Postdoctoral position

We are looking for an enthusiastic postdoctoral researcher interested in studying immune responses to melanoma tumors and autoimmune reactivity to melanocytes for NIH funded research.

We utilize primary cell cultures established from mice and humans, including T cells lines and clones. Keywords frequently used in the lab are TCR affinity, vaccines, murine models, depigmentation, T cell cloning, and HSP70. Send inquiries, resume and/or contact information to:

I. Caroline Le Poole Ph.D.
Associate Professor of Pathology
Loyola University Chicago
2160 S. 1st Ave, Bldg 112, Rm 203
Maywood, IL 60153
Tel. 708-327-2032
Email ilepool@lumc.edu

Senior Scientist

For over a century Avon has been a leading global beauty company for women, with a commitment to innovation through research and development.

We are currently recruiting for a Sr. Scientist role. The candidate's focus will be to conduct laboratory research for the validation and characterization of new molecular targets related to skin biology and evaluation of technologies for cosmetic product development.

The candidate will work with Avon scientists to determine areas of research and independently design and carry out the experimental phase of the research. A successful candidate will demonstrate the ability to independently design, execute, and evaluate experimental protocols. In an interpretable manner, the candidate will be required to archive all results in written and computer format as well as communicating the experimental outcomes to other scientists and/or team members to guide the progression of research projects. The candidate will also be expected to provide scientific input into the on-going projects of other scientists and/or teams in Avon R&D.

The Candidate requires a Ph.D degree in Biology, Immunology, Cell Biology or other related field. The candidate must have at least 5 years of experience in cell and tissue culture techniques, experimental design and data analysis. Knowledge and experience with qPCR, non-radioactive in situ hybridization, and cell co-culture would be an advantage. Experience or exposure to medicinal chemistry or natural products would be helpful. The candidate must have a proven record of productivity in their field of research. The candidate must also demonstrate strong communication and writing skills. The candidate must act professionally in all situations, and function well in a matrix and team-oriented environment. Flexibility and adaptability to changes are also key characteristics.

To apply for this position please send a resume to jobs40@avon.com. or visit our website at www.avon.com and refer to Job ID 4119.

Lisa Platek
Human Resources Recruiter
Avon Products Research and Development
1 Avon Place
Suffern, NY 10901
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Senior Scientist

The Senior Scientist will be responsible for basic and applied research in the areas of skin pigmentation and UV protection. The candidate will be involved in the design, performance and analysis of in vivo and in vitro studies, including the use of microscopic image analysis of histological data. Experience in both in vivo and in vitro studies is desired, including in vivo treatments, and techniques such as enzymatic activity analysis, DNA/RNA/protein extraction, and Immunohistochemistry. Responsibilities for the position also include documenting studies in clinical records, research notebooks and reports, presenting research data, reading scientific literature and participating in research discussions and seminars.

Qualifications

BS/MS in biology or related sciences with hands on experience required in both in vivo and in vitro work. Molecular biology and/or biochemistry knowledge and experience in various biological research techniques (RT-PCR, Western blot, IHC, enzymatic activity analysis, DNA/RNA/protein extraction) are desired. Microscopy and computerized image analysis experience is a plus. Ability to design studies, execute and analyze data, make conclusions and suggestions for next steps is required, as well as the ability to learn new techniques and procedures. Ability to engage in both individual studies and in teamwork is a must.

To apply for this position, contact:

Miri Seiberg, PhD
Principal Research Fellow

Skin Biology and LAS
The Johnson & Johnson Skin Research Center, CPPW,
a unit of Johnson & Johnson Consumer Companies, Inc
199 Grandview Rd, Skillman, NJ 08558
Phone: 908-874-2325
Fax: 908-874-1254
e-mail: MSEIBER@CPCUS.JNJ.COM

Assistant/Associate/Full Clinical Professor Medical Oncology and Hematology University of California, Irvine

Two new positions in medical oncology and hematology are available at the Assistant/Associate/Full Health Sciences (Clinical) Professor level (rank dependent on qualifications) in the Department of Medicine, Division of Hematology/Oncology at the University of California, Irvine, site of an NCI designated comprehensive cancer center. Applicants must have MD or MD/PhD and be BE/BC in Medical Oncology. These positions are for Academic Clinicians who, in addition to patient care activities, are interested in participating in established clinical trials and teaching. Time and resources to assist in the development and execution of novel translational research and develop investigator-initiated trials will be made available. One position is for an individual with interest in melanoma and the other for an individual with interest in pancreatic and hepatobiliary cancers.

UCI is an equal opportunity employer committed to excellence through diversity. Send curriculum vitae with names and telephone numbers of three references and a statement of your academic goals to:

Randall F. Holcombe, M.D.
Chief, Division of Hematology/Oncology
Associate Director, Chao Family
Comprehensive Cancer Center
c/o Krista Hollinger, Divisional MSO
101 The City Drive
Bldg 56, RT 81, ZOT 4061
Orange, CA 92868
Tel: 714-456-5153
Email: kholling@uci.edu

Postdoctoral Position

A postdoctoral position available in the laboratory of Dr. Andrew Aplin in the Center for Cell Biology and Cancer Research at Albany Medical College, NY. Research will focus on the critical signaling proteins involved in anchorage-dependent cell growth of melanocytes and that may be aberrantly regulated in melanoma cells. Further details and recent publications can be obtained at <http://www.amc.edu/academic/research/CBCResearcher.cfm?ID=170>

Albany Medical College is located in the scenic Hudson River Valley, offering affordable housing, easy commutes and quick access to cultural (e.g., Saratoga, 45 min; Tanglewood, 1 hr), and outdoor activities (Adirondack State Park, 2 hr).

Candidates with a recent PhD or MD/PhD with a strong background in molecular and cellular biology are encouraged to apply. Excellent financial compensation and benefits are provided. Please submit a resume and the names of references to:

Andrew E. Aplin, Ph.D.
Center for Cell Biology & Cancer Research
Albany Medical College,
47 New Scotland Avenue
Albany, NY 12208
Email: aplina@mail.amc.edu

The Albany Medical College is an equal opportunity, Affirmative Action Employer

Postdoctoral Research Associate

Fox Chase Cancer Center.
Two NIH-funded postdoctoral positions are available to work on the development of neural crest-derived melanocytes and enteric neurons in mice. We are interested in the signals required for proper migration and differentiation of these lineages during mouse embryogenesis and use various genetic manipulation techniques and existing mutants for our studies. Fox Chase Cancer offers competitive salaries to its postdocs and was recently named one of the best places to work for Postdocs ([http://www.fccc.edu/news/2003/Best-Places-for-Postdocs-02-20-](http://www.fccc.edu/news/2003/Best-Places-for-Postdocs-02-20-2003.html)

[2003.html](http://www.fccc.edu/news/2003/Best-Places-for-Postdocs-02-20-2003.html)). Candidates with a recent PhD or MD/PhD with strong background in molecular biology, genetics or developmental biology are encouraged to apply. Please submit CV, and names of 3 references to:

Dr. Myung K. Shin
Program in Cellular and Developmental Biology
Fox Chase Cancer Center
Philadelphia, PA 19111, USA
Email: MK_Shin@fccc.edu

**Report on the 14th Annual Meeting
Knickerbocker Hotel, Chicago, Illinois
September 13-16, 2007**

Session 1; Cell Biology and Pigmentation

Chairs: Andrzej Slominski, Vincent Hearing and Giselle Thibaudeau

by **Giselle Thibaudeau**

The first session of the 2007 Annual PASPCR meeting was titled *Cell Biology and Pigmentation* and presented exciting novel findings that will contribute to our understanding of pigmentation and pigment pattern formation at several levels. Presentations provided new information regarding the modulation of melanosome tethering in dendritic tips of melanocytes, subsequent transfer of melanosomes to keratinocytes, and signaling and cross-talk within the epidermal/melanocyte unit. Here are the highlights and take-home messages.

Chediak-Hagashi Syndrome (CHS) is characterized by mild hypomelanosis of the skin, hair and eyes. This hypomelanosis results from the retention of enlarged melanosomes in the melanocyte cell body and the absence of melanosome transfer to the keratinocytes. Here, **Wendy Westbroek** and colleagues (*The Rab27a/Melanophilin/Myosin Va tripartite complex does not target enlarged melanosomes to actin filaments in Chediak-Higashi melanocytes*) demonstrate that Rab27a/Melanophilin/Myosin Va tripartite complex, necessary for melanosome transfer to the dendrites and subsequent transfer to keratinocytes, does not form in melanocytes of patients with CHS. Absence of melanosome tethering to the actin in dendritic tips of CHS melanocytes could begin to explain the skin hypomelanosis associated with CHS.

Sheila Rocha and colleagues (*RNAi knockdown semaphorin 6D decreases melanosome transfer from melanocytes to keratinocytes*) describe the first instance of modulating melanosome transfer via semaphorins and present a novel means of analyzing the transfer process. Neural crest-derived origin of melanocytes is interesting in light of semaphorins having a role in neural path finding. Along with their receptors, the plexins, semaphorins may be involved in skin pigmentation. Here, Semaphorin 6D knockdown in keratinocytes resulted in significant inhibition of melanosome transfer. While melanocytes can transfer melanosomes to several keratinocytes, keratinocytes

accept melanosomes from only a single melanocyte, regardless of multiple melanocyte contacts. Collectively results indicate a role for semaphorins in melanosome transfer and skin pigmentation.

Glynis Scott and colleagues (*Semaphorin 7a promotes spreading and dendricity in human melanocytes through δ α 21-integrins and Plexin C1*) describe the function of Semaphorin 7a on human melanocyte dendricity. They show that Sema7a is produced by human keratinocytes and fibroblasts in vitro, is expressed in the skin in vivo, but is not expressed in melanocytes. Melanocytes express both Sema7a receptors, α 21-integrin and Plexin C1. Both receptors have important, but opposite, effects in mediating the dendricity response to Sema7a. Binding of Sema7a to α 21-integrin stimulates dendricity, whereas silencing Plexin C1 stimulates dendricity. Interestingly, Plexin C1 is a negative regulator of cofilin, an actin severing protein. Sema 7a is identified as a novel paracrine factor for melanocyte dendrite formation through opposing actions of α 21-integrin and Plexin C1 signaling. In addition, Cofilin is suggested to play an important role in mediating these effects of Plexin C1.

Janice L Brissette and colleagues (*Foxn1, epithelial cells, and the patterning of pigmentation*) show that epithelial cells influence melanocytes and organize the epithelial/melanocyte unit, and effectively, engineer their own pigmentation through the action of the epithelial cell-specific transcription factor, Foxn1. Foxn1 is a product of the nude locus and is an epithelial cell-specific transcription factor. Foxn1 induces Fgf2 secretion, and attracts melanocytes to the region containing Foxn1-positive cells. The melanocytes then transfer the melanin to these Foxn1-containing cells. Foxn1 is shown to regulate pigmentation via Fgf2. Foxn1 initiates terminal differentiation and serves to identify keratinocytes as the target for melanosome transfer. In this way, Foxn1 functions to activate a distinct set of traits giving rise to a pigment recipient phenotype, through which epithelial cells recruit pigment donors and stimulate melanin transfer. These pigment recipients constitute a specialized counterpart to the melanocytes and provide a blueprint that instructs melanocytes where to place the pigment.

Gregory S Barsh and colleagues (*A new ligand for melanocortin receptors*) have recently identified K locus involvement in the dominant inheritance of black coat color in the dog. K is distinct from, but interacts with Agouti and Mc1r,

does not correspond to the predicted location of any previously known pigmentation gene, encodes a protein secreted by keratinocytes, and whose human ortholog is a beta-defensin gene. The beta-defensins are thought to play important roles in the innate immune system, protecting the body surface from microbial attack. Beta-defensins bind with high affinity to Mc1r and modulate melanocortin signaling by preventing agouti from binding Mc1r. The result is that beta-defensins have significant effects on pigment type switching. The mechanism of K involvement ($K^{\nu} < K^{br} < K^B$) leads to interesting questions regarding defensins involvement in pattern generation. Collectively, results provide insight into comparative mammalian genetics in general, and melanocortin receptor signaling specifically.

Miri Seiberg and colleagues previously showed that PAR-2 regulates pigmentation by controlling phagocytosis of melanosomes. Here (*LIGR, a protease-activated receptor-2-derived peptide, enhances skin pigmentation without inducing inflammatory processes*) they show that LIGR stimulates Rh0-GTP activation and induces skin pigmentation by activating only a subset of the PAR-2 signaling pathways and with no induction of inflammatory mediators. LIGR is a more specific regulator of PAR-2 induced pigmentation compared to SLIGRL (another known PAR-2 activator). The suggestion is made that topical treatment of LIGR may result in tanning by enhancing the natural pigment content of the skin, without enhancing inflammatory processes and without the need of UV exposure.

Session 2; Malignant Transformation

Chairs: David Norris and Brian Nickoloff
by **David A. Norris, MD**

Frank Meyskens presented work from the University of California at Irvine supporting his hypothesis that the initiation of melanoma is epigenetic and involves ultraviolet light, Fe, Cobalt/Copper. They propose that the anti-oxidant action of melanin is turned to pro-oxidant by metals cobalt and copper, producing free radical generation through the Fenton reaction. Epidemiological studies have shown that industrial metal exposure in the printing and electrical industry and in surgical implants are associated with increased risk of melanoma.

Valerie Trapp from UC Irvine have developed an interesting three dimensional culture system in which

spheroids containing melanoma cells and tumor derived endothelial cells are co-cultured and tested for the effects of anti-angiogenic molecules. Using this system, they found that tumor stromal cells producing high levels of thrombospondin 1 (TSP-1) made co-cultured melanomas highly susceptible to anti-angiogenic therapy.

Jack Longley from the University of Wisconsin proposes a paradigm shift in how we think about MAGE (Melanoma antigen). MAGE may be more than just a melanoma marker and a target for immunotherapy, but may be an important component of pathways controlling melanoma growth. MAGE expression is c-kit dependent, and siRNA knockdown of MAGE expression inhibits melanoma cell growth by inducing apoptosis. This effect may be p53 dependent, and MAGE inhibition may be an important factor in promoting apoptosis induced by drugs that induce p53.

Zalfa Abdul-Malek of the University of Cincinnati summarized a large body of work demonstrating that the melanocortin-1 Receptor is important in protecting human melanocytes from the effects of ultraviolet radiation. Melanocytes with certain MC1R alleles are naturally susceptible to the genotoxic and cytotoxic effects of UVR, and transfection of such cells with "wild type" MC1R restores protection. Protection can be blocked by using analogues of the agouti-signaling protein, further illustrating the importance of control of pigmentation and photoprotection at the level of the MC1R.

Thomas Hornyak from the Dermatology Branch of the National Cancer Institute addressed whether polycomb group proteins are involved in melanoma initiation or transformation. Polycomb group proteins are epigenetic gene silencers involved in normal development, stem cell maintenance, and oncogenesis. Hornyak focused on the role of two of these proteins in melanoma: BMI-1 which mediates repression of CDKN2A which encode p16, and EZH2 whose increased expression is associated with increased proliferation and decreased survival in melanoma. Interestingly, EZH2 levels were low in normal melanocytes and nevi, but elevated in melanomas. BMI-1 levels were uniformly high in melanocytes, nevi and melanomas, and siRNA silencing of BMI-1 had no effect on cell proliferation and anchorage-dependent growth. Hornyak speculates that in senescent nevi, EZH2 expression suppresses CDKN2A, decreasing the tumor suppressor p16, and leading to malignant transforma-

tion.

Xiao-qi Wang of Northwestern University questioned whether different isotopes of Gm3 gangliosides correlated with metastasis in melanoma. Using a 2 layer Matrigel invasion assay, and silencing or over expression of key components of the pathway, they have shown that GM3 increases invasion acting sequentially through urokinase plasminogen activator and receptor, p38 MAPK, and matrix metalloproteinase 2 (MMP2). The authors propose that targeting GM3 may be a therapeutic strategy for melanoma therapy.

Session 3; Stem Cell Biology and Differentiation
Chairs: James Grichnick and Deborah Lang
by **James Grichnick**

The first talk was titled *The Chemokine SDF-1 regulates the development of Neural crest progenitors of the melanocyte lineage in Mouse skin* by **Abdelhak Belmadani**, Dongjun Ren and Richard J. Miller. Dr Belmadani presented data on the chemokine SDF-1 (CXCL 12) and its receptor CXCR4. SCF-1 was shown to be a chemoattractant for CXCR4 bearing cell in culture. In CXCR4 mutant mice DCT positive cells were noted aberrantly in the epidermis presumably due to defect migrating into the hair follicle and increased apoptosis was noted in these cells. Thus this pathway may be critical for the migration of melanocytic precursors into the hair follicle.

The second talk was titled *Expression of melanocyte specific proteins and growth factor receptors in tissue and cultured cells from lymphangioliomyomatosis* by Jason A. Wilson, Chul Jong Park, Ljiljana Minwalla, Amy Koshoffer, Teresa A. Smolarek, and Raymond E. Boissy. **Dr Boissy** discussed the curious entity of lymphangioliomyomatosis. This neoplastic process was found to partially express pigmentation associated genes. Expressed genes included HMB-45 and Mart-1. Three of 5 specimens also expressed KIT and 2 of 5 also expressed the alpha receptor for estrogen. Similar expression patterns were also noted in culture.

The third talk was titled *A new mouse model for the detection of melanocyte stem cells* by Sara K. Powell, Kacey P. Young, Maria Nelson, Rebecca S. Plummer, and Deborah Lang. **Dr Lang** presented her work demonstrating that the PAX-3 promoter is ideal for the detection of melanocytic stem cells. In her system the PAX-3 promoter was linked to b-galactosi-

dase in transgenic mice. Unlike Sox-10 that remains on in the differentiated melanocytes, PAX-3 is down regulated – limiting its expression to the melanocytic stem cell compartment. PAX-3 expression was noted to increase in response to stress induced follicle regrowth post depilation and may serve as a unique marker for the isolation of melanocytic stem cells.

The forth talk was titled *Evidences for an ectopic synthesis of melanin in adipose tissue* by Manpreet Randhawa, Tom Huff, Julio Valencia, Vincent Hearing, Zobair M. Younossi, Ancha Baranova. **Dr Randhawa** presented data supporting the presence of melanin and pigmentary enzymes in the adipose tissue of obese individuals. Hypothetically, melanocortin stimulatory pathways in the obese state may also secondarily drive the aberrant expression of pigmentary pathways in fat cells. They were able to demonstrate the expression of Mart-1, MSH, and Tyr immunohistochemically and the presence of melanin PTCA determination in excised adipose tissue. Further they were able to demonstrate up regulation of MC1R, Tyr, TYRP-1 and MITF with alpha-MSH on mature adipocytes in vivo.

The fifth talk was titled *Early, over-expression of the endothelin signaling pathway leads to the generation of ectopic melanoblasts* by Avner Ittah, Shyla Mirabal, Roman J. Garcia, Lydia Kos. **Dr Kos** indicated that over-expression of the endothelin-B receptor (Ednrb) alone in transgenic mice actually resulted in a decrease in the number of melanocytes. This was speculated to be secondary to sequestration of a limited amount of ET-3 by the Ednrb receptor being prematurely driven by the nestin promoter. This defect was corrected by increasing the expression of ET-3 (under control of kartin 5 promoter) but this also lead to the ectopic expression of melanoblasts in the roof plate over the neural tube.

The sixth talk and final presentation was titled *Use of doxycycline-regulated transgenic mice to identify and characterize melanocyte label-retaining cells* by Ha-Young Hwang, Ganesh Diwakar, Thomas Hornyak. **Dr Hwang** report of the use of a DCT “tet off” system coupled with a K5 driven tetracycline transactivator inducing the expression of H2B green fluorescent protein. He was able to show that is the presence of tetracycline non-proliferative cells were localized in the hair bulge region (CD34 co-localization). This system may permit the isolation of viable melanocytic stem cells by fluorescence-activated cell

sorting.

Session 4; Pathology and Treatment of Pigmentary Disorders

Chairs: Sancy Leachman and Claudia Hernandez
by *Sancy Leachman*

The Pathology and Treatment of Pigmentary Disorders section of the PASPCR Meeting followed the Keynote Lecture by Victoria Holloway Barbosa on Sat. Sept. 15, 2007. The session began with a presentation given by **Yoshinori Miyamura** (Hearing Laboratory) on *De novo melanogenesis increases UV protection of human skin*. This abstract analyzed the protective effects of tanning fair skin using different UV sources (UVA, UVB, and SSR). Although the tanning response appeared similar clinically, UVA-induced tanning was not as photoprotective (as measured by CPD formation) as UVB- or SSR-induced tanning. These results suggest that chronic UVA tanning leads to increased pigmentation and melanin dispersion, but may also have negative effects regarding photoprotection.

The next presentation, *Skin pigmentation induced by repeated UV exposures and the role of melanin in photoprotection*, was given by **Sergio Coelho**, also from the Hearing Laboratory. Upon analyses of several biomarkers (cyclobutane pyrimidine dimers (CPD), nuclear accumulation of p53 protein, pigment content assessed by Fontana-Masson staining, melanocyte function by melanosomal protein expression, apoptosis by TUNEL assay and determination of Ser46-phosphorylated p53), these investigators found that the amounts of CPD decreased while the numbers of p53-positive cells increased with increasing cumulative doses of UV even though apoptosis, melanocyte function, and melanocyte density showed no dependence on cumulative doses. These findings led investigators to hypothesize that it is possible that the pigmentation from incremental doses of repeated UV protects against additional DNA damage as evaluated by CPD formation, but other mechanisms cannot be excluded at this time.

In a "first ever" for the PASPCR, a high school science fair winner, **James Fichtelman**, presented data obtained with the help of Jim Grichnik on the use of *Planaria as a model system for the study of pigment biology*. In this presentation, Mr. Fichtelman showed that *Planaria* have different pigmentary phenotypes and exhibit anticipated responses to UVR, including tanning and tumor formation. This makes this

organism worthy of consideration as a tool for the study of pigment cell biology. Another member of Dr. Grichnik's group, Adela Cardones, presented an interesting case of *alpha-MSH induced neovogenesis*. This case report demonstrated in a human subject, that nevocenic cells can be stimulated by alpha-MSH and that alpha-MSH levels support the growth of a subset of nevi. Importantly, the discussion following this talk raised the question of whether such pharmacologic increases in alpha-MSH also has the potential to enhance tumorigenesis, a potential cause for concern in patients who are using alpha-MSH agonists for cosmetic purposes.

Esteban Dell'Angelica also discussed *An immunoblotting assay to facilitate the molecular diagnosis of Hermansky-Pudlak syndrome*. Dr. Dell'Angelica (UCLA) introduced an immunoblotting-based screening assay (developed in collaboration with the group of Bill Gahl at the NIH) to narrow down the number of candidate genes to be sequenced for each patient with a new diagnosis of Hermansky-Pudlak syndrome (HPS). Because the eight known types of HPS are due to structural defects in any of four protein complexes (AP-3 and BLOC-1 through -3), the assay attempts to determine by immunoblotting which complex is defective in each patient.

A highlight of the session was **Bill Paven's** presentation on *Comprehensive analysis of the SRY-box 10 locus (SOX10): Implications for diseases of neural crest derivatives*. Dr. Paven presented an overview of his current research program at the NIH. Part of his laboratory is conducting a mutagenesis screen to identify genes, that when mutated, increase the severity of neural crest defects in a SOX10 mutant that models Waardenburg Syndrome type 4. Another part of his laboratory is looking to identify the transcriptional regulatory networks that both control SOX10 gene expression and are controlled by SOX10 protein levels.

Session 5; Genetics and Gene Expression

Chairs: Vijay Setaluri and Richard Spritz
by *Vijay Setaluri*

The session on Genetics and Gene Expression was chaired by Drs. Richard Spritz and Vijay Setaluri. Six abstracts of the total 10 abstracts submitted in this category were chosen for oral presentation. These presentations covered 3 areas- genetics of vitiligo, genetics of disease related iris phenotypes, genetic ap-

proaches to identify novel melanocyte genes and their regulation.

Dr. Spritz reviewed the published data on the recently identified vitiligo susceptibility gene *NALP1* on chromosome 17p and described the results of the additional studies that showed genetic association of *NALP1* with other vitiligo-associated autoimmune diseases. He also presented data that suggested the high-risk vitiligo-susceptibility *NALP1* genotype is associated with apoptosis defect in blood mononuclear cells. An additional candidate gene found to contribute to the disease risk is *PTPN22*. These studies illustrate the need for a multidisciplinary approach to unravel the genetics of vitiligo.

The next listed presentation in the Program on the role of microenvironment changes in vitiligo was cancelled due to conflict in Dr. Zhou's travels. The next presentation from Michael Anderson's group demonstrated the value of the utility of studying iris phenotypes as a means of identifying new candidate genes involved in human glaucoma. In an elegant presentation, **Colleen Trantow**, a graduate student in Dr. Anderson's laboratory, described her studies on previously unrecognized iris disease phenotypes in *beige* and *nm2798* mutant mice and the potential involvement of *beige* and *Dct* genes in massive accumulation of pigment and progressive pigment dispersion in iris, respectively.

In his second presentation, **Dr. Spritz** presented his studies on comprehensive genetic analysis of Caucasian patients with oculocutaneous albinism and autosomal recessive ocular albinism. Based on the relative frequencies of the three different types of OCA and the pathological gene mutations associated with these OCAs, Dr. Spritz proposed that OCA1 is the most frequent cause of OCA among Caucasians.

The last 2 presentations were from Dr. William Pavan's laboratory. In this presentation, described the identification of *Betelgeuse* a novel locus in mouse melanocyte development using a sensitized genome-wide ENU mutagenesis screen. This screen is designed to identify ENU induced mutation(s) in additional genes that act synergistically with *Sox10* to increase the melanocyte defects in *Sox10^{LacZ/+}* mice. This is a powerful screen that will provide a resource for identifying additional genes involved in a complex pathway during melanocyte development *in vivo*. In the last presentation of the session, **Dr. Pavan** presented his studies on a genomic approach to identify cis-acting transcrip-

tional regulatory elements using a combination of gene expression pattern studies, comparative genome sequencing and informatics analysis on selected 14 neural crest-expressed genes. Dr. Pavan illustrated the strategy of whole mount *in situ* hybridization in wild type and neural crest transcription factor mutant mouse embryos to identify key transcriptional regulatory elements contributing to the expression patterns followed by a search for elements that are evolutionarily conserved in 7 different species. The goal is to assess if correlations exist between conserved sequence elements and sub-classes of genes defined by expression indices generated by the whole mount analysis. Overall, this session was well received. The presentations elicited spirited discussion as evidenced by the number of people clustering around the speakers at the end of the session. The pace of research in this area is bound to produce new and exciting results that will enhance our understanding of the complex biology of melanocytes.

Session 6; Immunobiology of Melanocytic Cells

Chairs: Ping Yu, José Guevara-Patiño and Caroline Le Poole

by *Caroline Le Poole*

This session was spearheaded by **Dr. Rosalie Luiten**, who flew in from Amsterdam to show us the latest data from her group related to T cell mediated cytotoxicity in vitiligo. These data are indeed very exciting: Dr. Luiten was able to show in an 'ex vivo' system that CD8+ cytotoxic T cells from perilesional vitiligo skin will kill melanocytes in normally pigmented skin from the same donor. To do this, isolated T cells were co-cultured with tissue explants. Impressive tissue damage was observed when T cells were added in large numbers, and melanocyte apoptosis was evidenced by confocal imagery using antibodies to CD70, gp100 and caspase-3. Data using cloned T cells from a melanoma patient showed less extensive damage to melanocytes, in line with the theory that T cells from vitiligo patients are better able to kill their targets.

The second lecture came from **Dr. Gisela Erf**. This was a comprehensive overview of the impressive data demonstrating the parallels between autoimmune vitiligo in the Smyth line chicken, and progressive depigmentation in humans. The chicken remains the only true model of spontaneous autoimmune vitiligo. Importantly, from studies of this model we can predict relevant precipitating factors in vitiligo as, for example, there is a clear association between vaccination against

a herpes virus and the development of depigmentation. Data related to the genetic predisposition to depigmentation in this model may be expected in the near future.

The final presentation was by **Dr. Ping Yu**, also a co-chair for the session. She presented data related to immune infiltrates found in halo nevi compared to melanoma tumors. The immunohistology was stunning, and very convincing data were presented to show that halo nevi have similar percentages of Treg among CD4+ T cells (50%) as control skin, whereas fewer (!) FoxP3 + CD4 T cells were found in melanoma. The apparent discrepancy to the reduced clinical efficacy of an immune response in melanoma is best explained by the significantly reduced expression of perforin by infiltrating T cells in melanoma. The common denominator among the novel data presented is the active search into mechanisms (dys) regulating the immune response in pigmentary disorders.

Session 7; *Biochemistry and Signaling Pathways*
Chairs: John Pawelek, Raymond Boissy and Vincent Hearing
by John Pawelek

Andrzej Slominski began the session with a review on the role of CRH in the mammalian pigmentary system. Prof. Slominski's group has now demonstrated that the same endocrine systems operating in the "hypothalamic-pituitary-adrenal axis" are operative in the skin. Among the large number of biological functions affected by this is hormonal regulation of the pigmentary system.

Feng Liu presented her work with Frank Meyskens that contrary to previous reports by others, they found no significant differences in MITF expression by cultured melanocytes from black, Hispanic, and Caucasian origins, even though melanin levels varied more than 6 fold, and positively correlated with skin type. During discussion it was agreed that differences with previous reports may be attributed to differences in culture conditions.

Sulochana Devi presented work in collaboration with R Kedlaya, N Maddodi, CS Weber, H Valdivia, and V Setaluri, showing that in cultured human melanocytes melanostatin1/TRPM1-mediated Ca⁺⁺ uptake, which can be regulated by UV through p53, plays a role in growth and differentiation of melanocytes and melanoma tumor progression.

Akira Hachiya along with S Kasamatsu, K Higuchi, A Ohuchi, T Kitahara, and R Boissy, reported that the production of the soluble form of C-kit, S-kit, abolishes stem cell factor-induced melanogenesis in human melanocytes. The results suggested that the production of S-kit may contribute to the regulation of human skin pigmentation.

Ana Luisa Kadekaro along with N Mosby, E Hess, R Kavanagh, V Swope and Z Abdel-Malek, presented the most interesting story of alpha MSH protection of human melanocytes against UVB-induced oxidative stress, in this case through a mechanism that involves p53 modulation.

Ashley Dills with Beverly Delidow presented a convincing study that retinoic acid induces coordinate expression of Wnt inhibitory genes in melanoma. This is the first example of coordinate RA-regulation of these factors and suggests the potential of Wnt inhibition as a strategy for melanoma therapy.

Heinze Arnheiter completed the morning, and indeed the conference, with a keynote lecture on his exciting work regarding the role of MITF isoforms during pigment cell development.

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The Bibliography published in this issue covers the period December, 2005 through March, 2006. If you notice a paper that was not detected by this search that should be included, please send it to us and we will include it in the next issue. By its very nature, assignment of a reference to a particular category is arbitrary and we urge you to read through all categories to make sure you don't miss any pertinent to your field.

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