

PASPCR

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Newsletter



Introduction...

by *Bill Oetting*

The annual meeting of the PASPCR, held at Cape Cod, was a success! Jean Bologna, Ruth Halaban and John Pawelek did a great job of hosting the meeting and congratulations are in order. See the letter from our President for more laudatory statements. If you missed the meeting, in this issue you will find meeting reports for the different sessions. You can also see pictures taken at the meeting on the PASPCR web site. The link is near the top of the home page.

We are sad to report that on August 16, Thomas B. Fitzpatrick, M.D., Ph.D., passed away. Dr. Fitzpatrick had a major influence on the emergence of a new field of scientific inquiry, pigment cell research and this influence continues today. On page 4 we have included a letter written by Dr. John Parrish about the life of Dr. Fitzpatrick.

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 your contributions 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 can be found at:

<http://www.paspcr.org>

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 September 18 - 23, 2005 at the Natcher Conference Center at the National Institutes of Health in Bethesda, MD.

There is a possibility that the URLs for the PASPCR and IFPCS will be altered, due to a potential change in the web server. I do not know what the future URLs will be, but I will keep you informed. Some individuals have bookmarks that link directly to specific pages (such as the mouse coat color gene page). These will not work once the URL is altered. Remember, the PASPCR home page will always be available through the URL **paspcr.org** and the IFPCS home page will always be available using **ifpcs.org**.

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Richard A. King
past-President PASPCR

Calendar of Events:

Nov. 29-30, 2003 The 17th Annual Meeting of the Japanese Society for Pigment Cell Research (JSPCR)
Seminar House of Kao Corp., Japan
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July 22-24, 2004 International Skin Cancer Conference, Zurich, CH
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2004 XIIth Meeting of the PanAmerican Society for Pigment Cell Research, to be held in Irvine, California.
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2005 XIVth International Pigment Cell Conference (IPCC), to be held in Bethesda, MD, USA.
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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.

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Letter from the President

Our eleventh annual meeting in Cape Cod was met with enormous success thanks to the superb organization and the high caliber of the scientific presentations. Special thanks go to Jean Bolognia, John Pawelek, Ruth Halaban and their staff who made this meeting such a success. The highlight of the meeting was the most gracious reception hosted by Aaron and Millie Lerner. It was indeed a pleasure to see them, and we thank them for their tremendous hospitality. Interesting, this memorable reception coincided with the first PASPCR/Aaron B. Lerner lecture that was given (appropriately so) by Ruth Halaban in recognition of her outstanding contributions to pigment cell research. Congratulations to Ruth.

Of course, no meeting is successful without the attendees. It was great to see all the usual friendly faces, and even more delightful to meet new participants that we welcome warmly to our society. A very gratifying feeling is that many of the new participants expressed how much they enjoyed the scientific and social interactions and vowed to become regular attendants of our future annual meetings. Personally, I

look so much forward to our annual PASPCR meeting, not only to learn about the new breakthroughs in pigment cell research, but also because the meeting is a reunion for our growing family of PASPCR members. We are thrilled that our membership this year has grown significantly in number. Increasing our membership is a major goal that the officers and council members have set. I thank in particular our Secretary-Treasurer Raymond Boissy who put a tremendous effort to make the membership campaign so successful.

As a society, we pride ourselves for supporting young investigators. This year, we continued with the tradition of providing travel awards to young investigators who are members of PASPCR and presented at the meeting. Also, as in previous years, we awarded three young investigators awards to Prashiela Manga, Ph.D., a junior faculty member, Catherine Van Raamsdonk, a postdoctoral fellow, and to Yari Marin, graduate student. We also acknowledge and appreciate established scientists that have made a great impact on pigment cell research by providing them with honorary membership to PASPCR. This year, this award has a special meaning since it was given to one of the founders of our society and the first editor of PCR, Dr. Joseph Bagnara. Our most distinguished award is the Career Achievement Award, which was given this year to Dr. Richard King in recognition of his tremendous contributions to the understanding of the genetics of human pigmentary disorders, mainly oculocutaneous albinism.

What is remarkable is that after each annual meeting, we speak of the meeting as the best we ever had. This is an indication of the continued strive of our society to achieve excellence in science. It is most gratifying that our specialty is receiving the recognition it deserves from various disciplines of scientific research. The success of PASPCR is only achieved and will only continue through the success and contribution of its members. I urge you to support our society by participating in its various functions and activities, such as serving on its various committees, contributing to the newsletter, recruiting new members, and participating in its annual meetings. I look forward to seeing you in June, 2004 in Irvine, California. Until then, my best wishes to you for continued success and productivity.

Sincerely,

Your President Zalfa Abdel-Malek

**Thomas B. Fitzpatrick, M.D., Ph.D.
1919-2003**

Thomas B. Fitzpatrick, M.D., Ph.D., often proclaimed as one of the world's leading dermatologist, died on August 16 at his home at the age of 83. He was the Professor and Chairman of the Department of Dermatology at Harvard Medical School and Chief of the Massachusetts General Hospital Dermatology Service from 1959 to 1987. During 40 years at Harvard, Fitzpatrick trained many of today's top dermatologists in academia, industry and practices and is considered a father of modern academic dermatology.

Fitzpatrick graduated from the University of Wisconsin (AB), Harvard Medical School (M.D.) and University of Minnesota (Ph.D.). During World War II he served in The Army Chemical Center, where he met Aaron B. Lerner M.D., Ph.D. and began a historical collaboration to explain pigmentation of skin. Fitzpatrick was Professor and Head of Dermatology at the University of Oregon from 1952 to 1958 before being recruited to Harvard Medical School at age 39 as its youngest Professor and Chairman.

In 1971 Fitzpatrick published the first multiauthor dermatology textbook in the USA and served as organizer and senior editor for four subsequent editions of this seminal book about skin and skin disease for dermatologists and non-dermatologist physicians. He also organized a Color Atlas of Dermatology, a book on skin for lay persons, and ten other scientific books about specific aspects of Dermatology.

For over 50 years, Fitzpatrick was a major influence in basic and clinical research. His work resulted in over 250 original scientific publications. His fundamental observations and creative applications of knowledge markedly advanced the understanding of pigmentation, aging and disorders of the skin. Fitzpatrick described cutaneous markers of certain neurological disorders and many other markers and skin signs of systematic illness. He was one of the first to apply the electron microscopy to the study of the skin, and with colleagues discovered and named the melanosome, the basic subcellular organelle of pigmentation. He helped in defining and profiling the clinical and microscopic characteristics of early melanoma and advanced the cause of widespread screening to detect curable lesions. He and his colleagues invented oral psoralen photochemotherapy

(PUVA) a treatment of psoriasis which is now used worldwide to control a variety of disabling skin diseases. He organized scientific quantitative studies of sunscreens and championed their widespread use throughout the USA and Europe.

Fitzpatrick never removed himself from the front lines of patient care. He consulted and taught in both inpatient and outpatient settings and had an unrestricted, busy, non-super-specialized practice of general dermatology. Until weeks before his death, Dr. Fitzpatrick treated difficult problem cases referred from dermatologists all over the world, and his own very loyal patients, who included many rich and famous persons. He delivered primary care one on one, facing cosmetic blemishes and very sick patients with serious systemic illness. He consulted his textbook in front of patients, examined scrapings from scaly toes, performed biopsies, learned and tried new treatments, and looked at microscopic slides himself. He never refused to see any patient any time with any student or doctor.

Dr. Fitzpatrick was a member of more than 20 professional societies in the US and abroad. These include the prestigious American Academy of Arts and Science and the Institute of Medicine of the National Academy of Sciences. He was a founder of the Dermatology Foundation and served as president of the Society for Investigative Dermatology and served in leadership positions of many other academic organizations. Many of the personal qualities and specific scientific contributions that made Fitzpatrick such a powerful influence were recorded in a Festschrift issue of the Journal of Investigative Dermatology (80: 3s-5s, 1983).

Fitzpatrick was most honored and cherished as a teacher. Many medical students working under his supervision decided to become dermatologists. Dermatology residents were influenced for life by the content, approach and style of his care of patients. His comments at teaching conferences were absorbed with reverence; he invariably proposed a clever action plan to solve the most difficult medical riddles. The most powerful components of Fitzpatrick's teaching style were his encyclopedic knowledge, vast experience, relentless problem solving, and his compassionate individualization of treatment.

For his contribution to the training of Japanese dermatologists and his frequent travels to Japan to lecture and teach, he was awarded the prestigious

“Order of the Rising Sun” in 1987. An endowed chair at Harvard Medical School was established in his name in 1982, an honor seldom accorded to an incumbent professor.

Fitzpatrick’s most contagious traits were his childlike curiosity and his genuine joy of work, wonderful gifts that were spread to dermatologists throughout the world during his half-century of dedicated service. His leadership is measured not so much by the number of people he led but by the number of leaders he created.

Dr. Fitzpatrick is survived by his wife Beatrice Devaney Fitzpatrick and four children Thomas, Beatrice, Scott and Brian as well as three grandchildren.

In lieu of flowers please consider contributions to The Fitzpatrick Fund for Education c/o Danielle Wachs, MGH Development Office, 100 Charles River Plaza, Suite 600, Boston, MA 02114. Funeral services will be private. A memorial service will be held at The Memorial Church at Harvard Yard at Harvard University at 10 AM, October 21, 2003.

John Parrish

We are looking for:

items to include in the *PASPCR Newsletter*. Please send in information that you have on:

Meetings of interest
Reports on meetings you have attended
News about PASPCR members
Short stories
Interesting graphics

or anything else you think that the PASPCR membership would be interested in reading. Please email your contributions to bill@lenti.med.umn.edu.

Thanks!

Congratulations.....

...to those who were presented awards at the 11th Annual Meeting of the PASPCR, held in Cape Cod:

A **PASPCR Honorary Membership** was presented to **Dr. Joe Bagnara**.

The **Career Achievement Award** was presented to **Dr. Richard King**.

The **Aaron Lerner Lectureship**, Sponsored by the Johnson & Johnson Consumer Companies, Inc., was given by **Dr. Ruth Halaban**.

Young Investigator Awardees

Student

Yari E. Marin
Rutgers University
“Implication of NF-kappa B in mediating the transforming activity of Grm1 in mouse melanoma cells.”

Post-Doctoral

Cathy D. Van Raamsdonk
Stanford University
“G protein signaling expands the embryonic population of dermal melanocytes.”

Junior Faculty

Prashiela Manga
University of Cincinnati
“A role for Tyrp1 in determining melanocyte sensitivity to 4-(tert)butylphenol.”

Travel Awardees

Avner Ittah, Florida International University
Bianca Jefferson, Mississippi State University
Ana Luisa Kadekaro, University of Cincinnati
Valery Kempf, Duke University
Jin Namkoong, Rutgers University
Yari Marin, Rutgers University
Reena Rupani, Yale University
Alexander Sanokhvalou, Duke University
Deborah Spaulding, University of Oklahoma



Joe Bagnara after receiving his Honorary Membership at the 11th annual meeting held at Cape Cod.

A letter from Joe Bagnara

The recently held 11th Annual Meeting of the Pan American Society for Pigment Cell Research was an outstanding success and a real pleasure for me and my wife Lou. The fact that I was the recipient of the Honorary Member Award for 2003 was, of course, one of the highlights for me and I am exceedingly grateful to PASPCR for so honoring me. It is great to know that I am still remembered for whatever contributions I may have made over these many years. My sincere thanks go to all who considered and elected me as this year's awardee.

Another source of pleasure stemmed from reminiscences that arose from again being in Falmouth and Woods Hole. I would like to share some of these with our membership because they emphasize just how far we have come during the last 50 years of research on pigmentation. Some 48 years ago, as a third year graduate student, I was privileged to be accepted as a

student in the summer embryology course at the Marine Biology Laboratory (MBL) at Woods Hole. The laboratory where it was taught and the dormitory where I stayed are only a few hundred yards from Aaron Lerner's house where many of us just had the pleasure of the gracious hospitality of Aaron and Millie. As I sipped wine and nibbled on snacks my thoughts went back to that course in 1955 that was taught by several of the most eminent developmental biologists of the time. Among these were Mac Edds, Cliff Grobstein, Clem Markert, John Saunders, Nelson Spratt, J.P. (Trink) Trinkaus, and Ed Zwilling, names that unfortunately my not register with most who read this because it was a long time ago and one's time in the sun is ever so fleeting. I was enthralled by the course and the luminaries that it featured, and thus you can imagine how disappointed I was not to have been able to accept their invitation to spend the second half of the summer there on scholarship. Alas, I was a poor graduate student and I needed that time to finish my second foreign language requirement (necessary in those days), work on my dissertation research, and earn some money since Lou and I were to be married in September.

Despite the importance of the Woods Hole course, it seemed rather extravagant for me to spend the first half of the summer away from our lab in Iowa City where I had just sunk my teeth into my research project and especially in the light of my penurious state. Fortunately, my advisor, Professor Emil Witschi, came to my aid. He offered to pay for my Woods Hole experience in return for my harvesting hundreds and hundreds of shark pituitary glands that he wished to use for bioassay of gonadotropic hormonal content. This seemed especially appealing since I had discovered during the previous year that a hypophysial hormone seemed to control tadpole iridophores (then called guanophores). Thus, if I took with me to Woods Hole a few hundred of my hypophysectomized tadpoles, it would be possible to use them in the assay of the guanophore-stimulating capacity of pituitaries from sharks and other marine fish. So, off I went to Boston by train carrying with me two thermos jugs of hypophysectomized woodfrog tadpoles. When I arrived at the MBL, a bit earlier than the other students in the course, the teaching assistant, Roger Milkman (later quite a successful geneticist) helped me by finding a large aquarium for the tadpoles. He was much intrigued by their shimmering golden appearance brought about by the dispersed and iridescent iridophores. When the students and faculty arrived

and were curious about these strange creatures that were swimming in the tank, he explained that they were whale spermatozoa!

When it came time to do my work for Professor Witschi, I went to the MBL dock where a huge pile of freshly caught dogfish (small sharks, *Squalus* and *Mustelus*) awaited me. Here, surrounded by hungry sea gulls, I spent the whole day with a sharp blade in my hand, tearing through the cartilaginous brain case to dig out pituitary glands for preservation in ethanol. You should be reminded that at this time only a few of the hormones of the adenohypophysis had been even partially purified and the structure of ACTH was in its early stages of chemical elucidation. Aaron Lerner's group was then working on the structure of alpha-MSH, but there was still some doubt of its status as a true hormone. This remained so a little longer in light of its identity with the first 13 amino acids of ACTH. At the same time, the status of beta-MSH was being contemplated by other labs. At Woods Hole, I found that when tested on my hypophysectomized tadpoles, extracts of these dogfish pituitaries, as well as those of other fish, possessed profound melanophore-dispersing activities and moreover, they exerted an iridophore-contracting effect so powerful that in a day or two the iridophores disappeared from sight as their purine crystals were resorbed. During the next year or so, as alpha-MSH was attaining true hormonal status, I was able to show that this hormone affected both melanophores and iridophores and that molecular requirements for these effects were identical.

During these times, frogs were fundamental in studying basic problems in pigmentation as we were in the golden age of comparative pigment cell biology. At this time, the major focus was on the hormone as an active agent for the concept of hormone receptors had not yet really emerged. It was not until the current age of molecular genetics that receptor biology realized its present important state. So, some 50 years later, here we are, at the opposite end of the MSH story, the melanocortin receptors, and in particular, MC1R, the focus of so much of the research presented at this meeting. Work on cold-blooded vertebrate pigmentation has become passé, an exercise in the esoteric, a pursuit of knowledge for its own sake. Perhaps as the pendulum of time returns, in a stroke more rapid than 50 years, attention will again focus on lower vertebrate pigmentation and such problems as characterization of the amphibian melanophore melanocortin receptor will be solved in conjunction with answering the question of whether the putative melanophore inhibiting factor(s) (MIF) are the homologs or the analogs of mammalian agouti protein.

Let me end by extending my personal thanks to Aaron and Millie Lerner for opening their home to all of us and for providing such a gracious reception.

Sincerely,

Joe Bagnara



**Meeting Report
for the
11th Annual Meeting of the PASPCR**

Here is the report on the different sessions of the 11th Annual Meeting of the PASPCR, held at Cape Cod, MA. I would like to thank all of the authors for their contributions.

**Session #1, Melanocortin/MSH/agouti protein
by Zalfa Abdel-Malek**

The scientific program of the 11th Annual PASPCR meeting opened with the session on melanocortins/MSH/Agouti protein. The keynote speaker was **Greg Barsh** (Stanford Univ.) who presented on the “genetics and genomics of pigment type-switching”. In his presentation, Greg described the roles of α -MSH and Agouti signaling protein as antagonists that switch to eumelanin and pheomelanin synthesis, respectively. He also discussed the roles of *Attractin* and *Mahoganin* in regulating the signaling of Agouti via the Mc1r. Greg presented new data on coat color mutations in dogs. His new results ascribed dominant black coat color to a dominant allele of *Agouti*, variegation of black and yellow coat color to an allele of *Mc1r*, and the dark coat color of the German Shepherd pedigree to a loss-of-function of *Agouti*. The highlight of the presentation was that the Barsh laboratory might have discovered a new pigmentary gene that might be responsible for the dominant black color. By crossing Labrador and Greyhound, Greg and his group found that the dominant black mapped to a region of dog chromosome 16 that has not been known to regulate pigmentation. Future identification of this putative gene will extend the ever-growing list of pigmentary genes, and will shed further light on the complexity of regulation of eumelanin and pheomelanin synthesis in mammals.

Zalfa Abdel-Malek (Univ. of Cincinnati) presented on the impact of loss-of function mutations in the *MC1R* gene on the response of human melanocytes to UV radiation. Three human melanocyte cultures that were refractory to α -MSH exhibited a marked increase in apoptosis after UV exposure, compared to melanocytes with similar melanin content and functional *MC1R*. These three cultures were either homozygous or compound heterozygous for allelic variants of the *MC1R* that are known to be associated with red hair phenotype, poor tanning ability and increased risk for melanoma. Recently, her laboratory identified two additional cultures with loss-of-function *MC1R* and aberrant response to UV. New results obtained in the Abdel-Malek laboratory

revealed that α -MSH promotes the survival of human melanocytes following UV exposure by inhibiting apoptosis. The survival effect of α -MSH, however, was absent in melanocytes with loss-of-function *MC1R*. Increased apoptosis of the melanocytes is interpreted as an indication of inefficient repair of UV-induced DNA damage, which results in apoptosis or mutagenesis. The possibility that loss-of-function mutations in *MC1R* increase mutagenesis might explain why these mutations increase the risk for melanoma.

Francois Rouzaud (NIH) provided evidence for a new variant of *MC1R*, namely *MC1R* 350, which differs from the known *MC1R* 317 by an additional 33 amino acids in the carboxy terminal domain of the protein. This 350 variant results from alternative splicing of the open reading frame that leads to a longer *MC1R* mRNA. Expression of this variant by a subset of various human melanocyte cultures did not correlate with their pigmentation. Included in the additional 33 amino acids are 5 cysteine residues that are thought to confer stability to the receptor by anchoring it more firmly in the membrane. Results obtained from ¹²⁵I-labeled α -MSH and autoradiography suggested that *MC1R*, as other G protein coupled receptors, might exist as a dimer. The *MC1R* 350 variant adds another level of complexity of the *MC1R* and its function as a regulator of human pigmentation.

Catherine Van Raamsdonk (Stanford Univ.) reported on the role of Gq signaling in the regulation of dermal melanocytes. By investigating specific mouse mutants identified by dark skin (DSK), two dominant mutants, DSK1 and DSK 7 were found to cause an abnormal expansion of melanocytes in the dermis during early embryonic development. DSK1 and DSK2 resulted in missense alterations in the G alpha q genes *Gnaq* and *Gnal1*, respectively, and interacted in an additive manner. Mice that are null for the endothelin B receptor (that signals through Gq) and DSK7 mutant totally lacked pigment. Further investigation of the loss and gain of function mutations in G alpha q genes revealed that signaling through Gq determines the extent of dermal pigmentation. The effects of DSK7 on eumelanin synthesis were independent of *MC1R* signaling. The significance of this study is that it highlights a new signaling pathway through Gq that regulates dermal pigment cells and possibly contributes to known pigmentary abnormalities in humans, such as blue nevus or Mongolian spot.

**Session #2, Subcellular Organelles and Protein Trafficking
by Seth Orlow**

Dan Hebert (U Mass) presented work done in his laboratory and in collaboration with Ruth Halaban's lab

regarding the early maturation and processing of tyrosinase. Tyrosinase undergoes extensive glycosylation requiring an extended sojourn in the early secretory pathway, involving the participation of chaperones such as calnexin and Erp57. Disulphide bond formation and oligomerization are also involved. Mutations in tyrosinase and perhaps Tyrp-1 can both interfere with proper Tyr folding. John Hammer (NIH) reviewed the interactions of myosin Va with rab 27a and melanophilin. Myosin Va's interaction with melanosomes depends upon the tail sequences in the melanocytic splice form. Clearly rabGTPases and their interacting proteins play key role in the movement of vesicular cargo by molecular motors.

Dick Swank (Roswell Park) reviewed the 13 mouse loci associated with Hermansky-Pudlak syndrome (HPS)-like phenotypes. He and his collaborators have been able to reproduce the storage-disorder-like lung disease typically lacking in any of the mouse HPS homozygotes by creating double mutants, thus offering a tractable small animal model to understand the pathogenesis of the oft-fatal lung involvement and a means to test potential therapies. J.C. Valencia (Hearing lab, NIH) presented data on the complex processing of gp100. gp100 appears to be transported from the Golgi via the AP-1 adaptor complex, and is extremely highly represented in coated vesicle fractions from a human melanoma cell line MNT-1.

Reena Rupani (Yale) spoke on metastatic human melanomas, spontaneous nonmetastatic mouse melanoma and a constructed mouse melanoma x macrophage hybrid. The human melanoma and the hybrid each exhibited coarse melanin granules that have previously been observed in a very high percentage of human melanomas in tissue sections. These organelles contained the endolysosomal marker CD63, the melanosomal matrix protein gp100, and stained for the glycan recognized by the lectin LPHA. EM analysis is consistent with an autophagosomal origin for these organelles.

H. Watabe (NIH) presented data on aberrant tyrosinase processing in amelanotic SKMel28 cells. Misfolded Tyr is trapped in the ER and its levels can be increased via proteasomal inhibitors consistent with them being involved in degradation of the misfolded protein. Treatment of the cells with vacuolar proton pump inhibitors such as bafilomycin results in increased expression of Tyr in stage II melanosomes and Tyr exit from the ER, but the effects may be complex and may actually inhibit exit from the Golgi.

D. T. Spaulding (Univ Oklahoma) reviewed differences between Caucasian and Black melanocytes. Tyrosinase levels (8-10 fold higher in Black

melanocytes) exhibit similar levels of Tyr protein. However by use of acridine orange, fluorescence in Black melanocytes is much lower than in Caucasian, consistent with a much higher internal pH. Na/H antiporters may well be involved, as several are expressed in melanocytes, and amiloride suppresses pigmentation by Black melanocytes. One particular exchanger, NHE3, may be expressed at higher levels in Black melanocytes, suggesting that it plays a role in alkalinizing the melanosome.

Dick King (Univ. Minnesota) reviewed the question of tyrosinase gene mutations in patients presenting with congenital white hair. While in general those born with white hair are more likely to have OCA1, mutations in OCA2 are also a possibility and need to be looked for if a molecular diagnosis is sought.

Finally, **M. Endo** (Jimbow lab, Sapporo) reported upon investigations into the effects of Tyrp-1 on Tyr mediated cytotoxicity. While coexpression of Tyrp1 with Tyr can indeed increase tyrosinase activity and melanin production in both melanocytic and non-melanocytic cells, Tyrp1 can suppress the cytotoxicity of "unprotected" Tyr expression. This toxicity is apparently non-apoptotic in mechanism. Mutations in the domains of Tyrp1 homologous to Tyr's copper binding domains has more dramatic effects on Tyrp1's ability to stimulate pigmentation with little effect on its cytotoxicity-protective abilities. Conversely, missense Tyrp1 mutations had greater effects on cytotoxicity protection than on pigmentation. Deletions affected both. These data suggest that different sites on Tyrp1 may be responsible for the observed pigment enhancing vs. cytotoxicity protecting effects.

Session #3, Cellular Interactions and Signal Transduction *by Miri Seiberg*

Lynn Margulis (U Mass) discussed symbiogenesis, a cellular evolutionary process of acquisition of genomes by merger, which led to the creation of "new features" like cellular organelles. She claimed that the accumulation of mutations alone cannot lead to the generation of a new species, and is more likely to result in a sick or dead organism, while genome acquisitions could clearly result in the creation of new species. The talk was centered on the creation of the first eukaryotic, nucleated cell, from a merger between an eubacterium and an archaebacterium. The origin of the nucleus and its cytoskeletal system (nucleus + nuclear connector + motile structures like kinetosome and undulipodia) were discussed.

Natalie Ahn (Univ. of Colorado-Boulder) provided an overview on functional proteomics, and discussed the

pros and cons of two technical approaches, the 2D-PAGE/MS and the multidimensional LC/MS/MS methods. One example demonstrated how the Rho signaling pathway was followed with 2D gels, identifying posttranslational modifications involved in Rho-induced migrations. Another example described the search for melanoma markers for disease progression. Technical issues in peptide sequence assignment during data analysis were discussed. An improvement in the validation of peptide assignment was achieved through the integration of multiple search algorithms.

The first **Aaron B Lerner Honorary Lecture**, given by **Ruth Halaban**, (Yale) was titled "Tyrosinase: not only black and white". Ruth had reviewed the many activities of tyrosinase: catalysis of the production of melanin precursors, proton/pH sensing, self regulatory quality control, coupling of aromatic compounds, and possibly crosslinking. The role of DOPA in the maturation of tyrosinase, resulting in proper folding and movement from the ER to the Golgi was emphasized. Inactive or misfolded tyrosinase is retained in the ER of albino melanocytes and melanoma cells, resulting in proteolytic degradation and no pigment production. Inactive tyrosinase in albino cells was detected using Tyramide cross-linking. An increase in culture media pH could shift tyrosinase of melanoma cells to the Golgi, resulting in pigment production.

VJ Setaluri (Wake Forest) described the role of PI3 kinase and AKT2 signaling in melanosome biogenesis. Inhibition of PI3K blocked the sorting of newly synthesized Tyrp-1 into melanosomes. A yeast two hybrid screen identified a PDZ-domain protein, GIPC, which temporarily interacts with the newly synthesized Tyrp-1. GIPC binds APPL, which is an AKT2-binding protein, and a complex of these three proteins was identified. AKT2 phosphorylation was found stronger in non-pigmented melanocytes.

Ana Luisa Kadekaro (Univ. Cincinnati) discussed endothelin1 and alpha MSH as melanocyte survival factors following UV irradiation. An increase in viable cells, and a reduction in apoptotic cells, was demonstrated following treatment with either ET1 or alpha MSH. Forskolin could mimic the MSH effect, suggesting the involvement of cAMP in the survival mechanism. ET-R antagonists and MC1-R mutants abrogated the protective effects of their respective ligands, demonstrating that the UV-survival signaling involves these receptors. Melanin synthesis per se is not involved in the protective effect, as was documented by rescuing tyrosinase-negative, albino melanocytes, with these agents. The AKT/PKB pathway was shown to be involved in the process.

Session #4 Melanoma, Epidemiology, Vaccine, and Apoptosis *by John Pawelek*

Two lectures provided an overview on recent advances in the epidemiology of melanoma and novel drugs that target simultaneously apoptotic and survival pathways. **Dr. Marianne Berwick** (Memorial Sloan-Kettering Cancer Center) discussed new epidemiological data showing that the biological behavior of melanoma differs according to whether it developed under conditions of high or low exposure to UV irradiation. Intriguingly, prognosis of melanoma is better in high incidence populations than low incidence populations and improves in a population as incidence increases. This behavior likely reflects the fact that populations in areas with higher levels of UV have increased awareness of melanoma risk and so detect melanoma earlier, leading to better prognosis.

Dr. Maria Soengas (Univ. Michigan) presented the results of a study using the proteasome inhibitor Bortezomib (Velcade™, previously known as PS-341) as a putative candidate to bypass melanoma chemoresistance. Bortezomib induced massive cell death in 24 aggressive melanoma cell lines without compromising the viability of normal melanocytes. Biochemical studies indicated that Bortezomib activates both non-apoptotic and apoptotic programs in melanoma cells. Thus, casp-8, casp9, -3 and -7 were activated even in cells with low Apaf-1 and high survivin, Bcl-xL, Bcl-2, Mcl-1, FLIP or XIAP. Moreover, the cytotoxic effect of Bortezomib was also independent on the functional status of p53, p19, p16 and the Ras/BRAF/ERK pathway. Fluorescence-based tumor imaging techniques revealed a significant effect of Bortezomib *in vivo* (mouse xenografts), particularly in controlling melanoma metastasis. This effect was significantly enhanced by small molecule inhibitors of Bcl-xL.

The morning symposium then continued with three talks selected from the submitted abstracts. **Dr. V Alexeev** (Jefferson Medical College) discussed the role of activating mutations in the *c-kit* gene in the survival of mouse melanocytes and melanoma cells. Expression of a c-Kit mutant receptor did not induce features of transformation of the mouse melanocyte cell line Melan-C, either *in vitro* or *in vivo*, or increased apoptosis. Conversely, a constitutively active c-Kit receptor induced apoptosis in melanoma. These results highlight the divergent biology of normal and tumorigenic melanocytes.

Dr. Dorothy Bennett (St. George's Hospital Medical School, England) discussed the role of the Inkk4a-Arf locus in the generation of immortal mutant mouse

melanocyte cell lines. Melanocytes isolated from mice with one or two *Ink4a-Arf* copies deleted fail to senesce in culture and form immortal cell lines without going into crisis. Dr. Bennett has been able to generate 15 immortal mutant mouse melanocyte cell lines by crossing C57BL6/J mutant mice, carrying mutations for Hermandsky-Pudlak, MITF and others, with *Ink4a-ARF* null mice, and isolation and culture of epidermal melanocytes. Availability of such cell lines will be a useful tool for the biological characterization and in-depth molecular analysis of the respective phenotypes.

Finally, **Dr. K. Fitch** (Stanford Univ.) presented results from a large-scale ENU mouse mutagenesis project. One of the mutants analyzed (*Dsk5*) was an hypermorphic mutation of the *Egfr* gene. These animals display excess epidermal pigmentation in the footpads that progresses with age. Pigmentation is associated with hyperkeratosis and melanocytosis. Dr. Fitch postulated that the mutation likely affects keratinocytes and secondarily the melanocytes. The *Dsk5* mouse model could be used to better understand the pathological link between keratinocyte activation and melanocyte proliferation.

Session #5, Genetic and Comparative Pigment Cell Biology by Lynn Lamoreux

One of the rewarding aspects of pigment cell research is the breadth and depth of the resources that are available to us. Session # 5, Genetic and Comparative Pigment Cell Biology, sampled a range of animal systems, from human to insect, with special attention to the mouse, and including chickens and chameleons. Unfortunately mentioned only in passing were the fish models that carry so much potential for pigment cell biologists.

Seth Orlow (NYU School of Medicine) reviewed two "Multipass Membrane Proteins that Control Pigmentation". The *p* locus of mice encodes a 12 TM protein whose absence results in tyrosinase positive albinism, with much reduced pigmentation of eumelanin pigmentation of the coat and eyes, and also causes albinism in man. It seems the protein encoded at the *p* locus controls trafficking of tyrosinase, but it is not clear how this is accomplished. Dr. Orlow reported studies in yeast that suggest the protein may facilitate intracellular glutathione transport that might play a role in folding of cysteine-rich tyrosinase. The *underwhite* locus in the mouse encodes MATP, another 12 TM membrane. Mutation at this locus results in pale pigmentation of the hair coat of the mouse that seems superficially similar to that caused by pinkeye, but does not strongly affect eye pigmentation. The effect of the

mutation upon melanosome structure and melanization in mouse hair bulb melanocytes is extreme, and mutation in the human can also cause albinism.

This MATP mutation is known also in Medaka fish where it has been referred to as *b*, and also greatly reduces melanization on the body but not the eyes, and in humans as the AIM-1 locus, implicated in OCA4 albinism.

Yasu Tomita (Hokkaido University, Japan) reported the identification of several Japanese patients exhibiting "An Autosomal Dominant Oculocutaneous Albinism Caused by a Mutation in OCA4 Gene, AIM-1". He compared phenotypes and genotypes. Unlike other types of albinism, this exhibits a dominant negative phenotype in the patients examined.

Marjan Huising (NIH) reported "New Findings in Hermansky-Pudlak Syndrome", a disorder or biogenesis of lysosomes and related organelles, of course including melanocytes. She reviewed the six genes that are now identified to cause HPS in humans (HPS1 to HPS6) and described the protein complexes of which they are a part. These include the adaptor complex AP3 and six different protein complexes known as BLOCs (biogenesis of lysosome related complexes).

Emelia Costin (Texas A&M) spoke on the "Characterization of Two new Mouse Melanocyte Lines Carrying the Slaty and Slaty Light Mutations". The slaty locus encodes DOPAchrome tautomerase (*Dct*), an important regulatory enzyme that plays a pivotal role in the biosynthesis of melanin and the rapid metabolism of its toxic intermediates. In both of these cell lines, *Dct* activity was reduced compared with wild type cell lines. Confocal microscopy is being used to evaluate intracellular trafficking.

There were two reports of the effect of 4-TBP on melanocytes.

Prashiela Manga (Univ. Cincinnati) discussed "A Role of Tyrp1 in Determining Melanocyte Sensitivity to 4-(tert)-butylphenol", a chemical that can cause chemical leukoderma and vitiligo. Their research had previously shown that exposure to 4-TBP in culture is preferentially cytotoxic to melanocytes in culture and results in dose dependent initiation of apoptosis and that Tyrp1 expression increased the sensitivity of transfected cells to 4-TBP. This study compared melanocytes obtained from normal individuals with those from an individual with OCA3 (mutant Tyrp1) and concluded that upregulation of Tyrp1 expression increases sensitivity of melanocytes to 4TPB. Tyrp1 may utilize increased amounts of 4TBP as substrate and produce

toxic intermediates at concentrations that eventually cause melanocyte death.

Caroline Le Poole (Loyola University) concentrated on the relationship between stress proteins and apoptosis in “HSP70 and the Response of Melanocytes to 4-TBP”, using normal and immortalized melanocytes and fibroblasts. Overexpression of HSP70 enhances MHC Class I expression and sensitizes melanocytes to T-cell-mediated cytotoxicity. Exposure to 4-TPB sensitized melanocyte killing by HLA-matched, melanocyte reactive T cells. Thus it appears that exposure to 4-TBP elevates HSP70 expression by melanocytes which can elicit enhanced cellular immune responses to melanocytes, thus contributing to vitiligo.

Gisela Erf (Univ. Arkansas) used Smyth Chickens to study vitiligo at the organismal level, Evaluating environmental impact on “Circulating Melanocyte-Specific Auto-Antibodies and Feather Infiltrating Lymphocytes in young Smyth Line Chickens prior to Visible onset of Vitiligo”. Turkey herpesvirus (HVT) administration at hatch is associated with a high incidence of vitiligo in these chickens. Groups of chicks were separately inoculated with HVT or with control. Antibodies to the HVT were identified in feathers several weeks before the onset of vitiligo.

Beautiful pictures of chameleons accompanied **Randy Morrison's** (McDaniel College) report, “An Analysis of Skin Color in Panther Chameleons from Different Regions of Madagascar using Reflectance Spectrophotometry”. The colors are interesting both because of their creation upon the body of the animal, studied by wave-length spectra obtained using reflectance spectrophotometry, and because of the evolutionary implications of geographical distribution of the color forms in the wild. Data were analyzed in terms of hue, brightness, saturation. This approach offers the potential to correlate the spectral (noninvasive) data with ultrastructure of the multiple chromatophores that make up a complex and highly specialized phenotype.

Insects are one of the earliest models of pigment formation. **Manickam Sugumaran**, (U Mass) in “Molecular Interactions of Insect Phenol Oxidases”, described the significance of phenol oxidase to both the sclerotization pathway, or hardening of the exoskeleton, and the melanogenic pathway. Melanin biosynthesis is associated with three physiologically important processes in the insect: immune response, wound healing, and pigment formation. Thus the phenol oxidases in insects are required for a complexity of processes such that their absence is life threatening. To perform these multiple tasks, phenol oxidase

participates in a number of protein complexes, three of which were discussed.

Session #6, Melanoma, Genetics, Animal Models and Angiogenesis *by Vince Hearing*

The Melanoma, Genetics, Animal Models and Angiogenesis Session was held on Saturday afternoon at the PASPCR meeting. The turnout was excellent and there was ample discussion following each of the talks, which are summarized as follows:

Boris Bastian (Univ. of California-San Francisco) began the session discussing the use of array comparative genomic hybridization (CGH) to examine gene expression in nevi (with limited proliferation) and melanoma (with unlimited proliferation). His studies were aimed to characterize the genetic differences between those 2 populations, and they performed their analyses on microdissected paraffin-embedded tissue sections. In brief, they found that melanomas had many differently expressed genes while the nevi had relatively few. Cyclin D1 was a gene commonly up-regulated in the melanomas, and they found that amplifications of different genes varied by their type and location on the body, e.g. acral, mucosal and non-acral melanomas. Mutations in bRAF also varied significantly by melanoma type, but there was not a correlation in that expression and disease outcome/survival. They confirmed the results obtained with the array CGH by fluorescence in situ hybridization (FISH). Interestingly, they found aberrant melanocytes often 1 – 2 cm outside of the malignant area, and these are considered precursor cells of melanoma in situ, herein termed field cells. Bastian provided a mechanistic basis for these findings in relation to genetic hits from the environment (e.g. UV) and stated that wider margins might be necessary during surgical excision of melanoma outside of the standard excision margins.

Lynda Chin (Dana-Farber Cancer Center) then followed with a lecture about the genetically engineered mouse models for melanoma, and discussed the standard melanoma-signature mutations in CDK4, INK4a, bARF and Myc. They developed a model based on transgenic mice generated via the Ras gene linked to the tyrosinase promoter to obtain specific expression in melanocytes, and then they activated p16/INK4a or p19/ARF in knockout mice to induce melanoma-genesis. The question arose as to whether those tumors harbored secondary mutations, and they used the array CGH technique to assess that, and found that the answer was yes. That also found similar amplification regions as were found in the human melanoma studies discussed earlier by Bastian. The induced mouse tumors had similar histopathologic properties and characteristics

to human melanomas. UVB cooperates with ARF but not with p16 in melanoma-genesis, i.e. UVB targets the Rb pathway for melanoma promotion. When Ras was put on a tetracycline-inducible promoter, they found that continued expression of Ras was required for tumor growth, which reflects in part angiogenic factors secreted by these cells that is required for the continued development of the malignant cell population.

Juan Recio (George Washington University) then presented another mouse model for melanomas that was developed in Glenn Merlino's laboratory. In that model, hepatocyte growth factor (HGF)-transgenic mice were produced and then were exposed to UVB to induce tumorigenesis. Neonatal UVB exposure was strictly required to generate melanomas, which is consistent with observations of the role of UV on melanoma-genesis in humans (i.e. dependency on UV doses in early life). The question was posed as to what genetic mutations contributed to the development of malignant melanoma. They found that INK4a/ARF gene expression is lost in the mouse melanomas produced in this system, and this loss correlated well with the UV-induction of the tumors. Array CGH was used to show the loss of INK4a in mouse melanomas. These tumor lines are proving useful to dissect the cMet-signal pathway, and Recio described a pathway in which CD44 interacts with cMet to stimulate ERK1/2 to then stimulate Egr1 and proliferation. HGF activates ATF2 and CRE transcription factors. This model will be quite useful to further characterize the signaling pathways involved and also to elucidate mechanisms involved in UV-induced melanoma-genesis.

Y.E. Marin (Rutgers University) then presented the first of 2 papers from Suzie Chen's group on the role of Grm1 in mouse melanoma cells. She discussed the involvement of NFkappaB (a transcription factor frequently deregulated in many types of cancers) in Grm1 gene expression. Ectopic expression of Grm1 is sufficient for melanoma-genesis in mice (Grm1 is the receptor for glutamate). An important question is what are the cellular signaling events involved in this process. They created a transgenic albino mouse (termed TG-3) which then gave rise to amelanotic melanomas. NFkappaB is an excellent candidate, and they found that it was constitutively active in this system. It is normally regulated by IKK phosphorylation.

Suzie Chen (Rutgers University) then continued this theme, explaining that Grm1 is a G protein coupled receptor, and that its altered function results in melanomas in mice. They linked the Dct promoter to the Grm1 cDNA to allow for specific expression in melanocytes and generated a transgenic animal. They found that it worked through the phosphorylation of ERK1/2 and their next goals are to establish the

intermediate links between Grm1 and ERK1/2 that are important to this process.

Michael Detmar (Mass General Hospital) then presented another overview lecture on angiogenesis and lymphangiogenesis. Those 2 processes are induced by hypoxia and by hyperplasia of keratinocytes which stimulates release of VEGF (vascular endothelial growth factor) which in turn stimulates endothelial cell proliferation. He mentioned that PlGF (placental growth factor) was also a minor player in these processes but wouldn't be discussed further. VEGF can facilitate skin inflammation, which can be very persistent. VEGF-transgenic mice develop severe and persistent inflammation which eventually develops into skin lesions. This can be abrogated in thrombospondin 1 transgenic mice. The inflammatory response to UV can also be abrogated in those transgenic mice. Many therapies have been developed which are based on inhibiting angiogenesis to reduce melanoma outgrowth; those have been tested in mouse models and a phase I clinical trial is currently underway on melanoma patients. But is there a distinct relationship between lymphangiogenesis and melanoma? Actually there is no clear correlation between blood vessel size or number and metastatic or nonmetastatic melanomas. However, there is a large (~2-fold) difference in lymphatics between those 2 types of melanomas suggesting that lymphangiogenesis may play a more important role than angiogenesis. This turns out to be an excellent prognostic indicator.

John Pawelek (Yale) then presented his laboratory's work on coarse melanin as a specific indicator of malignancy and corroborating evidence of the hybrid melanoma theory. Coarse melanin occurs in virtually 100% of metastases and not so frequently in primary tumors. They used tissue microarray to show that LPHA staining is commonly positive in metastases of many types of tumors, not just melanomas. Pawelek also discussed a patient who had a bone marrow transplant and who subsequently developed renal cell carcinoma. The metastases in that patient had genetic markers not only for the patient but also for the donor, proving that at least in those metastatic tumor cells, hybridization of cells from both sources had occurred. For those interested, a paper will be published in the Sept 1 issue of Cancer Research detailing some of the above.

Toshihiko Hoashi (Tokyo Koseinenkin Hospital, Japan) then discussed their study on the expression of various MMPs (matrix metalloproteinases) and their inhibitors (TIMPs) in various types of melanomas. He described their expression in lentigo malignant melanoma (LMM), superficial spreading melanoma (SSM), acral lentiginous melanoma (ALM), nodular melanoma (NM) and desmoplastic melanoma (DM).

He reviewed the substrate specificity of the different MMPs, noting that MMP9 was particularly interesting because it degraded collagen type IV, a major component of the basement membrane and thus was an advantageous protease to permit metastasis. Many previous studies showed contrasting results on the expression of various MMPs and TIMPs in different types of melanomas, and Hoashi's study (which examined matched samples of primary and metastatic tumors from the same patients) found that in general there were no dramatic changes in the expression of any of the MMPs or TIMPs between primary and metastatic melanomas in the 16 patients they examined, although there were slight increases in MMP7 and TIMP1 in all lymph node metastases. However, MMP9 expression in DM was dramatically increased in all 6 metastatic tissues examined compared with the expression of MMP9 in primary tumors, suggesting an important role for MMP9 in the metastatic spread of those tumors.

Shosuke Ito (Kumamoto Univ. School of Medicine, Japan) then summarized his group's research on the presence and analysis of melanogenic intermediates in the serum and urine of melanoma patients. They found that eumelanin and pheomelanin intermediates are partially secreted from melanomas and are then methylated and can be detected in the serum and urine. They found an excellent correlation of 5-SCD content with the progression of disease. In serum, 4-AHP (4-aminohydroxyphenylalanine) is of comparable significance to the presence of 5-SCD (5-S-cysteinyldopa), both being markers of pheomelanin production. In sum, the latter compound is of great potential value in diagnosing the course of disease in melanoma patients.

Session #7, Transcriptional Regulation *by Ray Boissy*

The symposium entitled "Transcriptional Regulation" focused on the pioneer field of transcriptional regulation of the embryonic commitment of the melanocytes and their eventual survival. This is an exciting field because many new regulatory molecules/transcription factors have recently been identified and their coordinate interactions are now being elucidated.

The symposium began with a presentation entitled "Genomic analysis of neural crest-melanocyte development" by **William Pavan** (NIH). The expression and associated functions of SOX10, PAX3 and MITF during embryogenesis were presented. An analysis of their temporal expression in normal embryogenesis and specific alterations in various mutants of these transcription factors has led to their

proposed sequential interactive role in melanocyte commitment.

The elaborate role of MITF in signaling pathways of the melanocytes was reviewed by **David Fisher** (Dana-Farber Cancer Center). Two significant associations with MITF have recently been identified. The gene for an associated transcription factor TFEB, when translocated in papillary renal cell carcinomas, results in aberrant expression of several melanocytic antigens. In addition, a downstream target of MITF activation is the anti-apoptotic molecule Bcl2. Associated with mutation of MITF has been neurosensory deafness, as exemplified in Waardenburg syndrome type II or Tietz syndrome.

The lack of melanoblast development and the associated otic morphologic/physiologic defect in *Mitf^{Mi-Wh/+}* mice was presented by **Thomas Hornyak** (Henry Ford Hospital). "The overexpression of beta-catenin affects the proliferation and migration of melanoblasts" was presented by Lionel LaRue. Catenin appears to both mediate the interaction between cadherins and the cytoskeleton and to function as transcription factors. Aberrant expression of catenin in transgenic mice and melanoma cells demonstrated the role of this molecule in regulating proliferation and migration of melanoblasts.

Finally, the role of a unique transcription factor, FOXN1, in pigmentation was presented by **Loren Weiner** (Mass General Hospital, Harvard Univ.). Mutations of FOXN1 are responsible for the immunocompromised, hairless "nude" mouse. A transgenic mouse expressing FOXN1 in the basal epidermis via the keratin 5 promoter resulted in the development of interfollicular melanocytes. It was proposed that from the population of hair follicle destined melanoblast there was recruited cells that homed into the interfollicular epidermis via signaling cytokines regulated by FOXN1. This symposium clearly demonstrated the numerous intricate and interactive roles of known and unknown molecules regulating melanocyte development.



Positions - Wanted and 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.

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-02-20-2003.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

Postdoctoral Position

Polarized Kit-ligand expression in the epidermis: Its role in human melanocyte homeostasis

A postdoctoral position (fully funded for the first year with the possibility of a 2 year extension) is immediately available in the Department of Pathology, Centre Medical Universitaire at the University of Geneva, Switzerland. The project is supervised by Dr. Bernhard Wehrle-Haller and Prof. Beat Imhof and is within the frame of a collaboration between the University of Geneva and Industry.

The aim of this project is to understand the role of kit-ligand in melanocyte homeostasis in the adult

epidermis and how manipulation of kit-ligand expression or localization in keratinocytes affect melanocyte behavior. The project will employ cell-biological, pharmaceutical, biochemical as well transgenic approaches (mouse) to develop methods to modify Kit-ligand localization (polarity and cell surface expression) *in vivo* and to study melanocyte behavior in response to such altered Kit-ligand presentation. For references and rationale see Wehrle-Haller and Imhof (2001, *J. Biol. Chem.* 276, 12667-74) and Grichnik et al., (1998, *J. Invest. Dermatol.* 111, 233-38).

The Centre Medical Universitaire provides a stimulatory research environment located within the City of Geneva. Research in the department is centered around problems of autoimmunity, wound healing, inflammation, cell-cell junctions and cell migration. Geneva, located at the lake of Geneva in close proximity to the French Alps, provides a rich multicultural environment facilitating social integration.

Interested candidates preferably having experience in one or more of the aforementioned domains should send their CV (e.g. e-mail) including names and contacting information of two references to:

Bernhard Wehrle-Haller PhD
Department of Pathology
Centre Medical Universitaire
1. Rue Michel-Servet
1211 Geneva 4
Switzerland
Tel/Fax: 0041 22 702 5735 / 5746
[Bernhard.Wehrle](mailto:Bernhard.Wehrle@medecine.unige.ch)
Haller@medecine.unige.ch

Postdoctoral Research Position

A postdoctoral position is available immediately to study the transcriptional co-repressor and co-activator activities of the oncogenic protein Ski in human melanomas (PNAS (USA) 97:5924-5929, 2000). Seeking individuals with experience in EMSA, *in vitro* transcription-translation, site-directed mutagenesis and yeast two-hybrid screening. Interested individuals should send inquiries and applications (including CV, a brief description of past experience and future research interests, and the name of three references) to:

Estela E. Medrano, Ph.D.
Huffington Center on Aging
Baylor College of Medicine
One Baylor Plaza N-803.01
Houston, TX 77030

Baylor College of Medicine is an Equal Opportunity Employer

Research Associate/Post Doctoral Fellow Position Available

Position available for either an entry level postdoctoral fellow or a more senior research associate to study the molecular and cellular biology of the melanocyte in general and the pathophysiology of vitiligo in specific. The research project will focus globally on the role of survival factors and apoptotic regulators on the viability of melanocytes in the skin and in culture. In addition, the project will focus on the genetic and cellular susceptibility of melanocytes from patients with vitiligo to under apoptosis in response to various stimuli. Postdoctoral fellow candidate should have experience with routine molecular and cellular techniques including cell culturing, site directed mutagenesis, and protein biochemistry. Research Associate candidate should have similar experiences utilizing the melanocyte system. Candidate will become part of an interactive research group focusing of various aspects of pigmentation in the Department of Dermatology and on skin physiology in the Skin Sciences Institute within the University of Cincinnati College of Medicine. Send curriculum vitae and list of three references to:

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Postdoctoral Fellows - Cancer and Developmental

Biology - Two NIH-funded positions are available for fellows interested in studying the Hedgehog signaling pathway in development and disease using skin as a model system. One project centers on defining the function of the Hedgehog pathway

during skin appendage morphogenesis (*Dev. Biol.* 205: 1-9, 1999); a second project focuses on understanding how deregulated activation of this pathway gives rise to basal cell carcinomas (*Nature Genet.* 24: 216-7, 2000). Applicants should have a solid background in molecular and cell biology, with experience in transgenic animal models desirable but not required. Interested individuals should send a CV, letter of interest, and names of three references to: Dr. Andrzej Dlugosz, University of Michigan, Department of Dermatology and Comprehensive Cancer Center, 3310 CCGC, Box 0932, 1500 East Medical Center Drive, Ann Arbor, MI 48109-0932 Email: dlugosza@umich.edu. The University of Michigan is an Equal Opportunity Employer.

Postdoctoral Fellow - Postdoctoral positions are available immediately to study post-embryonic development in zebrafish. NIH-funded research is aimed at identifying the genetic and cellular bases for development of the adult pigment pattern and somatic metamorphosis. The lab uses a wide variety of methods including genetic screening, genetic mapping and positional cloning, gene expression analysis, cell transplantation and classical histology. Postdoctoral fellows would be expected ultimately to develop independent research programs and would have the opportunity to participate in ongoing genetic screens for mutants affecting post-embryonic development.

For more information see:

<http://www.biosci.utexas.edu/IB/faculty/parichy/research.htm>

<http://www.biosci.utexas.edu/IB/faculty/parichy/pubs.html>

Applications including CV and contact information for three references should be sent to:

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The Bibliography published in this issue covers the period June, 2003 through August, 2003. 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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