Introduction...
by Bill Oetting

The organizer and location for the 2004 PASPCR Annual Meeting has now been decided. The 2004 meeting will take place at Irving California. Frank Myskins will be our host for this meeting in California. There are no specifics yet, but considering how successful the IPCC meeting was at Anaheim in 1996, this meeting will be one to look forward to. As more information becomes available, I will include it in the PASPCR Newsletter.

The 2003 meeting in Cape Cod is also going in to final preparations and the web site should be on-line soon.

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. The PASPCR Web site is not only includes new information, but it is also a repository on the history of our society, our goals and our bylaws. This site also includes a registration page for potential new members and I have received well over 30 requests over the last two years. This is a very cost effective means to disseminate information about 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. A visit to our web site may be the first time that an individual has heard about the PASPCR, and we should present ourselves in the best possible light. I look forward to your comments and inclusions.

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”. We also want to note any change of affiliation or address that you may have had to help us keep our membership list up-to-date. 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.

New IFPCS domain name

The IFPCS web site can now be reached by using either www.ifpcs.org or www.ifpcs.info. The domain name ifpcs.org will become the official domain name for the IFPCS web site and the ifpcs.info name will be eventually dropped when its registration expires. The domain name ipcc.info will soon contain information on the 19th meeting of the IFPCS.

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The PASPCR Web Site can be found at:
http://www.paspcr.org
The PanAmerican Society for Pigment Cell Research

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Calendar of Events:

Dec. 14-18, 2002 The American Society for Cell Biology 42nd Annual Meeting is to be held in San Francisco, CA.
For more information: www.ascb.org

Sept. 3-7, 2003 XIth Annual Meeting of the Pan-American Society for Pigment Cell Research, to be held in Cape Cod, MA.
Contact: Dr. Jean Bolognia.
E-mail: jean_bolognia@qm.yale.edu.

Sept. 17-20, 2003 XIIth Annual Meeting of the European Society for Pigment Cell Research, to be held in Gent, Belgium.
Contact: Prof. JeanMarie Naeyaert.
E-mail: JeanMarie.Naeyaert@rug.ac.be.

2004 XIIth Meeting of the PanAmerican Society for Pigment Cell Research, to be held in Irvine, California.
Contact: Frank Myskins.

2004 XIIth Meeting of the European Society for Pigment Cell Research, to be held in Paris, France.
Contact: Dr. Lionel LaRue
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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.

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**SILVER Corporate Patrons**
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We are looking for:

Items to add to the *PASPCR Newsletter*. Please send 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!

**XIth Annual Meeting of the PASPCR**  
*by John Pawelek*

Mark your calendars for the XIth annual meeting of the PASPCR, “Molecular and Genetic Characterization of Melanocytes and Melanoma Cells”, Sept. 4-7, 2003, Sea Crest Inn, Cape Cod Bay, North Falmouth, MA. An exciting slate of overview speakers is on tap for the program, which will be arranged from selected abstracts. Registration and abstract forms will be mailed to members and will be on the website in early 2003.

Organizing Committee: Jean Bolognia, Ruth Halaban, John Pawelek

From the Sea Crest Inn Website:

The Sea Crest Inn is the perfect romantic get-away for couples. Located in a quiet part of Cape May over looking one of the largest beaches on the island and close to the shopping area, fine restaurants and historical homes.

With features such as Cape May’s newest ocean front suites with fully equipped kitchens, the only outdoor whirlpool in Cape May and extra large balconies, why stay anywhere else.
And now for the rest of the story.

Richard Swank (with Ed Novak) have been instrumental to our identification and understanding of genes that affect the synthesis and trafficking of intracellular vesicles, including melanocytes. Here is the story of the beginnings of this very important area of pigment cell research.

On the Trail of Mouse Pigment mutants – more than pretty colors

Richard Swank, Ph.D.

The impetus for our initial forays (in approximately 1976) into the analysis of mouse pigment mutants was the desire to identify mouse genes which affected the biogenesis and/or function of lysosomes. There had been prior descriptions of mutations in both human and animal models, which cause loss of expression of individual lysosomal enzymes. However, there were very few examples of mutations which affected the trafficking of vesicles to lysosomes or the intracellular movement of lysosomes within cells as, for example, during secretion of lysosomes. This is where the melanosome and the wealth of mouse pigmentation mutants came to the rescue. There was one example, noted as early as 1967 by M. Lutzner et al, of a mammalian pigment mutant gene which also produced abnormalities in lysosomes. That was the beige pigment mutant, a model for Chediak-Higashi syndrome, with its diagnostic giant lysosomes. Further, in 1976, J. Holland documented a deficiency of serotonin within platelet dense granules of the beige mouse.

Inspired by this example, we decided to search among the large class of naturally-occurring mouse pigment mutations for abnormalities of lysosomal secretion. Ed Novak, a long-time dedicated research associate, and I fairly quickly determined that several mouse hypopigmentation mutants displayed greatly depressed secretion into urine of lysosomal enzymes from proximal tubule cells of the kidney of testosterone-treated mice (a system whose physiological significance is still debated, but whose 20-50 fold induction of lysosomal enzyme synthesis and secretion is a wonderful experimental advantage). Soon thereafter we found that, similar to the beige example, these mutants had striking abnormalities in another subcellular organelle, the platelet dense granule. Loss of the platelet dense granule constituents serotonin, adenine nucleotides and calcium produced nonfunctional platelets leading in turn to prolonged bleeding times. The platelet abnormality was the clue that the mouse hypopigmentation mutants were appropriate models for the inherited human disease, Hermansky-Pudlak Syndrome (HPS), which presents with hypopigmentation, prolonged bleeding times and fibrotic lung disease due to defects in melanosomes, platelet dense granules and lysosomes respectively.

With the above promising results in hand, we decided to perform a more systematic analysis for prolonged bleeding times in available mouse pigment mutations. In this endeavor we are of course indebted to the legions of mouse geneticists who over >100 years identified many color variants in their mouse colonies, transferred them to common inbred strain backgrounds, usually C57BL/6, and mapped them to approximate positions on chromosomes. We are also highly indebted to several staff investigators at The Jackson Laboratory, particularly Muriel Davisson, who served as hosts while we tested individual coat color mutants in their vast research collection (and simultaneously enjoyed the tremendous natural attributes of Mount Desert Island).

Over many years, as the shipments of new pigment mutant mice arrived from the Jackson Laboratory, we determined that a surprisingly high proportion (15 of 40 hypopigmentation mutants) had platelet storage pool deficiency, usually accompanied by hyposecretion of lysosomal enzymes. These results established the genetic relatedness of this group of 3 organelles, suggesting that they share multiple steps in their biogenesis/processing and that, as in yeast, many genes regulate vesicle trafficking in mammals. These suggestions
were supported by the experiments of Seth Orlow and others who produced convincing biochemical and cell biological evidence for the relatedness of melanosomes and lysosomes.

The most fun part of these experiments was the discovery phase. Every time a new box of mutants arrived at the lab it was like Christmas all over again since there was the possibility of the discovery of another gene which affected multiple subcellular organelles. Admittedly, I have a very high preference for discovery over hypothesis driven research. Clearly both approaches are necessary for progress, but for me discovery is just more fun. To paraphrase that ultimate discovery-driven researcher, Craig Venter – who, when criticized that he ignored hypothesis driven research, replied “My hypothesis is that we know almost nothing.” Over many years innumerable graduate and summer students and postdocs, with Ed Novak always in the background, participated in this fun. My own son Doug visited the lab one day to see how one performs bleeding times on mice and promptly found prolonged bleeding times in strain RIIIS/J. This discovery eventually led to characterization of this strain as a model for the most common type of von Willebrand Disease in humans.

While the finding of platelet dense granule and lysosomal defects in the mouse hypopigmentation mutants was gratifying, an obvious unknown in all these mutants was the nature of the genes mutated in these naturally occurring mutants. Since I am a biochemist by training, we first tried, over several years, a host of biochemical approaches, including 2-D separation of proteins, to identify missing or altered gene products - all ultimately unsuccessful. Fortunately, we reside in a mouse genetics department at Roswell Park. Our genetic colleagues suggested very early on that we should attempt to identify the mouse HPS genes by positional cloning. We were initially rather skeptical as we knew that the successful early positional cloning projects had required large laboratories working many years. However, we also knew that the technologies of positional/candidate cloning were constantly improving. Another important factor was that Roswell Park was and remains an ideal place to undertake these studies because of the availability of expert mouse geneticists such as Rosemary Elliott, Ken Gross, Bill Held, Ken Manly and (prior to his untimely death in 1995) Verne Chapman. Also, Roswell was a good place to produce the large (>1000 animal) backcross experiments required to construct the required high-resolution genetic maps for positional cloning because mouse costs were relatively cheap compared to most institutions.

So, many large interspecific mouse backcrosses and constructions of high-resolution genetic maps were completed over the last 10 years. Together with many talented collaborators, especially Richard Spritz, these genetic maps have led to the identification of a significant number of mouse HPS genes. On average these positional cloning projects, including the mapping and the associated molecular approaches have required about 4 years per identification. The availability of the complete mouse and human genome sequences has reduced this time to about 1.5-2 years.

The 11 mouse HPS genes cloned by us and others fall into two classes. The first class includes genes well known to encode protein regulators of intracellular vesicle trafficking. This makes perfect sense given the organelle abnormalities of HPS. These genes include the beta (pearl) and delta (mocha) subunits of the AP-3 adaptor complex, the alpha subunit of the Rab geranylgeranyl transferase gene (gunmetal) and Rab27a (ashen). The second class is composed of 7 novel genes including the pale ear, pallid, cocoa, light ear, muted, ruby-eye and ruby-eye-2 genes. This is a very interesting class. First, the fact that they are novel means that the eventual elucidation of their mechanisms of action will reveal new mechanisms by which the synthesis of these important mammalian subcellular organelles are controlled. Second, these genes are not found in lower eukaryotes such as yeast. This indicates that a novel class of genes has evolved to regulate the synthesis of very specialized mammalian subcellular organelles such as platelet dense granules and melanosomes.
The other very interesting, and still actively evolving, feature of these genes is the accumulating evidence from many laboratories that they likely affect other several other organelles and many mammalian tissues in addition to those mentioned above. For example, some HPS mutants are hyperactive, probably from an affect on selected brain synaptic vesicles. Others have balance problems, likely due to abnormal vesicle trafficking to inner ear otoliths. The ashen and gunmetal mice are defective in secretion of granules of cyto-toxic T-Cells. Pearl mice have night blindness. Other studies have implicated some of these genes in antigen presentation. So the effects originally observed on melanosomes in these mutants have expanded into many areas of biology. These results are likely the tip of the iceberg and are in fact not unexpected given the ubiquitous tissue expression of almost all HPS genes.

Our long-range hope in all these studies has been that they will eventually lead to improved therapies for HPS, which currently is treated only symptomatically. Together with our long time colleague Michael McGarry, we have made the gratifying observation that the bleeding abnormality in all the HPS mice is corrected by bone marrow transplantation. However, other severe complications of HPS, including the fatal lung disorder, remain uncurable. Hopefully, HPS gene identifications will ultimately contribute to therapy of these complications, either directly through gene therapy or through drug targeting of downstream protein targets.

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Positions Wanted and Available

The PASPCR Web Site and the PASPCR Newsletter contains positions wanted and available related to pigment cell research. This information is presented as a service to the members of the Pan-American Society for Pigment Cell Research (PASPCR).

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 and Available can be seen on page 15 of this Newsletter.

Image from Bill Pavan, N.I.H.
The First Annual  
Melanoma Research Congress

The congress intends to address most major issues in melanoma, from molecular genetics, basic biology and epidemiology to new approaches to therapy. It will bring together an international forum of speakers who will present the tremendous progress that has been made in recent years. The congress should also be of interest to the outsiders in the field who have an interest in melanoma.

Meenhard Herlyn

Wyndham Philadelphia at Franklin Plaza  
17th & Race Streets, Philadelphia, PA 19103  
(Phone: 215-448-2000; Fax: 215-448-2864)  
Sponsored by the Foundation for Melanoma Research

Conference Co-Chairs

Meenhard Herlyn  
The Wistar Institute  
Philadelphia, PA

DuPont Guerry  
University of Pennsylvania  
Philadelphia, PA

A comprehensive international congress on all fields of melanoma research with over 70 invited speakers

Symposia
1. Immunotherapy of Melanoma
2. Molecular Epidemiology of Melanoma
3. Biology of Melanoma
4. Molecular Biology of Melanoma
5. Tumor Microenvironment

Concurrent Sessions
1. Experimental Melanocyte Transformation  
2. Biology of Melanoma Metastasis
3. Genetics of Familial Melanoma  
4. Melanocyte Development & Melanoma
4. Immunobiology I  
5. UV, Pigmentation, and Melanoma
7. Immunobiology II  
6. Biomarkers
9. Tumor Progression  
10. Advocates and Melanoma Research
11. Genomics and Proteomics of Melanoma  
12. Selected Poster Presentations
13. Melanoma Staging and Sentinel Lymphnodes
14. Apoptosis and Differentiation in Melanoma

Poster Sessions
A. Immunobiology and Immunotherapy of Melanoma
B. Etiology, Biology, Prevention, and Novel Therapies of Melanoma

Request information: First Annual Congress for Melanoma Research, c/o Sandy Parson, The Wistar Institute, 3601 Spruce Street, Philadelphia, PA 19104, Tel. 215 898 3959, Fax: 215 898 0980, E-mail: Parsons@Wistar.upenn.edu
First Melanoma Research Congress

June 21-24, 2003

Saturday, June 21

3:00 pm to 10:00 pm  REGISTRATION

7:00 pm to 9:00 pm  KEYNOTE ADDRESSES

Steven A. Rosenberg, National Cancer Institute, Bethesda, MD
"Rational Immunotherapy of Melanoma"

Ronald A. DePinho, Dana Farber Cancer Institute, Boston, MA
"Modeling Cancer and Cancer Genomics"

9:00 pm to 10:00 pm  OPENING RECEPTION

Sunday, June 22

8:00 am to 10:30 am  Plenary Session 1, Immunotherapy of Melanoma
      Giorgio Parmiani, Chair

Pramod K. Srivastava, University of Connecticut School of Medicine, Farmington, CT
"Developing New Targets for Melanoma Vaccines"

Pedro Romero, Ludwig Institute of Cancer Research, Lausanne, Switzerland
"Peptide-Based Therapeutic Vaccines for Melanoma: Monitoring Specific T Cell Responses"

Jeffrey S. Weber, Norris Cancer Center, USC, Los Angeles, CA
"Novel Adjuvants and Delivery Systems for Melanoma Peptide Vaccines"

Giorgio Parmiani, Instituto Nazionale Tumori, Milan, Italy
"Vaccination of Melanoma Patients with Autologous Heat Shock Proteins 96"

Soldano Ferrone, Roswell Park Cancer Institute, Buffalo, NY
"Escape Mechanisms and T Cell Based Immunotherapy of Melanoma"

10:30 am to 11:00 am  Break

11:00 am to 12:30 pm  Concurrent Session 1, Experimental Melanocyte Transformation

Introduction: Estela Medrano, Baylor College of Medicine, Houston, TX
"The Ski Protein: A Key Regulator of Melanoma Tumor Progression"

Carola Berking, Ludwig-Maximilians University, Munich, Germany
"Induction of Melanoma in Human Skin by Synergistic Effects of Growth Factors and UVB Radiation"
Maria Soengas, University of Michigan Comprehensive Cancer Center, Ann Arbor, MI
“Apoptotic Factors in Melanoma Progression and Drug Response”

Glenn Merlino, National Cancer Institute, Bethesda, MD
“Modeling Gene-Environment Interactions in Malignant Melanoma”

Concurrent Session 2, Genetics of Familial Melanoma

Introduction: David Hogg, University of Toronto, Canada
“Search for Novel interacting Partners of p16”

Alisa M. Goldstein, National Cancer Institute, Bethesda, MD
“Prediction and Modification of Melanoma Risk”

Nicholas K. Hayward, Queensland Institute of Medical Research, Brisbane, Australia
“High Risk, Low Risk and Modifier Genes for Melanoma”

Julia A. Newton-Bishop, St. James University Hospital, Leeds, UK
“The Management of Patients with a Family History of Melanoma”

Concurrent Session 3, Immunobiology I

Introduction: Dorothee Herlyn, The Wistar Institute, Philadelphia
“T Cells in Organotypic Melanoma Culture Systems”

Andrea Anichini, Instituto Nazionale Tumori, Milan, Italy
“Maturation of Antigen-specific CD8+ T Lymphocytes at Tumor Site”

Nick Restifo, National Cancer Institute, Bethesda, MD
“In Vivo Requirements for Destruction of Large Established Tumors by Self-Specific CD8+ T Cells”

Yutaka Kawakami, Keio University School of Medicine, Tokyo, Japan
“Identification of Human Melanoma Antigens with Genetic and Immunological Methods”

12:30 pm to 2:00 pm
Break

2:00 pm to 4:00 pm
Plenary Session 2, Molecular Epidemiology of Melanoma
Marianne Berwick, Chair

Margaret A. Tucker, National Cancer Institute, Bethesda, MD
“Host Factors and Risk of Melanoma”

Timothy Rebbeck, University of Pennsylvania School of Medicine, Philadelphia, PA
“Pigmentation Genotypes and Phenotypes in Melanoma Etiology”

Marianne Berwick, Memorial Sloan-Kettering Cancer Center, New York, NY
“Gene-Environment Interactions in Melanoma: New Data”

Richard P. Gallagher, BC Cancer Agency, Vancouver, Canada
“Sunscreens, Cancer and Precursor Lesions: What Can Sunscreens Prevent and What Can’t They?”
Adele Green, Queensland Institute of Medical Research, Brisbane, Australia
“How Can Molecular Studies Assist in Melanoma control?”

4:00 pm to 4:30 pm  
**Break**

4:30 pm to 6:00 pm  
**Concurrent Session 4, Immunobiology II**

Introduction: Jürgen Becker, University of Würzburg, Germany
“Shift from Systemic to Site Specific Immunity by Tumor-targeted Interleukin2”

Ingegerd Hellström, Pacific Northwest Research Institute, Seattle, WA
“Approaches to Vaccination for Melanoma”

Walter J. Storkus, University of Pittsburgh School of Medicine, PA
“Repolarizing Anti-Melanoma Th Responses to Melanoma”

Francesco Marincola, National Cancer Institute, Bethesda, MD
“A Dynamic Picture of the Tumor Microenvironment”

**Concurrent Session 5, Tumor Progression**

Introduction: David Elder, Hospital of University of Pennsylvania, Philadelphia, PA
“Overview of Tumor Progression in Melanoma”

Allan Halpern, Memorial Sloan-Kettering Cancer Center, New York,
“Confocal Microscopy for Melanoma Diagnosis”

Dorothea Becker, University of Pittsburgh, Pittsburgh, PA
“Molecular and Optical Imaging Analysis of Atypical Nevi and Melanoma”

Menashe Bar-Eli, MD Anderson Cancer Center, Houston, TX
“Role and Regulation of PAR-1 in Melanoma Progression”

**Concurrent Session 6, Genomics and Proteomics of Melanoma**

Introduction: Barbara Weber, University of Pennsylvania, Philadelphia, PA
“Genomic Approaches to Gene Discovery in Melanoma”

David Speicher, The Wistar Institute, Philadelphia, PA
“Analysis of Melanoma Progression and Identification of Serological Markers Using Global Protein Profiling”

Katheryn Resing, University of Colorado, Boulder, CO
“Proteomics of Melanoma Cell Lines and Cultured Melanocytes”

Mark Nelson, Arizona Cancer Center, Tucson, AZ
“Gene Amplification in Melanoma”

8:00 pm to 10:00 pm  
**Poster Session A, Immunobiology and Immunotherapy of Melanoma**
Monday, June 23

8:00 am to 10:00 am  
**Plenary Session 3, Biology of Melanoma**
Dorothea Becker, Chair, University of Pittsburgh

Meenhard Herlyn, The Wistar Institute, Philadelphia, PA  
“The Dynamic Roles of Cell Surface Receptors in Melanoma Development”

Ze’ev Ronai, Mount Sinai School of Medicine, New York, NY  
“Transcriptional Switch in the Regulation of Apoptosis in Melanoma”

Ruth Halaban, Yale University School of Medicine, New Haven, CT  
“Suppression of pRb Function in Melanomas”

Lynda Chin, Harvard Medical School, Boston, MA  
“The Biology and Genetics of Melanoma”

Jeffrey M. Trent, National Human Genome Research Institute, Bethesda, MD  
“Genetic-Based Genomic Dissection of Melanoma”

10:00 am to 10:30 am  
**Break**

11:00 am to 12:30 pm  
**Concurrent Session 7, Biomarkers**

Introduction: Dirk J. Ruiter, UMC St. Radboud, The Netherlands  
“Biomarkers in Melanoma. What’s New?”

David Hoon, John Wayne Cancer Institute, Santa Monica, CA  
“Clinical Utility of Molecular Markers (RNA/DNA) as Prognostic Indicators of Malignant Melanoma Disease Outcome”

Anja Bosserhoff, University of Regensburg, Germany  
“Role of MIA in Early Development of Malignant Melanoma”

DuPont Guerry, IV, University of Pennsylvania, Philadelphia, PA  
“Integration of Immunohistological Biomarkers into More Effective and Efficient Prognostic Models”

**Concurrent Session 8, UV, Pigmentation, and Melanoma**

Introduction: Vincent J. Hearing, National Cancer Institute, NIH, Bethesda, MD  
“Overview of UV Effects on Melanocyte Function”

Dorothy Bennett, St. George’s Hospital Medical School, London, UK  
“Familial Melanoma Genes, Melanocyte Immortalization and Melanoma Initiation”

Rick Sturm, The University of Queensland, Brisbane, Australia  
“Human Pigmentation and Melanoma: MC1R Genotype, Pheno type and Population Genetics”

Thomas R. Fears, National Cancer Institute, Bethesda, MD  
“Individual UV Exposure and Melanoma Risk”

Frances Noonan, George Washington University, Washington, DC  
“Photobiology of Melanoma in the HGF/SF Transgenic Mouse – UVA or UVB?”
Concurrent Session 9, Bioinformatics for Genomics and Proteomics

Introduction: Tovia Libermann, Harvard Institutes of Medicine, Boston, MD
“Functional Genomics of Human Cancer”

Phyllis Gimotty, University of Pennsylvania, Philadelphia, PA
“A Biostatistical Perspective on the analysis of Gen Expression Data in Melanoma”

Louise Showe, The Wistar Institute, Philadelphia, PA
“Gene Expression Profiling”

12:30 pm to 2:00 pm Break

2:00 pm to 4:00 pm Plenary Session 4, Molecular Biology of Melanoma
Chairperson: Meenhard Herlyn, The Wistar Institute, Philadelphia, PA

Richard Wooster, The Wellcome Trust Sanger Institute, Hinxton, Cambridge, Great Britain
“The Cancer Genome Project - Systematic Cancer Gene Discovery”

Kapaettu Satyamoorthy, Center for Cellular & Molecular Biology, Manipal, India
“Molecular Mechanisms of Signaling in Melanoma”

Boris C. Bastian, UCSF Comprehensive Cancer Center, San Francisco, CA
“Genomic Analysis of Melanocytic Neoplasia”

Ulf R. Rapp, University of Würzburg, Germany
“Role of Raf Kinases in Development and Treatment of Melanoma”

4:00 pm to 4:30 pm Break

4:30 pm to 6:00 pm Concurrent Session 10, Melanocyte Development and Melanoma

Introduction: David E. Fisher, Dana Farber Cancer Institute, Boston, MD
“Mitf and Transcriptional Regulation in the Melanocyte Lineage”

Heinz Arnheiter, National Institutes of Health, Bethesda, MD
“The Role of Mitf in Melanoblast Proliferation and Differentiation”

William J. Pavan, Genetic Disease Research Br., NIH, Bethesda, MD
“Functional Genomic Analysis of Melanocyte Development”

Michael Wegner, University of Erlangen, Germany
“Melanocytes and the Transcription Factor Sox10”
Concurrent Session 11, Melanoma Staging and Sentinel Lymphnodes

Introduction: Charles Balch, American Society of Clinical Oncology, Alexandria, VA
“Melanoma Staging: Migrating from the Light Microscope to Molecular-Based Staging Criteria”
Alistair J. Cochran, UCLA School of Medicine, Los Angeles, CA
“The Sentinel Lymph Node: A Singular Experiment of Nature”
Hans Starz, Klinikum Augsburg, Germany
“Sentinel Lymph Nodes and Melanoma Staging”
Douglas Reintgen, Lakeland Regional Cancer Center, Lakeland, FL
“Lymphatic Mapping and the Molecular Staging of the Sentinel Lymph Node”

Concurrent Session 12, Selected Poster Presentations

6:00 pm to 7:30 pm
Poster Session B, Etiology, Biology, Prevention, and Novel Therapies of Melanoma

Workshop, Advocates and Melanoma Research
Chair: DuPont Guerry, University of Pennsylvania, Philadelphia, PA

7:30 pm to 10:00 pm
Reception/Dinner
Sponsored by the Melanoma Research Foundation
Inauguration of the Society for Melanoma Research
Advocate Speaker

Tuesday, June 24

8:00 am to 10:00 am
Plenary Session 5, Tumor Microenvironment
Mary Hendrix, Chair

Robert Benezra, Memorial Sloan Kettering Cancer Center, New York, NY
“Id as a Target for Inhibiting Tumor Angiogenesis”
Mary Hendrix, University of Iowa Cancer Center, Iowa City, IA
“The Role of the Microenvironment in Facilitating the Plasticity of Melanoma Cells”
Pamela Robey, National Institutes of Health, Bethesda, MD
“The Microenvironment of Bone and Its Marrow”
Ann Richmond, Vanderbilt University School of Medicine, Nashville
“NF-kB, Chemokine/Chemokine Receptors, and Melanoma”
Michael Detmar, Massachusetts General Hospital, Charlestown, MA
“Angiogenesis and Lymphangiogenesis in Melanoma Progression”

10:00 am to 10:30 am
Break
10:30 am to 12:30 am Concurrent Session 13, Apoptosis and Differentiation in Melanoma

Introduction: Dario C. Altieri, University of Massachusetts Medical School, Worcester, MA
“Survival Checkpoints in Cancer”

Licia Rivoltini, Instituto Nazionale Tumori, Milan, Italy
“Role of Pro-apoptotic Molecules in Melanoma Escape from Immune Recognition”

Peter Hersey, University of Newcastle, NSW, Australia
“Overcoming Resistance of Melanoma Cells to Apoptosis”

Frank Meyskens, University of California, Irvine, CA
“Redox Changes are the Primary Driver for the Anti-apoptotic Phenotype of Human Melanoma Cells”

Concurrent Session 14, Biology of Melanoma Metastasis

Introduction: Ruth Muschel, University of Pennsylvania, Philadelphia, PA
“Mechanisms of Tumor Cell Arrest in the Vascular Bed: Models for Metastasis”

Peter Friedl, University of Würzburg, Germany
“Plasticity in Melanoma Cell Migration Strategies”

Danny R. Welch, University of Alabama, Birmingham, AL
“CRSP3-TXNIP-KiSS1: A Pathway for Melanoma Metastasis Suppression”

James McCarthy, University of Minnesota Medical School, Minneapolis, MN
“MT-MMPs in Invasion and Growth of Melanoma”

For more information:
http://www.foundationformelanomaresearch.org and http://www.wistar.upenn.edu/herlyn
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 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 rational 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.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:

Raymond E. Boissy, Ph.D.
Professor of Dermatology and Cell Biology, Neurobiology, & Anatomy
Department of Dermatology
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231 Albert Sabin Way, ML-0592
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Principal Scientist- Clinical Research - Skin Science Research
Unilever employs over 200 scientists at our New Jersey Laboratory who are dedicated to innovative and scientifically rigorous skin research programs. Our world sales exceed $40 billion so our programs have solid financial funding allowing for an innovative and challenging research culture. We currently have a full time opening that provides a unique opportunity to apply your basic science skills to human studies that impact the condition of skin for hundreds of millions people worldwide. We are seeking an expert in pigment biology or photobiology who can advance our knowledge and link laboratory research to clinically defined improvements of consumer skin problems. As a member of our skin research team, you will have an opportunity to work with other scientific experts in many fields including cell biology, biochemistry, measurement science and physical chemistry. You will also be encouraged to establish and maintain close ties to research in academic and government research communities.

We offer a competitive salary, benefits including tuition assistance and relocation, and a dynamic environment filled with learning and discovery beyond conventional scientific boundaries. Applicants must be authorized to work in the USA. For consideration please forward your CV to: Human Resources, Dept. CR-SID, Unilever Research US, 45 River Road, Edgewater, NJ 07020 or E-Mail: job.mca@unilever.com. Please place only the letters “CR-SID” as the subject of your e-mail. Unilever is an Equal Opportunity Employer m/f/d/v.

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.
Bibliography:

The Bibliography published in this issue covers the period June, 2002 through August, 2002. 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.

**MELANINS, MELANOGENS & MELANOGENESIS**

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**MELANOCYTES & KERATINOCYTES**


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MELANOMA & METASTASIS


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Smalley KSM, Eisen TG: Differentiation of human melanoma cells through p38 MAP kinase is associated with decreased retinoblastoma protein phosphorylation and cell cycle arrest. MELANOMA RES 12:187 (2002).


**MSH, POMC, GROWTH FACTORS & RECEPTORS**


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DEVELOPMENTAL BIOLOGY


DIFERENTIATION


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**MISCELLANEOUS**


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