PANAMERICAN SOCIETY for PIGMENT CELL RESEARCH

P A S P C R
N e w s l e t t e r

Volume 4 Number 4 **December,1996**

Introduction . . . **by the Publications Committee**

The **PASPCR** *Newsletter* is published quarterly and is intended to serve as a means of communication for the members of our Society. As such, we invite our membership to actively contribute to the *Newsletter*; help us to update the Job Listings, Calendar of Events, Meeting Reports, Abstracts in press and other items of general membership interest. If you attend a scientific meeting at which you heard about work which you think will be of interest to the membership of the **PASPCR**, please write a few paragraphs summarizing what was presented and share it with us. If you should have a change of affiliation or address, we'd like to know that, too. 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 any of the members of the Publications Committee.

NEW! WorldWideWeb Pages for the PASPCR. The PASPCR now has its own WWW home page. We plan this to be a major source of current information for the PASPCR membership. The address is: **http://lenti.med.umn.edu/paspcr**. This site contains information on the goals of the society, future meetings, council information, past issues of the PASPCR newsletter as well as links to other sites including the InterPig DataBase, past and future International Pigment Cell Conferences (IPCC) and the International Federation of Pigment Cell Societies (IFPCS).

We have now included the membership directory on that page; please notify us if you wish any or all of your information to be deleted or modified on that site.

Please check out the PASPCR web site and send any comments and/or suggestions to either the PASPCR WebMaster Bill Oetting at bill@lenti.med.umn.edu or to Vince Hearing at hearingv@dc37a.nci.nih.gov.

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PanAmerican Society for Pigment Cell Research

c/o **Dr. James J. Nordlund**
Department of Dermatology
University of Cincinnati
231 Bethesda Avenue
Cincinnati, OH 45267-0592
FAX: (513) 558-0198

The **PASPCR Newsletter** is published quarterly; for further information and/or to submit articles, contact the:

Publications Committee:

Dr. Kenneth A. Mason (chair)
University of Kansas
Department of Biochemistry
Lawrence, KS 66045
Phone: 913/864-4279
FAX: 913/864-5321

Frank L. Meyskens, Jr.
University of California - Cancer Center
101 City Drive
Orange, CA 92668
Phone: 714/456-6310
FAX: 714/456-5039

David A. Norris
Department of Dermatology
University of Colorado Medical Center
4200 East 9th Avenue
Denver, CO 80262
Phone: 303/372-1142
FAX: 303/372-1159

Calendar of Events :

Dec 7 - 11, 1996 36th Annual Meeting of the American Society for Cell Biology and 6th International Congress on Cell Biology, to be held in San Francisco, CA, (contact: ASCB Secretariat, 9650 Rockville Pike, Bethesda, MD 20814-3992; FAX: 301/530-7139)

April 23 - 26, 1997 Annual meeting of the Society for Investigative Dermatology, Washington, DC, (contact: the SID, Suite 500A, 1101 Cedar Ave., Cleveland, OH 44106, FAX: 216: 844-6859)

Jun 10 - 14, 1997 4th World Conference on Melanoma to be held in Sydney, Australia (contact: The Melanoma Foundation, PO Box M123, Camperdown, NSW 2050 Australia; FAX: +61 2/550-6316)

Jun 15- 18, 1997 VIIth PASPCR Annual Meeting, to be held in Providence, RI (contact: Dr. Walter C Quevedo, Jr., Brown University, Division of Biology and Medicine, Providence, RI 02912; FAX: 401/863-1971)

Jun 22 - 24, 1997 International Meeting "Pigmentary Disorders from a Global Perspective" to be held in Bali, Indonesia (contact: Bureau PAOG, Tafelbergweg 25, 1105 BC Amsterdam, The Netherlands; FAX: +31 20/696-3229)

Oct 9- 11, 1997 7th ESPCR Annual Meeting, to be held in Bordeaux, France (contact: 7th ESPCR Meeting Bordeaux, c/o Congres Seminaires Organisation, 81, Boulevard, Pierre 1er, 33110 Le Bouscat, Bordeaux, France)

Oct 30 - Nov 3, 1999 XVIIth International Pigment Cell Conference, to be held in Nagoya, Japan (contact: Dr. Shosuke Ito, Fujita Health University School of Health Sciences, Toyoake, Aichi 470-11, Japan; Phone: +81-562-93-2595; Fax: +81-562-93-4595; Email: sito@fujita-hu.ac.jp)

Welcome to New Members

by James J Nordlund

We welcome the following new members to the **PASPCR** . . .

Michael D. Brown Glynis A. Scott

If anyone is interested in joining our Society or wishes to sponsor a member, application forms can be obtained from Dr. James J. Nordlund at the PASPCR Secretary/Treasurer's office.

Corporate Sponsors

by James J Nordlund

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

GOLD Corporate Patrons	*SILVER Corporate Patrons*	*Corporate Patrons*
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VIIth Annual Meeting of the PanAmerican Pigment Cell Society

by Walter C Quevedo Jr

The VIIth Annual Meeting of the PASPCR will be held in Providence, RI from June 15th - 18th, 1997 at The Westin Hotel. Keynote Speakers:

Vincent J Hearing, PhD, Senior Investigator, National Cancer Institute, Bethesda, MD

Yutaka Kawakami, MD, PhD, Visiting Scientist, Surgery Branch, National Cancer Institute, Bethesda, MD

Richard L Sidman, MD, Bullard Professor of Neuropathology, Harvard Medical School, Boston, MA

James H Wyche, PhD, Associate Professor of Medical Science and Associate Provost, Brown University, Providence, RI

The keynote speakers will provide the foundation for four important themes of the meeting, 1) The biogenesis and structure of melanosomes, 2) Novel approaches to the development of anti-melanoma vaccines and other treatments, 3) The origin, distribution, and functional significance of ocular melanin, and 4) The nature and significance of programmed death (apoptosis) of normal melanocytes and melanoma cells. Symposia, mini-symposia and workshops are being planned on these themes as well as others reflecting the broadest interests of the Society's members. A new addition will be the offering of one or two "Sunrise Course(s)" with an early morning session on each of three days. Each course will be designed to provide interested members with basic information, state of the art technical methods and materials, and up-to-date modes of access to literature and archival technical data central to a particular sub-field of pigment cell research. More detailed information about the potential courses will be provided in the Second Announcement of the meeting that will be distributed early in February, 1997. The social program, still tentative, will include a reception and banquet at the Brown University Faculty Club and an outing recapturing Rhode Island History. Travel stipends to attend the meeting will be available for members of our Society as outlined in the following section.

The Organizing Committee welcomes suggestions. They should be directed to Hal Swartz [program] (E-mail: Harold.M.Swartz@Dartmouth.EDU) or [other matters] Walt Quevedo (E-mail: Walter_Quevedo_Jr@brown.edu).

PASPCR Travel Stipend / Young Investigator Award Information

by James J Nordlund

The next meeting of the PASPCR is not so far away, a little over 6 months. In a few months you will be preparing abstracts and presentations for this meeting. You should also start to consider requests for travel support and candidates to receive the Young Investigator Awards.

- **Travel Awards:** Travel awards up to $300 are given for students and new faculty needing assistance to attend the annual meeting of the PASPCR. The criteria require that the individual be a student, post-doctoral fellow or member of the faculty for less than 5 years. The candidates must be a coauthor on an abstract and preference is given to those presenting the abstract. A candidate can receive no more than three travel awards.
- **Young Investigator Awards:** There are three young investigator awards, one for students, one for post-docs and one for young faculty (less than 3 years total as a member of a faculty). The award is given both for the work being presented at the PASPCR annual meeting and for other accomplishments. Nominations are made by preceptors and mentors or other individuals. The nomination form with criteria will be included in your packet announcing the meeting and inviting you to attend that will come from Walt Quevedo. The nomination form must be completed and returned in a timely fashion to Jim Nordlund, *Secretary/Treasurer*. An anonymous committee will select the winner who will be named at the meeting in Providence. All awards are not necessarily given each year.

PASPCR General Business Meeting

by James J Nordlund

Meeting of the General Assembly, PanAmerican Society for Pigment Cell Research
Thursday, October 31, 1996 12:30 - 1:00 pm Disneyland Hotel, Anaheim, CA

1. The meeting was called to order by Sally Frost-Mason, *President*.
2. The *Secretary/Treasurer*, Jim Nordlund, noted that we generated $17,000 in funds and spent most of these for support of travel for young members and for the Newsletter. The Society has about $20,000 in reserves.
3. The President presented a plaque to Richard King for his 6 years as first *Secretary/Treasurer* and gave certificates to the outgoing members of the Council, Ray Boissy, Gert Jacobsohn and Dewayne Townsend.
4. Walter Quevedo presented highlights about the Providence meeting and encouraged all to attend.
5. David Norris discussed plans for the 1998 meeting in Colorado with a possible theme of photobiology and pigmentation.
6. Sally Frost-Mason listed the candidates for membership on the Council to be elected in the December ballot. They are (in alphabetical order):
   Greg Barsh, Jean Bolognia, Lynn Lamoreux,
   Kenneth Mason, John Pawelek, Setaluri Vijayasaradhi

   Dr. Frost-Mason encouraged members to make other nominations and discussed the mechanism of getting five signatures and sending the name of the candidate to Jim Nordlund
7. The membership was reminded that young investigator awards will require a formal nomination procedure from the student's mentor along with supporting data to be sent to the *Secretary/Treasurer* before the Providence meeting.
8. The meeting was adjourned.

Pigment Cell Research Wants YOU!

by James J Nordlund

THE PRICE OF THE JOURNAL ***PIGMENT CELL RESEARCH*** COMES TUMBLING DOWN. Hope that the stock markets don't crash like the cost of our pigment journal. Your subscription to ***Pigment Cell Research*** will be about 45% less than in previous years. The real price was over $200 but most were getting it at discount for about $160. At the recent meeting in Anaheim, Munskgaard agreed to revise its pricing schedule for the journal ***Pigment Cell Research*** for Society members only. For an annual fee of $95 total you can have your own copy of ***Pigment Cell Research***, the official journal of the International Federation of Pigment Cell Societies.

You get a lot for this small price. For example, you will get the bimonthly issues of the latest and best of pigment biology. In addition you will get the proceedings of the meeting of the European Society for Pigment Cell Research to be held in France later this year. You will get the proceedings of the upcoming PASPCR meeting in Providence, RI under the chairmanship of Walt Quevedo. You will get the proceedings of the XVIth meeting including the abstracts and the published manuscripts.

We need everyone's support. We need to get most of the members subscribing to keep this journal flourishing. Joe Bagnara did a yeoman's job getting it up and started and Dr. Jiro Matsumoto is doing a great job making the issues bigger and better.

An application for the journal will be included with your dues statement for 1997.

SUPPORT YOUR JOURNAL AND SOCIETY. SUBSCRIBE TO THE JOURNAL WHEN YOU RENEW YOUR MEMBERSHIP TO PASPCR.

News from the PASPCR Council

by James J Nordlund

Minutes of the Meeting of the Council, PanAmerican Society for Pigment Cell Research
Tuesday, October 29, 1996 8:30 - 10:30 am Board Room, Bonita Towers, Anaheim, CA

In Attendance: Sally Frost-Mason, *President*; Jim Nordlund, *Secretary-Treasurer*, Vince Hearing, Dick King, Frank Meyskens, Ken Mason, and Bill Oetting. A quorum was declared.

1. Dr. Sally Frost-Mason, *President*, opened the meeting and greeted all members.
2. The minutes of the meeting of May 22, 1996 were corrected by changes in spelling and accepted.
3. The *Secretary/Treasurer* presented his report. Richard King transferred $10,000 in February, 1996. We have generated $7319 from dues, sponsors and interest. The Society spent $4250 for travel awards for young investigators for the IPCC. Other major costs included $3220 for membership in the IFPCS and printing of the newsletter. We have a balance of $5047 in the checking account at the time of this meeting. There were several deposits outstanding and several debits due. The net amount should be about $5000 surplus. The Society has $21,000 in various long term saving accounts for reserves. This report was approved as presented.
4. There are three members of the PASPCR council who belong to the IFPCS council. Traditionally these include the *President, Secretary-Treasurer* and immediate *past-President.* Frank Meyskens suggested that it would be important to involve others by naming them to the IFPCS council. Other candidates will be considered although it was pointed out that the council of the IFPCS has its own difficulties and that seniority and continuity had some advantages. It was suggested that the representative at large from the PASPCR to the IFPCS be elected by ballot. Currently only Sally Frost-Mason and Vince Hearing are candidates for the presidency of the IFPCS council, a position allocated to a member of the PASPCR for the years 1996-1999. Vince Hearing was proposed as the candidate for this position. Dr. Frost-Mason declined the opportunity due to other responsibilities as Dean.
5. Dr. Quevedo and Swartz were not in attendance. There was significant information that an excellent scientific program was planned. Munksgaard will publish the abstracts. The *Secretary/Treasurer* was designated to discuss finances with Dr. Quevedo. At a breakfast meeting with Drs. Quevedo and Swartz, the budget appears to be balanced. The organizers are anticipating at least $1000-2000 surplus. It was recommended that the two organizers contact dermatologist colleagues who could assist them in contacting pharmaceutical companies so that

another $5000 might be raised to ensure a surplus. They will do this on return. A grant for $5000 is pending.

6. David Norris discussed briefly plans for the Denver meeting. It was noted that photobiology meetings might overlap with the Biology of the Skin/PASPCR meeting unless planned carefully. Norris will check the dates and suggested that the meeting be held either in Denver itself or Snowmass. He will report later to the Council about the better site.
7. Nominations for new members to the Council were reviewed. All have formally accepted their nominations. A ballot with a short statement from each candidate will be sent out in December, 1996. Any other candidates from the membership will be included but none had been received in the past or for this election.
8. Publications committee - Vince Hearing will continue to do the Newsletter which is very popular and worth the high cost of publication. A request for submissions of new items for the letter will be presented to the membership at large. The publications committee will meet with Mr. Hartmann about subscription rates as part of the IFPCS activities. They will report to the Council about the outcome.
9. Three members, Jean Bolognia, Hal Swartz and Zalfa Abdel-Malek attended the Photobiology meeting and presented an afternoon symposium that was well received. The PASPCR reimbursed each member $800 for travel to this meeting. A similar symposium will be held in Providence and members of the Photobiology Society will attend at no cost to them. No funds for Providence will be provided to the members of the symposium representing the PASPCR.
10. Frank Meyskens emphasized that it was most important to open this Society to as many members as possible and to involve them in the Council and other leadership activities. It was agreed that this goal was obtainable and very important.
11. Membership committee. It was suggested that investigators publishing work on pigment be invited to present abstracts or papers at the Society's meetings. Brochures passed out at other meetings can be of great interest to other scientists. It was suggested that investigative ophthalmology was experiencing a resurgence of interest in pigmentation due to the observations that the iris could develop new pigment when treated appropriately.
12. Frank Meyskens presented the following information about the IPCC:
    - Abstract books were sent out early. The agenda was kept flexible and some changes were made after the programs were mailed. New agendas were provided to each attendant.
    - There were 365 pre registered attendants. Of these 154 were from the PASPCR, 73 from the ESPCR and 59 from the JSPCR.
    - All platform presentations were competitive based purely on the scientific merit of the submitted abstract. There were some invited abstracts and presentations.
    - Patrick Riley, Shosuke Ito and Frank Meyskens chose the four best abstracts for an award.
    - The meeting should be revenue neutral. About $15,000 was available for travel assistance. The PASPCR contributed $10,000, NIH $5000, industry $25,000 and the IFPCS $15,000. The remainder came from registration. The total budget was in excess of $120,000.
13. There was no new or old business and the meeting was adjourned.

Respectfully submitted, James J. Nordlund, M.D., *Secretary-Treasurer*

XVIth IPCC (International Pigment Cell Conference) Program Summaries

The XVIth International Pigment Cell Conference was held from October 29th to November 3rd, 1996 at the Disneyland Hotel in Anaheim, California. Frank Meyskens was the Organizer of this meeting with Roger Bowers and Alistair Cochran serving as co-chairs of the Organizing Committee. Following are synopses of the various Symposia, Workshops and Poster Discussions written by Chairs of those sessions who are PASPCR Members. The meeting was a great success as those of you who attended already know; we are indebted to the organizers for putting on such a good show on behalf of the PASPCR. ***The Editor conveys a special thank you to all contributors of these summaries.***

Symposium I Economic and Societal Implication of Melanin and Melanogenesis **by Shosuke Ito**

M Chedekel summarized the current situation regarding the commercial application of melanin and suggested its potential use as an antioxidant. G Imokawa presented data on the roles of endothelin-1 in mitogenesis and melanogenesis. He showed that extracts of M. chamomilla, an antagonist of ET-receptor binding-mediated signaling, inhibit UVB-induced pigmentation on human skin. P Autier reported their ongoing study exploring the possibility that sunscreen use might foster proliferation of pigmented lesions of the skin. Preliminary rsults on 109 children indicated that the use of sunscreen tends to increase nevi count. Finally, G Prota presented his view on cosmetic applications of melanin and melanogenesis with special emphasis on the application of dopa derivatives in hair dyeing.

Symposium II Molecular Biology of Pigment Cells **by Vincent Hearing**

This Symposium began with an elegant Keynote Address given by N Dracopali, who discussed mutations in genes that regulate the G1 checkpoint of the cell cycle and how a large number of familial melanomas are associated with such mutations. Phosphorylation of the RB (retinblastoma) protein is important to the regulation of the G1 checkpoint, and the ability of cyclin-dependent kinases to phosphorylate RB is inhibited by a family of proteins, including $p16^{INK4a}$. Mutations in this $p16^{INK4a}$ gene have been identified in almost 50% of families with familial melanoma and these are thought to play roles in the generation of this type of melanoma. M Schartl presented his work on molecular mechanisms which lead to melanocyte transformation by the X^{mrk} receptor tyrosine kinase using *Xiphophorus* fish as a model. Mutations in this gene can lead to overexpression of this kinase which in turn initiates transformation of the pigment cells. Xmrk is closely related to the EGFR (epidermal growth factor receptor). They have used differential display to determine changes in gene expression in cells transformed with mutant X^{mrk} oncogenes. H Yamamoto reported on their analysis of the evolution of developmental systems of pigment cells, using the *tyrosinase* gene as a model. Similarities in upstream regulatory sequences of the TRP (tyrosinase related protein) family suggest that these genes are coordinately regulated. Cloning of these genes from ascidians revealed only a single gene, which was most similar to TRP1 and TRP2 rather than tyrosinase. K Toyofuku discussed his work on the importance of calnexin, a molecular chaperone, on the processing of tyrosinase, a step thought to be important to the regulation of melanogenic function of the enzyme. They have used cotransfection of calnexin and tyrosinase to examine the interactions, the results showing quite clearly that tyrosinase processing is markedly affected by coexpression of calnexin. DC Bennett discussed the cloning and mapping of a differentiation gene that regulates the state of differentiation of mouse melanoma cells. Transfection of this gene into B16 melanoma cells elicited increases in pigmentation and contact inhibition, along with decreases in tumor growth when inoculated into syngeneic mice. This gene was mapped to chromosome 14, and did not correlate with any known tumor suppressor gene or other cancer-related gene. Characterization of the function of this very important gene product awaits further study. S Porter reported on the regulatory sequences in far (15kb) upstream regions of the tyrosinase gene, and they are analyzing the mechanisms involved with those sequences. These sequences appear to be important to embryological development, particularly with respect to neural tube (optic cup) derived melanocytes. This Symposium was a fascinating insight into the varied molecular approaches being used to examine genetic regulation of pigment cell function and growth.

Symposium III Melanoma Research: Basic and Applied **by Frank Meyskens**

Six excellent papers comprised the content of this symposium. Cochran reviewed the current status of staging. Impressive data regarding the accuracy of sentinel node mapping were presented. Although large data bases have defined useful group predictions of outcome, increasingly sophisticated measurement of immunological and biochemical parameters is leading to the day when an individual's prognosis may be predicted with high accuracy. Such ability is likely to affect our post-surgical management of melanoma to a significant degree.

Three papers were concerned with manipulation of the melanin pathway. Y Mishima summarized his cummulate data about the use of neutron capture therapy; results continue to be encouraging. J Fruehauf reported on the cytotoxic action of busulfoxime (BSO), an inhibitor of GSH synthesis, on melanoma cells. BSO & busulfan was highly toxic to cells and cytotoxicity correlated to the melanin content of cells, suggesting that cells that have a higher oxidative stress (i.e. more active melanin synthesis pathway) are more sensitive to GSH depletion. E Link reported on elegant studies in mice and men that indicate that methylene blue is selectively taken up by melanoma cells. Based on these promising results a phase I/II study of Ab 211-labeled methylene blue is being planned. Finally, basic

work on two important melanoma associated proteins were reported. Studies of ICAM-I in cytokine and hyperthermia treated cells *in vitro* (J Nakayama) showed that this molecule was differentially expressed. Elegant studies by MY Hsu demonstrate that melanocytes switch from E-cadherin to N-cadherin expression during melanonogenesis, which may be in part explain the biologic basis for invasive and metastatic potential. The symposium was a stimulating one in as much as new prospects for the management of melanoma based substitutive biological observations were raised.

Symposium IV Photobiology of Melanocytes: Etiology and Prevention **by Lisa Zeise**

Summary pending; check the Web summary or watch for our next issue

Symposium V Melanogenesis and Pigmentary Disorders **by James Nordlund**

R Boissy opened the session with a superb review of the embryology of the pigment cell and its migration from the neural crest. Mutations in the c-kit proto oncogene are responsible for piebaldism. The *PAX* and *MITF* genes, both encoding transcription factors, when defective are responsible for various forms of Waardenburg's syndrome. The receptor for endothelin causes a syndrome of piebaldism and megalocolon. The various forms of oculocutaneous albinism are caused by mutations in the *tyrosinase* gene (OCA-1), the *p* gene (OCA-2) or the *TRP-1* gene (OCA-3).

M Mizoguchi studied the melanocytes of women with acquired facial dermal melanocytosis. This is a disorder that seems to have a predilection for oriental women and is characterized by formation of blue macules on the cheeks. She found in these lesions melanocytes in the lower dermis that seemed inactive but could be stimulated by various cytokines. She proposed that these melanocytes were embryological residue and were activated by cytokines during adult life to cause the syndrome.

RA Spritz presented information on the Hermansky-Pudlak syndrome (HPS). It is similar to albinism because the patients have marked pigmented dilution. In addition they have a bleeding diathesis, pulmonary disease, colitis and liver dysfunction. By homozygosity mapping, he and his coworkers mapped the gene for HPS to chromosome 10q23.1-q23.3. Occasional patients have a disorder that does not map to this locus, an observation suggesting several forms of HPS.

J Fryer presented on mutations at splice sites as a cause of OCA 1. Many point mutations, frame shifts and similar mutations have been identified. Using a lymphocyte line and PCR, Fryer and his colleagues studied splice sites in one family with albinism. They found that in this family a splice site mutation at the 5' end of exon 3 caused the entire exon to be deleted and exon 2 was spliced to exon 4. A second splice site mutation was identified in exon 1.

V Hearing studied the copper binding sites of the tyrosinase related proteins. There are two copper binding sites on the tyrosinase enzyme, CuA and CuB. He showed that elemental copper bound to the enzyme and that both sites required copper binding for normal enzyme activity. Other divalent cations could not substitute for copper in these sites. They found that CuB seemed to facilitate binding at the CuA site. The other two tyrosinase related proteins, i.e., TRP-1 and TRP-2, did not bind copper under the conditions of these experiments and these latter proteins might depend on other metal cations for activity.

This symposium was superb and provided a molecular basis for understanding some of the many disorders of pigmentation.

Symposium VI Comparative Developmental Biology of Pigment Cells **by Roger Bowers**

Matsumoto (the keynote speaker) presented an overview of the molecular biology of fish pigmentation, in particular the medaka. Causes of albinism in this fish, as found by Y Hori and associates, are due to an insertion of transposon-like sequence in exon 1 of the tyrosinase gene (mutant "i") and due to deletion of the short frame in exon 3 (Mutant "i4"). Matsumoto's group have produced transgenic homozygous orange-colored variant medakas carrying the cloned mouse tyrosinase gene in which the fish exhibits wild type pigmentation. The gene is stable and follows Mendelian genetics.

S Frost-Mason presented an evolutionary perspective of vertebrate chromatophore development entitled "From 3 pigment cell types to 1". She presented strong morphological, histological, cell and molecular biological evidence that the epidermal melanocyte of the mammal and bird may have evolved in a convergent manner from the 3 chromatophore cell types (melanophore, xanthophore, iridophore) found in fish, amphibia and reptiles.

D Bennett discussed differential gene expression in her murine immortal melanocytes, in her newly derived murine melanoblast line and in her newly derived murine melanoblast precursor line. She compared transcription factors, melanosomal proteins and growth related genes in these 3 lines and

the results showed that not all express the same genes except for *Pax3* and this difference may lead to a better understanding of cell differentiation and melanoma formation.

B Wehrle-Haller presented evidence that the early melanoblast migration is directed by localized steel factor. Migration is inhibited in mutant embryos that lack either steel factor (*Steel, Sl*) or its receptor (*dominant white spotting, W*). By studying various mutants that affect the presence or availability of steel factor, it was shown that the cytoplasmic domain of steel factor may have additional regulation functions for melanoblast migration not reflected in the COS cell system. The distribution of steel factor in the mutant will elucidate how steel factor regulates melanoblast migration and differentiation.

M Moody discussed the enhancement of the xanthophore lineage in guanosine-treated axolotl neural crest cells *in vitro*. Their results show that there is a specific developmental sequence which dictates where, when and what chromatophore type (black, yellow, white) differentiates. Axolotls can be treated with guanosine to suppress melanophore differentiation and simultaneously enhance xanthophore differentiation. Increasing one type of cell population is at the expense of the suppressed cell type population , suggesting transdifferentiation. This system may be a good model to study stem cell biology and transdifferentiation.

W Pavan presented results on genetic regulation of melanocyte patterning using 2 strains of piebald mice. Four loci are involved for the pattern difference. Chromosome 10 gene increased dorsal spotting and is probably steel factor, a conclusion supported by genetic and molecular biology analysis. The spotting pattern in the dorsal surface of the Mayer s/s mice is due to alteration in the normal function of the *steel* gene.

In posters related to this symposium and certainly no less important, T Fukuzawa *et al* showed that the melanization inhibiting factor in frog embryos was concentrated in the lateral and ventral skin and not in the dorsal skin at the external gill stage and that this changes as development proceeds. S Holder and G Thibaudeau presented evidence that axolotl neural crest cells from older embryos gave rise to more xanthophores than these same cells from younger embryos, that posteriorly located neural crest cells gave rise to more chromatophores that these same cells located in the anterior region and that guanosine treatment enhances xanthophore differentiation. R Kelsh and M Eisen characterized the colorless mutant in zebrafish and found that these embryos have essentially no melanophores and only a few normally pigmented xanthophores and iridophores. Any melanophores present are weakly pigmented and markers have shown that melanosomal related protein levels are low in these melanophores and that these cells do not migrate from their dorsal position in the embryo. K Mason *et al* showed that xanthine dehydrogenase is an excellent marker to identify differentiated xanthophores in axolotls. In another poster, they presented evidence that they have isolated a complete cDNA for axolotl TRP-1 and it is similar to its mammalian counterpart. A Masagaki and R Fujii presented evidence that shows the pigment pattern in pencilfish is changed at night by melatonin and this species may be used to study the action of β-melatonin and its analogs on melanophores. S Ali *et al* showed that nicotine caused fish apical melanophores to disperse their melanosomes whereas the basal melanophores aggregated their melanosomes. Frog melanophores dispersed their melanosomes due to nicotine as did the wall lizard. In another poster, they presented evidence in fish, frogs, toads and wall lizards that when histamine is bound to H1 receptors, it causes melanosome aggregation whereas when it is bound to H2 receptors, it causes melanosome dispersion. In a third poster, they showed that disinfectant phenolic compounds caused severe irreversible aggregation of scale *in vitro* fish melanophores. Hydroquinone was the most potent melanolytic agent. M Sugimoto and Hatayama presented evidence that nerve growth factor is involved in the regulation of the population of melanophores and in the density of adrenergic innervation in the medaka. Hirose *et al* showed that pigment cells in ascidians demonstrate a homology of chemical compounds but a difference in cell structures with higher vertebrate pigment cells and thus these chordates have a primitive form of pigment cell function and structure. R Morrison and Nagashima showed morphological evidence that the emergence of the embryonic pigment pattern in zebrafish is a highly dynamic process since the wild type adult pattern is quite different from the embryonic pattern. Okumoto *et al* presented evidence that melanosome movement in melanophores is under indirect control of the actin-myosin system which is located in a radial array in these dendritic cells. H Ono *et al* showed that the mouse tyrosinase gene introduced into the medaka is integrated into the fish genome and is capable of germ line transmission. M Goda and R Fujii found dendritic chromatophores that contained blue organelles in both the epidermis and dermis of 2 species of callionymid fish. These blue chromatophores were termed cyanophores and their blue organelles were termed cyanosomes. N Oshima *et al* showed that

prolactin caused pigment granule dispersion in erythrophores and xanthophores in the tilapia fish and that this response was seasonal in that it was greater in the spring and summer suggesting the involvement of prolactin in nuptial coloration. R Bowers *et al* presented evidence that IGF-II, EGF and insulin increased the number of *in vitro* adult highly differentiated avian primary culture melanocytes and that this was due to stimulation of migration from the feather piece onto the dish. Insulin also increased the viability of these cells. In a second poster, they showed that the number and migration distance of these same melanocytes can be doubled over control values by coating the dish with collagen type IV or fibronectin, suggesting that these cells still retain receptors for these ECMs from their embryonic days. In a third poster, they showed that the *in vitro* melanocyte premature cell death induced by unchanged media or media supplemented with α-MSH/dopa was identical ultrastructurally with the premature melanocyte death found in 2 chicken pigment mutants. Cytochemistry evidence also supported this similarity of cell death processes *in vitro* and *in vivo*. In a fourth poster, evidence was presented that showed that these adult highly differentiated *in vivo* avian melanocytes responded to α-MSH via putative receptors and that c-AMP is the second messenger for MSH in chickens as it is in mammals.

Workshop A Extracutaneous Melanin, Melanocytes and Melanogenesis **by Helene Z Hill**

The Workshop on extracutaneous melanin, melanocytes and melanogenesis began with an overview by RU Peter who pointed out that not all pigment cells in mammals derive from the neural crest. For example, the retinal pigment cells arise in the anterior neural tube. Pigment cells are found throughout the body in such varied sites as the meninges, peritoneum and blood vessels. In birds, pigment cells are prominent in the pericardium and in muscle. In fish, they are found in the lateral line among other sites. Extra-cutaneous melanomas in humans arise from many different primary loci. The functions of pigment are varied. It serves as camouflage, radiation protection and absorption, radical scavenging and as an anti-oxidant, mating signal and guidance for vasculature.

The first talk, entitled 'Melanin ~ the two-edged sword?' was by H Hill who studied mutation and survival in related cell lines that varied in pigment content. She found that induced eumelanin was photoprotective for mutations and survival but that constitutive melanin was only slightly photoprotective for survival for UVC and UVA but not for UVB nor a polychromatic lamp that resembled sunlight (FS20). In fact, albino melan-c cells were quite resistant to killing by FS20 compared to the pigmented melan-a and melan-b cells. DNA damage in the form of thymine dimers and 6-4 photoproducts appeared to be enhanced by pigment. In light of many conflicting reports in the literature concerning the role of pigment in light-induced damage to DNA, she emphasized that useful information regarding the role of melanin in the carcinogenesis of melanoma might only be gained by studying such biological endpoints as mutation and cellular transformation.

T Seikai described his studies performed of pigmentation abnormalities in flat fish. Ordinarily, the ocular side of these fish is hyperpigmented while the blind side is hypopigmented. The fish, a popular food item in Japan, are now produced by aquaculture. Under these conditions, there is a high incidence of hyperpigmentation on the blind side and hypopigmentation on the ocular side. Studies showed that irradiation of the blind side during metamorphosis inhibited mutation on this side. Nutritional factors during larval development are also important in the determination of pigmentation.

Prostaglandins are useful in the treatment of glaucoma. J Stjernschantz described the findings after chronic treatment of monkeys and patients in clinical trials with PGF2a, PGE2 and the FP receptor agonist latanoprost. After 3 to 12 months there was an increase in pigmentation in irises of brown but not blue eyes. There was no pigmentation change in nevi or freckles. The effect was due to increase in tyrosinase, not TRP1, which resulted in an increase in eumelanin.

Workshop B Dynamics of Invertebrate Pigment Cells **by K Ranga Rao**

This workshop, organized by S Negishi, included presentations on diverse aspects of pigmentation in arthropods. Rapidly-reversible color changes due to pigment translocations within epithelial chromatophores are displayed by many crustaceans, and are regulated by neuropeptides called pigment-concentrating and pigment-dispersing hormones. The cellular mode of action of one of these peptides, red pigment concentrating hormone (RPCH), was the subject of a report presented by LEM Nery, MA Silva, and AML Castrucci. Their *in vitro* experiments with the erythrophores of the shrimp *Macrobrachium* indicate that the action of RPCH involves phosphoinositol degradation that induces Ca^{++}/calmodulin complex formation and PKC activation.

Y Hasegawa and S Negishi have investigated the biochemical, cellular, and genetic basis of coloration in the terrestrial isopod, *Armadillidium vulgare*. The most common body color is black or grey, due to ommochrome containing chromatophores. Ultrastructural and biochemical studies indicate that albinism, as seen in the white phenotype, results from a defect in the synthesis or transport of the precursor before 3-hydroxykyneurenine in the ommochrome biosynthetic pathway.

A review of the hormonal control and pattern formation in insect pigmentation was presented by D Buckmann. Since chromatophores such as those in crustaceans are absent in insects, the latter are unable to display rapid color changes. Insects can undergo relatively slow color changes during the course of development or as morphological color adaptation. The role of environmental and endocrine factors, including the recent evidence for the involvement of neuropeptides, in the regulation of pigment synthesis was discussed. The characterization of the neuropeptides is in progress.

M Ashida presented biochemical and molecular evidence to establish that the insect prophenol oxidase is a protein homologous to arthropod hemocyanin. It appears likely that these molecules have originated from a common ancestral protein with a bi-nuclear copper cluster. Although time constraints in the workshop did not permit full disclosure of results, the activation of prophenol oxidase was reported to be triggered by minute amounts of microbial cell wall components and fungi--pointing to potential role as a defense mechanism.

Workshop C Regulating Mechanisms of Melanocyte Proliferation **by Zalfa Abdel-Malek**

An overview on the evolution of the methods for culturing normal human melanocytes was presented by Z Abdel-Malek. Cultured human melanocytes are an ideal *in vitro* model to investigate the regulation of human pigmentation. The first growth medium which allowed for the long term proliferation of human melanocytes relied on the use of tumor promoting phorbol esters and cholera toxin. Over the years, many investigators modified this initial procedure by replacing these artificial and toxic agents by physiologically relevant growth factors, most of which are synthesized by human keratinocytes and thus can function potentially as paracrine regulators of melanocytes. Such factors include basic fibroblast growth factor, leukotriene C4, hepatocyte growth factor, stem cell factor, endothelin-1 and α-melanocyte stimulating hormone. The observations that normal human melanocytes require several growth factors with different signaling pathways in order to proliferate in culture, suggested that mitogenic stimulation of these cells requires the crosstalk of different signal pathways. These pathways include the protein kinase C, cAMP/protein kinase A, and tyrosine kinase pathways.

The selected abstracts dealt with the role of: 1) microphthalmia-associated transcription factor (MITF), 2) extracellular matrix proteins, 3) oxidative damage, 4) cell cycle regulatory proteins, in normal melanocyte proliferation and differentiation.

M Tachibana presented data on the induction of melanocyte differentiation by MITF. Expression of this transcription factor in NIH/3T3 cells which constitutively express TRP-2 resulted in the expression of tyrosinase and TRP-1, and in a dendritic morphology. Data was also presented on two novel mutations of the *MITF* gene in individuals with Wardenburg Syndrome type 2 (WS2A) from two different families. WS2A is a dominantly inherited disease characterized by pigmentary abnormalities and deafness possibly related to loss of melanocytes from the stria vascularis of the inner ear. The above two mutations result in proteins that lack sequence-specific DNA-binding activity, and ability to transactivate the tyrosinase promoter, but do not disrupt the function of the wild-type MITF protein. These results suggest that the WS2A phenotype is caused by loss-of-function of the two alleles of the *MITF* gene.

S MacNeil presented on the effect of extracellular matrix (ECM) proteins on cutaneous and ocular melanocytes. She stated that different ECM proteins from different sources (e.g. human dermal fibroblasts or microvascular endothelial cells) stimulate tyrosinase activity in cutaneous melanocytes. Fibronectin, in particular, stimulates tyrosinase activity in ocular melanocytes, and increases intracellular calcium.

The abstract that was to be presented by A Thody offered a new explanation for why melanocytes are more vulnerable to oxidative damage than keratinocytes or fibroblasts. In addition to their lower level of anioxidant enzymes, melanocytes seem to be capable of producing superoxide anion and nitric oxide. The production of superoxide anion by the xanthine oxidase/xanthine system was reduced in the presence of human fibroblasts or keratinocytes, but increased in the presence of human melanocytes or B16 melanoma cells. Additionally, B16 melanoma cells also produced superoxide anion and nitrous oxide following UVB irradiation.

Data from E Medrano's laboratory describing the role of cell cycle regulatory proteins in end-stage differentiation of human melanocytes was presented by M Haddad. Human melanocytes can be induced to reach end-stage differentiation by chronic treatment with high concentrations of cAMP inducers, such as cholera toxin. At this stage, melanocytes did not respond to the addition of fresh medium with significant pRb phosphorylation, expressed a low level of cyclin D1, high level of p27, and a moderately high level of p21. Unlike proliferating melanocytes in which MITF becomes highly expressed, downregulated, and then highly expressed again, irreversibly arrested melanocytes continuously express a high level of MITF.

A Platz described mutations in cell cycle regulatory genes in sporadic human melanoma tumors. In 26 metastases from 25 patients, 4 tumors had mutations in CDKN2, 2 had mutations in CDKN2B, 3 tumors had mutated p53, and 2 had mutations in N-Ras. In addition 34 patients, 8 had codon 61 mutations of N-Ras, 10 of 19 mutations were G-C/A-T or A-T/G-C transitions, and 2 were C-G/G-C transversions at sites of adjacent pyrimidines. These results suggest that these mutations are UV-induced, and support a role of UV in the etiology of human melanoma.

Workshop D Biophysics and Chemistry of Melanin **by Hal Swartz**

Summary pending; check the Web summary or watch for our next issue

Workshop E Vitiligo **by David Norris**

Summary pending; check the Web summary or watch for our next issue

Workshop F Control of Melanogenesis **by John Pawelek**

Summary pending; check the Web summary or watch for our next issue

Workshop G The "Blues" Symposium **by Joseph Bagnara**

This workshop was organized by J Bagnara, J Bolognia and Y Hori in order to emphasize the reality that pigment cell researchers from very diverse areas deal with problems that are seemingly unrelated, but are in fact very similar. Blue coloration is a prime example of this fact. In his Introduction, Bagnara pointed out that blue colors among all the vertebrate groups have a physical basis and are truly "structural colors." With a few examples, he indicated that blue colors among the various vertebrates are related by either analogy or homology. As an example of the latter, it was shown that blue spots in some fishes are like the blue nevi of humans. A superb tone for the session was provided by C Bohren, an atmospheric physicist from Penn State, who, with unparalleled humor, poked holes into many of the physical misconceptions about blue coloration. "The physicists" were often foils for his humor. He emphasized the need for colorimetry in assessing blue colors.

The remainder of the session followed a phylogenetic approach and started with human cerulodermas. Blue nevi and mongolian spots were discussed by J Bolognia while Y Hori considered the Nevus of Ota and other nevi fuscocaeruli. A description of the nevi and treatments were presented. The results of ruby laser treatment were impressive. The blue colors of fish were discussed by R Fujii who emphasized the physical role of the reflecting platelet organelles of iridophores. He pointed out their function in light scatter, reflection, and thin-layer interference and explained how some of the respective hues of fishes could be achieved therein. A high point of his presentation was the novel demonstration of truly blue chromatophores (cyanophores) that contain a genuine blue pigment, as yet uncharacterized. P Fernandez presented numerous examples of blue coloration, either normal or "abnormal" among amphibians. He discussed the role of the dermal chromatophore unit in imparting both blue and green coloration. A high point of his presentation was his use of colorimetry to objectively describe skin colors through representation on a chromaticity diagram. R Morrison followed with an assessment of blue colors in several lizards, notably a scaly lizard, *Sceloporus jarrovi*. In this case, the role of thin-layer interference was emphasized. R also was given the task (but no time) to discuss blue colors of birds. He limited his words to bare patches of skin such as wattles. Here, blue coloration is attributed to structurally based events involving orderly arrays of extracellular collagen. W Quevedo concluded the formal presentations by considering the blue colors of mammals, notably those that occur as secondary sexual characteristics of adult male mandrills. He discussed the behavioral significance of the red, blue, and white pattern of the face and anogenital regions of such males and indicated that the "blue color depends upon a complex interplay of variable amounts of hemoglobin in dermal blood cells and immobile melanosomes of adjacent dermal melanocytes." Following the formal session, a brief free presentation from R Aquaron described a clinical manifestation of "blue ears" in patients with alkaptonuria who accumulate homogentisic acid. Altogether, the "Blues" symposium attracted a good audience and evoked lively discussion.

Workshop H Biology and Biochemistry of Melanosomes **by Seth J Orlow**

To kick off the session, Y Mishima gave an overview of the relationship between melanosomes and lysosomes. He reviewed data from his own lab on the transfection of genes encoding TRPs into amelanotic melanoma cells, as well as the data implicating coated vesicles in the trafficking of proteins to melanosomes. K Jimbow reviewed his lab's experience with identification of calnexin as a molecular chaperone implicated in the proper folding of tyrosinase in the endoplasmic reticulum as well as that of the small GTP-binding protein, rab7, in controlling trafficking of TRP-1 to melanosomes. Later in the session, P Gomez of Jimbow's group expanded on this latter subject. Rab7 was identified in 2-D gels of melanosomal proteins by overlay with radiolabelled GTP followed by partial sequence analysis and cDNA cloning. It colocalizes with TRP-1 to melanosomes. Melanoma cells transfected with a rab7 antisense construct show a more restricted perinuclear distribution of TRP-1, supporting the contention that rab7 may be involved in TRP-1 trafficking. K Araki spoke about the identification of rab3a, another small GTP-binding protein, with melanosomes both by copurification as well as by immunoelectron microscopy. A protein which interacts with rab3a, namely Rabphilin-3A, was also present in melanosomes of B16 melanoma cells. In contrast, RabGDI was ubiquitously distributed in many subcellular components. C Sakai described the effects of recombinant agouti signal protein (ASP) on immortalized cultured murine melanocytes (melan-a cells). ASP counteracted MSH's stimulatory effects on these cells, but even in the absence of added MSH, ASP inhibited tyrosinase mRNA and protein levels and, to a lesser extent those of TRP-1 and TRP-2. Melanosomes in ASP-treated cells tended to be rounder, more like the shape of pheomelanosomes. Interestingly, ASP seemed to counteract even the stimulatory effects of cholera toxin, suggesting that it might act through an additional signal transduction pathway in addition to its role as a noncompetitive anatgonist of MSH. Finally, J Hammer discussed his research on the product of the murine *dilute* locus, aka myosin V. This unconventional myosin has calmodulin binding sites and may serve to link the melanosome to the cytoskeleton in a calcium-dependent manner. The protein is indeed associated with melanosomes, colocalizing with such bonafide melanosomal proteins as TRP-1. It was long thought that the defect in dilute mice was their inability to extend dendrites. Using antibodies to the melanocyte cell surface receptor c-kit, Hammer's group has now shown that there is nothing wrong with dendrite extension in dilute mice or cultured melanocytes derived therefrom. Rather, the problem appears to be due to an inability to translocate melanosomes from their perinuclear area of origin down through the dendrites from whence they can be transferred to keratinocytes.

Workshop I Genetic Aspects of Albinism **by Richard King**

This workshop focused on recent studies of human albinism and *tyrosinase* gene expression in the mouse. J Matsunaga reviewed their experience with *tyrosinase* gene mutations in tyrosinase-negative OCA in the Japanese population. Four mutations have been identified: R77Q, R278TER, ΔC310, and P431L. Affected individuals were homozygous for R77Q/R77Q (n=2) or ΔC310/ΔC310 (n=4), or compound heterozygous for two different mutations. One individual was a compound heterozygote with R77Q on one allele and no detectable mutation of the homologous allele. Extensive evaluation of the promoter region of the *tyrosinase* gene on this allele, playing particular attention to the TDE region and the area of the $(GA)_n$ repeat did not reveal a mutation that would account for the loss of function associated with this allele. F Beermann evaluated the promoter of the *tyrosinase* gene using a *tyrosinase-LacZ* fusion gene in transgenic mice. Expression was found in several areas of the developing and the adult brain. Immunohistochemistry studies showed tyrosinase-specific bands in the brain and eye, although no enzyme activity was detected. The potential role of tyrosinase expression in the brain was discussed. JM Newton presented new data on the isolation of the mouse homologue (*Moa1*) of the human *Ocular Albinism 1* (*OA1*) gene. The gene product appears to have six transmembrane regions and exists in two isoforms. The gene is expressed in the skin and eye of the neonatal mouse but only in the eye of the adult mouse. MSH and ASP had no effect on *Moa1* expression. Analysis of tissue expression showed that the Moa1 protein co-segregated with TRP1 protein in the melanosomal-enriched fraction of pigmented tissue. W Oetting presented further data on the analysis of the *P* gene in human OCA2. Many silent and missense polymorphisms were found, as well as a large number of pathologic mutations. A screen on control individuals was used to establish the difference between a polymorphic and a pathologic mutation. The distribution of mutations in the gene was random and no functional domains were suggested by mutation distribution.

Poster Session #2 Melanogenesis **by John Pawelek**

Summary pending; check the Web summary or watch for our next issue

Poster Session #3 Biophysics and Chemistry of Melanin **by Hal Swartz**

Summary pending; check the Web summary or watch for our next issue

Poster Session #4 Pigment Cell Development and Dysfunction **by Walter C Quevedo Jr**

This session revealed the narrowing gap between studies of the paraclinical aspects of melanocyte dysfunction (albinism, vitiligo, hypermelanism etc.) in humans and the basic studies on the cell and molecular biology of melanocyte development, pattern formation and regulation. Particularly promising were the reports of progress made toward characterizing the mechanistic basis for the generation of pigment patterns in animals. These findings, when integrated with the new information on life and death responses of melanocytes to growth factors that was reported in this session, should provide new insights into the origin of symmetry in the expression of several human hypopigmentary disorders. The broad range of vertebrate and invertebrate animals under investigation was striking as was the emerging evidence for evolutionarily conserved and divergent features of pigment cell development that makes each animal species, regardless of where it sits in the phylogenetic "tree", relevant to all of the others.

Invitation to the XVIIth IPCC (International Pigment Cell Conference) by Shosuke Ito

Invitation to the XVIIth International Pigment Cell Conference Nagoya Congress Center
Nagoya, Japan October 30 - November 3, 1999

Dear Colleague:

After the inauguration of the International Federation of Pigment Cell Societies (IFPCS) in Kobe in 1990, the International Pigment Cell Conferences (IPCC) rotate among the European, American , and Asian continents, hosted by one of the three regional societies: the ESPCR, the JSPCR, and the PASPCR. The 15th IPCC was thus held in London in 1993, chaired by Professor Patrick A. Riley, and the 16th IPCC was recently held in Anaheim, California, chaired by Professor Frank L. Meyskens, Jr.

It is our great honor and real pleasure to inform you that the next 17th IPCC will be held in Nagoya, Japan in 1999, co-organized by the IFPCS and the JSPCR. We heartily hope that pigment cell biologists and clinicians will join together in Nagoya in October 1999 to present their latest achievements in the exciting world of pigment cell research. Your participation will be most important for the scientific success of this meeting.

The city of Nagoya, the 4th largest in Japan, enjoys a rich history of traditional culture and a reputation for world-renowned high-tech industries. Nagoya is located at the center of Japan and is easy to access: the Nagoya International Airport is directly connected with 30 cities around the world. The conference site, the Nagoya Congress Center, is newly built and has ample spaces for the participants to discuss and exchange ideas, which we believe will certainly bring about fruitful collaborations.

We will follow the good tradition of the IFPCS leadership in directing scientific programs to unify the three regional societies. Within such a framework, we wish to place special emphasis on poster presentations. We hope to provide a certain number of travel grants for young investigators to attend this meeting. In order to be eligible for such a grant, an applicant has to be a member of one of the three regional societies for at least one year prior to the meeting. We are also planning banquet and social activities in such a way to make your visit to Nagoya most enjoyable and memorable. It will be our great privilege to welcome you and your colleagues to Nagoya in 1999.

Shosuke Ito, Ph.D. — *Chair,* IPCC Nogoya
Kazumasa Wakamatsu, Ph.D. — *Secretary-General,* IPCC Nagoya

For further information please contact us at: Fujita Health University School of Health Sciences, Toyoake, Aichi 470-11, Japan; *Phone*: +81-562-93-2595; *Fax:* +81-562-93-4595; *Email*: sito@fujita-hu.ac.jp

Positions - Wanted and Available :

Cell Biologists - Unilever's Research and Engineering Division has two openings for the following position description. Changes in pigmentation of the skin are part of the adaptation response to a variety of conditions. These changes are caused and characterized by very marked changes in skin cell biology and biochemistry. We wish to recruit two scientists to be part of a new project team researching mechanisms of pigmentation and mode of action of certain skin lightening agents *in vivo*. The project will require the establishment and investigation of appropriate *in vitro* and *in vivo* models for pigmentation research. Expertise required: Candidates must have a good honours degree and PhD in a biochemical or cell biology subject with at least 3 years of research training in a good laboratory. The candidate must have a proven research record. Postdoctoral experience would be advantageous. Please send your CV quoting the Reference number MM960604 to: Bryony Leleux, Personnel Department, Unilever Research, Colworth Laboratory, Sharnbrook, Bedfordshire, MK44 1LQ; Email bryony.leleux@urcgb.sprint.com.

Predoctoral and Postdoctoral Positions - available for molecular biologists in the areas of drug discovery and metabolism research. Requires experience in gene cloning, DNA sequencing, recombinant protein expression and cell culture methods. Prior experience in dermatology research is desirable. Southern Research Institute is a diversified research and development organization. Our Life Sciences Division provides comprehensive preclinical drug development and testing capabilities as well as basic research in drug design and synthesis, pharmaceutical formulations, toxicology, virology, microbiology, and pharmacology. To apply, send resume or curriculum vitae to: Southern Research Institute, Attention: Suzann Allen, Human Resources, Department 118, P.O. Box 55305, Birmingham, AL, 35255-5305.

Faculty Position - Massachusetts General Hospital, Harvard Medical School, Cutaneous Biology Research Center. The Cutaneous Biology Research Center (CBRC) seeks a molecular, cellular or developmental biologist to establish a program in fundamental research relevant to skin pigmentation. Areas of reseearch can include but are not limited to pigment synthesis and transfer in melanocytes, genetics of mouse coat color and development/migration of neural crest cells. Applicants must have a Ph.D. and/or M.D. degree and relevant postdoctoral experience. Only applicants with a strong research record and the potential to develop extramurally supported research programs will be considered. Individuals with a demonstrated ability to develop imaginative approaches to important biological questions are particularly encouraged to apply. Rank/salary/start-up funds and space are negotiable depending on experience and qualifications. The CBRC occupies 45,000 square feet of fully equipped laboratory space in a new multidisciplinary research facility. Interested individuals should send curriculum vitae, reprints, a statement of research and future directions, along with the names, addresses and telephone numbers of three references to: Dr. Paul F. Goetinck, Chair, Faculty Search Committee, Cutaneous Biology Research Center, Massachusetts General Hospital - East, Building 149, 13th Street, Charlestown, MA 02129

INTERPIG DataBase

by Vincent Hearing

The INTERPIG database is on the InterNet! You can now access the InterPig DataBase at the following address: **http://lenti.med.umn.edu/paspcr/interpig.html**. Please note that as of this time, less than 5% of the various IFPCS members have contributed entries. Think of how useful and complete this list would be if everyone took the time to supply their information. Please take a moment to fill out the database data entry form (either online through the Web page or via Email) and send it back to Dr. Hearing. Please contact Vince Hearing or Bill Oetting if you need more information about these mechanisms of submission.

Members in the News

Vincent Hearing received the Takeuchi Medal at the XVIth IPCC in Anaheim.

Richard King received the Seiji Memorial Award and presented the Seiji Lecture at the XVIth IPCC in Anaheim.

Setaluri Vijayasaradhi has moved from The Rockefeller University; his new contact information is: Department of Dermatology, Bowman Gray School of Medicine, Wake Forest University, Winston-Salem, NC 27157; Tel: (910) 716-3273; Fax: (910) 716-7732; e-mail: setaluri@bgsm.edu

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The Bibliography published in this issue covers the period August, 1996 through October, 1996. 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. We have attempted to highlight any publications which include a member of the PASPCR with a star.

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COUNCIL MEMBERS - Sally Frost-Mason, (Lawrence, USA); Yutaka Mishima, (Kobe, JAPAN); Shosuke Ito, (Toyoake, JAPAN); James J Nordlund, (Cincinnati, USA); Stan Pavel, (Leiden, NETHERLANDS); Giuseppe Prota, (Naples, ITALY)

The IFPCS Council met several times during the IPCC held in Anaheim this Fall, and, among many other actions (as outlined below), elected as Officers for the next 3 years, VJ Hearing (PASPCR) as *President*, Y Hori (JSPCR) as *Vice-President* and BS Larsson (ESPCR) as *Secretary/Treasurer*. On behalf of my fellow Officers, I would like to thank everyone for their confidence, and I can assure you that we intend to work extremely hard as a cohesive unit during these next 3 years to continue the growth, innovation and interactions initiated during the first 2 administrations under Profs Mishima and Prota. This now completes the rotation cycle among the regional Societies and we will strive to make the IFPCS even more active and efficient in fostering scientific exchange among our members. In addition to other Council members who have stayed on (Drs VJ Hearing, S Ito, BS Larsson, Y Mishima, J Nordlund, and G Prota), we would like to welcome our new Council members (Drs S Frost-Mason, Y Hori and S Pavel) and say goodby to departing Council members (RA King and PA Riley).

I would like to extend the thanks of the IFPCS to Drs. FL Meyskens, RR Bowers and AJ Cochran (Chairman and co-Organizers), as well as to their Organizing and Scientific Program Committees, for the outstanding job they did for the XVIth International Pigment Cell Conference (IPCC) held in Anaheim. The social and scientific program, as well as the Conference facility itself, was outstanding and a great success.

The **XVIIth IPCC** will be held in Nagoya, Japan from October 30th - November 3rd, 1999 under the chairmanship of Prof S Ito and I hope everyone will make plans to attend that meeting. We have seen the preliminary plans for that Conference and it promises to be an outstanding one; Prof Ito is planning a number of stimulating social and scientific events for that meeting and travel support should be available for Young Investigators in the various regional Societies who might want to attend. Details of the XVIIth IPCC will be forthcoming as the meeting approaches.

The **IFPCS is on the InterNet**; the address is: **http://lenti.med.umn.edu/paspcr/ifpcs.html**. From our home page you can access links to the InterPig DataBase, news of the upcoming XVIIth IPCC, summaries of the previous IPCC, home pages of the regional Pigment Cell Societies, and other pertinent information. We invite you to visit us at this site and enjoy the wealth of information there; I would like to thank WS Oetting for his time spent in maintaining this site at no expense to the IFPCS. Let us know how we might improve this Web site to make it a more valuable resource to your work.

The **Publications Committee** (Y Mishima, chair, JJ Nordlund, S Pavel) met with Peter Hartmann of Munksgaard regarding subscription prices and other publications issues. They were able to negotiate a very favorable subscription rate available only to members of the regional Societies; this $95 (USD) rate represents about a 70% discount over the standard rate of $324 (USD). We urge all of our members to subscribe to ***Pigment Cell Research*** through their regional Society to obtain this rate and support the journal. The Editorial Report given by J Matsumoto was excellent and the IFPCS Council expressed its appreciation to him for his outstanding job as the Editor.

The **Special Experts Committees** (G Prota, chair), initiated 3 years ago to foster interactions in specific subfields of pigment cell research, will be expanded over the next several years and should culminate with interim Reports, Discussion Groups and eventually with IPCC Symposia and Workshops. The topics and chairs of each Special Expert Committee is: Albinism - RA King; Biology of Melanoma - FL Meyskens; Development Biology - S Frost-Mason; Hypo/Hyperpigmentation - Y Mishima; Ocular/Extracutaneous Pigmentation - G Prota; Vitiligo - JJ Nordlund; InterPig DataBase - PA Riley. These chairs will be coordinating the activities of their groups and anyone who wishes to provide suggestions and comments to those groups can address them to the relevant chair.

As a new initiative, we are trying to generate a source of funds to sponsor **IFPCS Travel Awards**; these will be intended for Young Investigators from each of the 3 regional Societies to visit other laboratories (preferably in other countries) to learn specialized techniques and/or to establish collaborations. Such support will be for 2-3 months only and will be competitive on an annual basis; each Society will determine its own mechanism for soliciting and awarding such awards. We are hoping to establish these awards beginning in 1997 and further information about these will be forthcoming from your own Society.

Finally, we have established an *ad hoc* **Women Scientists Committee** (S Frost-Mason, chair, M Mizoguchi, DC Bennett) that will discuss women's and other minority issues in pigment research; that Committee will make recommendations to the IFPCS Council as to how we might help deal with any concerns noted and improve the lot of those affected.

To conclude, I am highly optimistic that the next 3 years will see continued outstanding growth and opportunity in our 3 regional Pigment Cell Societies. Our field of pigment cell research is becoming a more popular and interactive one every day and I would encourage each of you to become more active in your Society and recruit others in the field to join. I would welcome any suggestions and comments you might care to make as to how the IFPCS might function more efficiently to promote cooperation and collaborations between our members. I would like to take this opportunity to wish each of you a safe, healthy and prosperous New Year.

With best regards,

Vincent J Hearing
President, IFPCS