PANAMERICAN SOCIETY for PIGMENT CELL RESEARCH

# PASPCR Newsletter

**Volume 3 Number 2** **June,1995**

**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.

The Publications Committee wants to make collaboration between members of the Society as easy as possible and toward that goal we are encouraging the use of E-mail and other forms of electronic communications. We encourage everyone who has an E-mail address to forward it to the Publications Committee. The Publications Committee is willing to help any member of the PASPCR get you in touch with the individuals who can set up your E-mail account and to some extent will help you get started.

The **PASPCR** has a Gopher server that can be found from your home Gopher under "International Organizations" as "PanAmerican Society for Pigment Cell Research (PASPCR)". Here you have access to past PASPCR Newsletters, the current ByLaws and membership list. We are currently working with the IFPCS to develop a list of resources available to PASPCR members. This list will include cell lines, antibodies, coat color mutants and other items of interest to pigment researchers. If you have any other

**IN THIS ISSUE . . . .**



enclosure: **IFPCS DataBase Form**

ideas for items to be placed on Gopher, please contact someone on the Publications Committee.

**PanAmerican Society for Pigment Cell Research**

c/o **Dr. Richard A. King**
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## Calendar of Events :

**June 25 - 28, 1995** VIth PASPCR Annual Meeting, to be held in Kansas City, Kansas, (contact: Dr Sally Frost-Mason, Department of Physiology, University of Kansas, 3038 Hayworth Hall, Lawrence, KS 66046-2106, FAX: 913/864-5321)

**July 11 - 15, 1995** 44th Annual Symp on the Biology of Skin, to be held in Snowmass Village, CO (contact: Cutaneous Biology Foundation, 4200 E 9th Ave, UCHSC Box B 144, Denver, CO 80262; FAX: 303/270-8272)

**Oct 19 - 21, 1995** 6th Meeting of the European Society for Pigment Cell Research, to be held in Lausanne, Switzerland (contact: ESPCR '95, Centre Pluridisciplaire d'Oncologie, CHUV-BH 06, CH-1011 Lausanne, Switzerland; FAX: +41 21/314-3957)

**Dec 2 - 3, 1995** Meeting of the Japanese Society for Pigment Cell Research, to be held in Osaka, Japan (contact: Prof. Toshiteru Morita, Dept of Biology, Osaka University, 560 Osaka, Japan; FAX: +81 6/850-5613)

**Dec 9 - 13, 1995** Annual Meeting of the American Society for Cell Biology, to be held in Washington DC (contact: ASCB National Office, 9650 Rockville Pike, Bethesda, MD 20814-3992; FAX: 301/530-7139)

**Oct 29- Nov 3, 1996** XVIth International Pigment Cell Conference, to be held in Anaheim, California, (contact: MMC/UCI Center for Health Education, PO Box 1428, Long Beach, CA 90801-1428, FAX: 310/933-2012)

**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)

## Welcome to New Members

**by Richard A King**

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

| | | |
|---|---|---|
| Gisela F. Erf | Jamal Z. Farooqui | Minao Furumura |
| Maher M. Haddad | Julius L. Harp | Jorg M. Klein |
| Weixiong Li | Milton R. Okun | Parthasarathy Ramasastry |
| Ryan J. Saetveit | Chie Sakai | Scott Wildenberg |

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

## Corporate Sponsors

**by Richard A King**

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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## 1995 PASPCR VI$^{th}$ Annual Meeting Site :

**by Program Committee**

**PROGRAM - Sixth Meeting of the PASPCR - Kansas City, MO, June 25-28, 1995**

**SUNDAY, JUNE 25**

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| 12:00 - 8:00 pm | **Registration - Ballroom Foyer** |
| 1:00 - 4:00 pm | **PASPCR Council Meeting,** in Conference Suite 641, The Ritz-Carlton |
| 4:00 - 7:00 pm | **Poster set-up and preview** - Posters will be set up in **Salon II** at The Ritz-Carlton and will be displayed till the end of the sessions |
| 7:00 pm | **Welcome** - Dr James Muyskens, Dean, College of Liberal Arts and Sciences, University of Kansas, Lawrence, KS - **Salon III** |
| 7:00 - 8:00 pm | **Keynote Address** by Shirley M Tilghman (Lawrence Gelb Lecture) - **Salon III** |
| | GENETIC AND PHENOTYPIC ANALYSIS OF MELANOCYTE DEVELOPMENT IN PIEBALD MICE. Shirley M Tilghman, William J Pavan, Myung K Shin, Thomas A Vasicek, Susanna Mac and Mickie Cheng |
| 8:00 pm | **Reception - Poolside** at The Ritz-Carlton |

**MONDAY, JUNE 26**

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| 8:00 - 5:00 pm | **Registration** Table Open |

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| | **Speaker Preview** Room Open - **Pavilion V** |
| 8:00 am | Continental Breakfast - **Salon II** |
| 8:30 - 9:30 am | **Keynote Address** by Garth L Nicolson - **Salon III** |
| | THE ROLE OF NEUROTROPHINS AND PARACRINE GROWTH FACTORS IN MELANOMA BRAIN INVASION AND METASTASIS. Garth L Nicholson |
| 9:30 - 10:00 am | Break - **Salon II** **Poster Viewing** |
| 10:00 - 12:00 | **Symposium I** - Greg S Barsh - **Salon III**<br>**"Developmental and Genetic Approaches to Pigment Cell Biology"** |
| 10:00 - 10:30 | MOLECULAR MARKERS NEAR MURINE GENES WHICH AFFECT PIGMENTATION AND PLATELET FORMATION/FUNCTION. Richard T Swank, Edward K Novak, Edward P O'Brien and Michael E Rusiniak |
| 10:30 - 11:00 | CELL-SPECIFIC REGULATION AND SIGNAL TRANSDUCTION OF THE MELANOMA-INDUCING ONCOGENE Xmrk IN *XIPHOPHORUS* HYBRIDS. M Schartl, A Schartl, C Winkler, N Dimitrijevic, B Malitschek, C Wellbrock, M Pagany, M Baudler and J Altschmied |
| 11:00 - 11:30 | THE ROLE OF ENDOTHELINS IN PIGMENT CELL BIOLOGY. CE Gariepy, K Hosoda, A Greenstein Baynash, RE Hammer, EG Puffenberger, A Chakravarti and M Yanagisawa |
| 11:30 - 12:00 | CONTROL OF PIGMENTATION PATTERNS BY THE *AGOUTI* GENE. Greg Barsh |
| 12:00 - 1:30 pm | Lunch - **The Ritz-Carlton Roof-Top Restaurant** **Poster Viewing** |
| 1:30 - 3:30 pm | **Minisymposium A** - Alistair J Cochran/Garth L Nicolson - **Salon III**<br>"**Mechanisms of Metastasis and Approaches to Therapy**" |
| 1:30 - 1:45 | DYSFUNCTION OF SIALYL TRANSFERASE IN PRIMARY MELANOMA CORRELATE WITH HIGH RISK OF METASTASIS. Alistair J Cochran, Duan-Ren Wen, Pi-Xiang Li, Odile Berthier-Vergnes, Jean-Francois Doré and Luc Thomas |
| 1:45 - 2:00 | MECHANISM FOR CYTOCIDAL EFFECT OF PHENOLIC THIOETHER AMINE, N-ACETYL-4-S-CYSTEAMINYLPHENOL, AS A TARGETED CHEMOTHERA-PEUTIC AGENT FOR MALIGNANT MELANOMA. Kowichi Jimbow, M Ota and P Thomas |
| 2:00 - 2:15 | DEVELOPMENT OF IODINATED N-ACETYL-4-S-CYSTEAMINYLPHENOL AS A TOOL FOR DEVELOPMENT OF RADIOCHEMOTHERAPY OF MELANOMA. Kowichi Jimbow, Shadrha Singh, Daniel Chang and Adrian Gili |
| 2:15 - 2:30 | INDUCTION OF P21 $^{WAF-1/SDI-1}$ BY P53-DEPENDENT AND INDEPENDENT MECHANISMS IN PROLIFERATING AND SENESCENT MELANOCYTES. Estela E Medrano, Sungbin Im, Fan Yang, David Eling and Zalfa Abdel-Malek |
| 2:30 - 2:45 | MACROPHAGE CHARACTERISTICS OF METASTATIC MELANOMA. Michael L Rachkovsky, Stefano A Sodi, David Bermudes, Jean L Bolognia, Ashok K Chakraborty, Jennifer F Madison and John M Pawelek |
| 2:45 - 3:00 | MELANOMA FORMATION IN NON-HYBRID *XIPHOPHORUS*. A Schartl, B Malitschek, S Kazianis, R Borowsky and M Schartl |
| 3:00 - 3:15 | ANALYSES OF HYBRIDS FORMED *IN VITRO* BETWEEN CLOUDMAN S91 MELANOMA CELLS AND NORMAL MOUSE MACROPHAGES. Stefano A Sodi, David Bermudes, Ashok K Chakraborty, Gisela Moellmann, Seth J Orlow, Michael L Rachkovsky, Susana Rosemblat, Agnes Keh-Yen, Jean Bolognia and John Pawelek |
| 3:15 - 3:30 | CULTIVATION AND DIFFERENTIAL CHARACTERISTICS OF HUMAN OCULAR MELANOCYTES DERIVED FROM THE CHOROID, IRIS OR THE RETINAL PIGMENTED EPITHELIUM. Huiquan Zhao, Yang Zhao, James J Nordlund and Raymond E Boissy |

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| 3:30 - 4:00 pm | Break - **Salon II** **Poster Viewing** |
| 4:00 - 6:00 pm | **Minisymposium B** - Seth J Orlow - **Salon II** |
| | "**Melanosomes: Biogenesis & Structure**" |
| 4:00 - 4:15 | CHARACTERIZATION OF MELANOSOMAL TYROSINE TRANSPORT IN NORMAL AND PINK-EYED DILUTION MURINE MELANOCYTES. Brian Potterf, Donna Durham-Pierre, Murray H Brilliant, Vincent J Hearing and William A Gahl |
| 4:15 - 4:30 | ON THE ANALYSIS OF MELANOCYTES CULTURED FROM A PATIENT WITH OCAII. Huiquan Zhao, William S Oetting, Yang Zhao, Richard A King, Murray Brilliant and Raymond E Boissy |
| 4:30 - 4:45 | ROLE OF TRANSMEMBRANE AND CYTOPLASMIC DOMAINS IN THE INTRACELLULAR TRANSPORT OF MELANOSOMAL MEMBRANE PROTEINS. Setaluri Vijayasaradhi, Babita Persaud and Lisa Silbert |
| 4:45 - 5:00 | MISROUTING OF TYROSINASE WITH A TRUNCATED CYTOPLASMIC TAIL AS A RESULT OF THE MURINE *PLATINUM* ($c^p$) MUTATION. SJ Orlow, F Beermann, A Schmidt, RE Boissy, YL Boissy and ML Lamoreux |
| 5:00 - 5:15 | TYROSINASE REQUIRES A MOLECULAR CHAPERONE, CALNEXIN/p90 FOR EFFICIENT POST-TRANSLATIONAL GLYCOSYLATION AND ENZYME ACTIVITY. K Toyofuku, J Park, H Chen and K Jimbow |
| 5:15 - 5:30 | THE EFFECT OF *BROWN* MUTATION OF THE INTRACELLULAR TRANSPORT AND STABILITY OF THE MOUSE AND HUMAN *BROWN* LOCUS PROTEINS. Yiqing Xu, Setaluri Vijayasaradhi and Alan N Houghton |
| 5:30 - 5:45 | INTERACTION OF MELANOSOMAL PROTEINS WITH MELANIN. Philippe D Donatien and Seth J Orlow |
| 5:45 - 6:00 | MODULATION OF MELANOGENIC PROTEIN EXPRESSION DURING THE SWITCH FROM EU- TO PHEOMELANOGENESIS. Takeshi Kobayashi, Wilfred D Vieira, Brian Potterf, Chie Sakai, Genji Imokawa and Vincent J Hearing |
| 7:00 pm | **KC Barbecue Buffet/Banquet** at **Benjamin Ranch** |
| | Buses will leave from The Ritz-Carlton beginning 6:30 pm |

**TUESDAY, JUNE 27**

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| 8:00 - 5:00 pm | **Registration** Table Open |
| | **Speaker Preview** Room Open - **Pavilion V** |
| 8:00 am | Continental Breakfast - **Salon II** |
| 8:30 - 9:30 am | **Keynote Address** - by Roger D Cone - **Salon III** |
| | THE POSITIVE AND NEGATIVE HORMONAL CONTROL OF PIGMENTATION. Roger D Cone and Dongsi Lu |
| 9:30 - 10:00 am | Break - **Salon II** **Poster Viewing** |
| 10:00 - 12:00 | **Symposium II** - Bryan Fuller - **Salon III** |
| | "**Signaling Pathways in Pigmentation Regulation**" |
| 10:00 - 10:30 | REGULATION OF TYROSINASE GENE TRANSCRIPTION BY MSH/cAMP. Deepa Rungta, Todd Corn and Bryan Fuller |
| 10:30 - 11:00 | REDUCED MELANIN LEVELS IN MOUSE MELANOCYTES TRANSDUCED WITH A DOMINANT-NEGATIVE CREB MUTANT. Mayra Alvarez-Franco, Elaine Cheng, Susanne Wagner, Gisela Moellmann, Yuhua Chang, Seth Orlow and Ruth Halaban |
| 11:00 - 11:30 | REGULATION OF MELANOGENESIS IN B16 MOUSE MELANOMA CELLS BY PROTEIN KINASE C $\alpha$. Harish Mahalingam, Ken-ichi Yasumoto, Shigeki Shibahara and Richard M Niles |

11:30 - 12:00 PARACRINE REGULATION OF HUMAN MELANOCYTES BY α-MSH, ENDOTHELIN-1, BASIC FGF, AND THE AGOUTI PROTEIN. Zalfa Abdel-Malek, Viki Swope, Itaru Suzuki, Sungbin Im, Estela E Medrano, James Nordlund, Michael Ollmann and Gregory Barsh

12:00 - 1:30 pm Lunch - **(On your own)** **Poster Viewing**

1:30 - 3:30 pm **Minisymposium D** - Kenneth Mason - **Salon II**
"**Pigment Cell Genetics and Molecular Biology**"

1:30 - 1:45 EFFECTS OF THE *AGOUTI* LOCUS ON LEVELS OF α-MSH IN SERUM, PITUITARIES, AND REGENERATING HAIR BULBS OF MICE. David G Monroe, Maureen R Diggins, Partha Ramasastry, Ryan J Saetveit and Nels H Granholm

1:45 - 2:00 AGOUTI PROTEIN SUPPRESSES EXPRESSION AND ACTIVITY OF TYROSINASE AND TYROSINASE-RELATED PROTEINS IN MURINE MELANOCYTES. Chie Sakai, Michael Ollmann, Takeshi Kobayashi, Brian Potterf, Wilfred D Vieira, Minao Furumura, Gregory S Barsh and Vincent J Hearing

2:00 - 2:15 EFFECTS OF RECOMBINANT AGOUTI PROTEIN ON MELANOCORTIN BINDING AND SIGNALING. Ying-Kui Yang, Michael M Ollmann, Ira Gantz and Gregory S Barsh

2:15 - 2:30 *MITF*, A GENE FOR WAARDENBURG SYNDROME TYPE II (WSII), DRIVES MELANOCYTE DIFFERENTIATION. Masayoshi Tachibana, Kazunori Urabe, Kimberly A Meyers, Kazuhisa Takeda, Yoshitaka Nobukuni, Stuart A Aaronson and Toru Miki

2:30 - 2:45 MAPPING OF THE GENE FOR HERMANSKY-PUDLAK SYNDROME IN A PUERTO RICAN POPULATION USING HOMOZYGOSITY MAPPING. Scott C Wildenberg, William S Oetting, Carmelo Almadovar, Marcy Krumwiede, James G White and Richard A King

2:45 - 3:00 THE GENOMIC STRUCTURE OF THE HUMAN *P* GENE ASSOCIATED WITH OCA2 (TYROSINASE POSITIVE OCULOCUTANEOUS ALBINISM). Murray H Brilliant, William S Oetting, John M Gardner, Ada Ching, Donna Durham-Pierre and Richard A King

3:00 - 3:15 MUTATIONS AND POLYMORPHISMS OF THE HUMAN *P* GENE ASSOCIATED WITH P RELATED OCULOCUTANEOUS ALBINISM (OCA2). William S Oetting, Murray H Brilliant, John M Gardner, James P Fryer and Richard A King

3:15 - 3:30 THE EFFECTS OF CHELATED IRON ON THE TYROSINASE-MEDIATED OXIDATIONS OF 5,6-DIHYDROXYINDOLE AND 5,6-DIHYDROXYINDOLE-2-CARBOXYLIC ACID. Emily Vass and Anthony J Nappi

1:30 - 3:30 pm **Minisymposium E** - Elizabeth Topp - **Salon III**
"**Molecular, Physical-Chemical and Genetic Aspects of Pigmentation**"

1:30 - 1:45 UVB AND MSH STIMULATE mRNA PRODUCTION FOR αMSH RECEPTORS AND POMC-DERIVED PEPTIDES IN MOUSE MELANOMA CELLS AND TRANSFORMED KERATINOCYTES. Ashok K Chakraborty, Andrzej Slominski, Genady Ermak, James Hwang, Jean L Bolognia and John M Pawelek

1:45 - 2:00 THE DIFFERENTIAL RESPONSES OF HUMAN MELANOCYTES FROM DIFFERENT SKIN TYPES TO UV LIGHT ARE ALTERED BY α-MSH. Sungbin Im, Estela E Medrano, David Mitchell, James Nordlund and Zalfa Abdel-Malek

2:00 - 2:15 EFFECT OF INDUCED PIGMENTATION IN CLOUDMAN S91 MOUSE MELANOMA CELLS ON MUTATION TO OUABAIN RESISTANCE AFTER UVC. Weixiong Li, Patrick Xin, George J Hill and Helene Z Hill

2:15 - 2:30 THE MITOGENIC AND MELANOGENIC STIMULATION OF HUMAN MELANOCYTES BY MELANOTROPIC PEPTIDES CORRELATES WITH

THEIR ABILITY TO BIND AND ACTIVATE THE MC1 RECEPTOR. Itaru Suzuki, Roger Cone, Sungbin Im, James Nordlund and Zalfa Abdel-Malek

2:30 - 2:45 MOLECULAR MECHANISM FOR THE CATALYSIS BY A ZINC-ENZYME: DOPACHROME TAUTOMERASE. F Solano, C Jiménez-Cervantes, J H Martínez-Liarte, JC Garcia-Borrón, JR Jara and JA Lozano

2:45 - 3:00 ULTRAVIOLET IRRADIATION INDUCES DOWN-REGULATION OF TYROSINASE ACTIVITY IN CULTURED HUMAN CHOROIDAL MELANOCYTES AND RETINAL PIGMENTED EPITHELIAL (RPE) CELLS. Huiquan Zhao, Yang Zhao, Raymond E Boissy and James J Nordlund

3:00 - 3:15 DO LAG KINETICS OF TYROSINASE HAVE ANY PHYSIOLOGICAL FUNCTION? Abburi Ramaiah

3:15 - 3:30 CHARACTERIZATION OF SOME SYNTHETIC ANALOGUES OF NEUROMELANIN BY X-RAY DIFFRACTION. Pier Raimondo Crippa, Melvin Eisner and Simon C Moss

3:30 - 3:45 DOES 5-AZACYTIDINE ALTER THE INCIDENCE AND EXPRESSION OF AUTOIMMUNE VITILIGO IN THE SMYTH LINE CHICKEN? GP Sreekumar and JR Smyth, Jr

3:30 - 4:00 pm Break - **Salon II** **Poster Viewing**

4:00 - 6:00 pm **Minisymposium C** - Randall Morrison - **Salon II**
"**Animal Models of Pigment Formation**"

4:00 - 4:15 AN ULTRASTRUCTURAL ANALYSIS OF PIGMENT PATTERN IN ZEBRAFISH. Randall Morrison and Kunio Nagashima

4:15 - 4:30 PHYSICAL AND HISTOLOGICAL ANALYSIS OF BLUE COLORATION IN FROG SKIN. Philip J Fernandez, WA Coghlan, Kere Arnold and Charles Zovko

4:30 - 4:45 MOLECULAR AND DEVELOPMENTAL MECHANISMS UNDERLYING DORSAL-VENTRAL DIFFERENCES IN MOUSE PIGMENTATION. Sarah E Millar, Yanru Chen, David MJ Duhl and Gregory S Barsh

4:45 - 5:00 GENETIC INTERACTIONS COORDINATING PIGMENT PATTERNING IN *PIEBALD* MICE. Hyangshuk Rhim, Mickie Cheng, Shirley Tilghman and William Pavan

5:00 - 5:15 SPONTANEOUS PIGMENT CELL TUMORS IN A CLONE OF THE AMAZON MOLLY *POECILIA FORMOSA*. A Schartl, HK Müller-Hermelink, G Krohne, R Wacker, I Nanda, M Schmid, I Schlupp, J Parzefall and M Schartl

5:15 - 5:30 PRENATAL X-RAY-PROVOKED TRANSGENERATIONAL ONCODETER-MINANTS IN XIPHOPHORINE PIGMENT CELLS. Annerose Anders, Christine Fleming, Fritz Anders, Helga Schneider, Eckart Schneider, Harald Gröger, Jürgen Kiefer

5:30 - 5:45 THE MULTI-THERAPY RESISTANCE FACTOR (MTRF) PRODUCED BY CLOUDMAN S91 MELANOMA CELLS IS INHIBITED BY RABBIT ANTISERUM. Shuangwen Zhou, Friedrich Kueppers, H Colleen Silva, George J Hill and Helene Z Hill

5:45 - 6:00 AGOUTI MEDIATED ALTERATIONS OF THIOL CONCENTRATIONS IN REGENERATING HAIR FOLLICLES, SERUM, AND EXTRAFOLLICULAR TISSUES OF AGOUTI MUTANT MICE. David E Granholm, R Neil Reese and Nels H Granholm

7:00 pm **Jazz Pub Crawl**

7:15 pm **Riverboat Gambling**

## WEDNESDAY, JUNE 28

8:30 - 10:00 am **Poster Session** and Strolling Brunch - **Salon II**

PRESENCE OF MELATONIN BINDING SITES IN S-91 MURINE MELANOMA CELLS. Ana LM Almeida, Regina P Markus, Maria A Visconti and Ana ML Castrucci

STRUCTURE OF THE MOUSE TRP-2 (DOPACHROME TAUTOMERASE) GENE AND SEQUENCE OF TWO NOVEL *SLATY* ALLELES. Peter S Budd and Ian J Jackson

RHYTHMIC COLOR CHANGE IN THE AMPHIBIAN, *RANA CATESBEIANA*. Carolina R Camargo, Maria Aparecida Visconti and Ana Maria de L Castrucci

MOLECULAR AND BIOCHEMICAL CHARACTERIZATION OF A UNIQUE MELANOGENIC INHIBITOR ISOLATED FROM TRANSPLANTED HUMAN SKIN. Jamal Farooqui, Ed Robb, Glenn Warden and James Nordlund

MECHANISM OF MELATONIN DESENSITIZING ACTION ON *BUFO ICTERICUS* PIGMENT CELL RESPONSES to α-MSH. Ana MC Filadelfi and Ana ML Castrucci

SEROTONIN AND N-ACETYLSEROTONIN EFFECTS ON PIGMENT CELLS OF THE TOAD *BUFO ICTERICUS*: CHARACTERIZATION OF MELATONIN RECEPTORS. Ana MC Filadelfi and Ana ML Castrucci

FUNDULUS HETEROCLITUS (LINNAEUS) MELANOPHORE: REVISITED. Maher M Haddad, Premchand Anne, Victoria A Kimler and John D Taylor

THE SYNTHESIS AND MECHANISTIC STUDIES OF STRUCTURED DOPA MIMETICS. Julius L Harp and Jesse M Nicholson

BINDING ASSAYS OF 8-METHOXYPSORALEN IN MELANOTIC AND AMELANOTIC HUMAN MELANOMA CELLS. Mauro C Isoldi, Ana Christina Scarparo, Adelaide Faljoni-Alário, and Ana Maria de L Castrucci

SUBSTRATE SPECIFICITY OF HUMAN TYROSINASES. Celia Jiménez-Cervantes, Enrique Benedito, Francisco Solano, Ghanem Ghanem, Véronique del Marmol, and José C García-Borrón

CLONING AND EXPRESSION OF A RED PIGMENT CONCENTRATING HORMONE (RPCH)-PRECURSOR mRNA IN THE BLUE CRAB, *CALLINECTES SAPIDUS*. Jörg M Klein, Carl J Mohrherr, Frank Sleutels, Nicola Jaenecke, John P Riehm and K Ranga Rao

THE EFFECTS OF CYSTEINE ON AXOLOTL NEURAL CREST CELL DIFFERENTIATION IN CULTURE. Jonathan Muyskens, Ken Mason and Sally Frost-Mason

CHARACTERIZATION OF ONCODETERMINANTS ADDING TO MELANOMA DEVELOPMENT TRANSGENERATIONALLY. Harald Petry, Kerstin Petry, Petra Brix, Christine Fleming, Arne Faisst, Gerhard Hunsmann, Wolfgang Lüke, Fritz Anders

COORDINATE AND NON-COORDINATE REGULATION OF GENES FOR MELANOGENESIS. Thomas P Powers and Richard L Davidson

INHIBITION OF UVR-INDUCED TANNING BY TOPICAL APPLICATIONS OF VITAMINS C AND E TO THE SKIN OF HAIRLESS MICE. Walter C Quevedo, Jr, Thomas J Holstein, Jacob Dyckman, Charles J McDonald, David Friedman and Ernest L Isaacson

SPECIFICATION AND MIGRATION OF MELANOCYTES IN THE AVIAN EMBRYO. Mark V Reedy and Carol A Erickson

DYNEIN AS A CANDIDATE MOTOR MOLECULE FOR PIGMENT AGGREGATION IN GOLDFISH XANTHOPHORES. M Carolina Tuma, Darl R Swartz, David Asai and Soo-Siang Lim

10:00 - 10:15 am Presentation of the **PASPCR Career Achievement Award - Salon III**

10:15 - 11:45 am **Symposium III** - Robert E Palazzo - **Salon III**

10:15 - 10:45 APPROACHING CENTROSOME BIOCHEMISTRY. Robert E Palazzo

10:45 - 11:15 AVIAN NEURAL CREST CELLS CAN MIGRATE IN THE DORSOLATERAL PATH ONLY IF THEY ARE SPECIFIED AS MELANOCYTES. CA Erickson

11:15 - 11:45 THE RETINAL PIGMENTED EPITHELIUM IS REQUIRED FOR DEVELOPMENT AND MAINTENANCE OF THE MOUSE EYE. Sophie Raymond and Ian J Jackson

12:00 **Society Business Meeting & Awards Presentation - Salon III**

## XVI$^{th}$ IPCC (International Pigment Cell Conference) by Roger Bowers, Frank Meyskens

The XVI$^{th}$ International Pigment Cell Conference will be held from October 29$^{th}$ to November 3$^{rd}$, 1996 at the Disneyland Hotel in Anaheim, California. Frank Meyskens is the Organizer of this meeting with Roger Bowers and Alistair Cochran serving as co-chairs of the Organizing Committee. More information regarding this meeting (and its interesting venue) will be forthcoming in future Newsletters and in separate mailings. Dr. Meyskens has asked that the following announcements be included in our Newsletter.

**Satellite Conferences**: No satellite conferences will be supported by the local organizing committee that are held within the time frame of the XVIth International Pigment Cell Conference, Tuesday, October 29, 1996; 6:00 pm to Sunday, November 3, 1996; 8:00 am. There are a wide number of venues possible to hold small or large satellite conferences either before or after the main pigment cell meeting. Our Memorial/UCI Educational Foundation will be happy to work with you in planning, for a small fee, and we request that we be notified of the intent of any satellite conference no later than June 1, 1995. If we are notified later than this date, accommodations and planning availability cannot be guaranteed.

**Competitive Stipend for Travel Support**: The Organizing Committee will provide funds in a competitive manner for graduate students, post-doctoral fellows and those within five years of formal academic appointment. The number of stipends will depend on the availability of funds and further information will become available during the second and subsequent informational mailings.

## PASPCR Secretary / Treasurer's Report : by Richard A King

Following is a synopsis of the PASPCR Council Meeting held by teleconference on January 19, 1995 . . .

Hearing opened the meeting, a quorum was declared present, and the new members were welcomed to the council: A. Houghton, W. Oetting and W. Quevedo. The minutes of the October 27, 1994, council meeting were accepted.

Frost-Mason, Nominating Committee chair, reported that the 1995 nominating committee is being organized. Jacobsohn will be the Council member and three members-at-large will be appointed. Hearing noted that the membership included individuals residing outside of the United States and corporate members and the committee and the nominees should reflect this. Hearing asked Oetting to chair the Publication Committee and be in charge of the Newsletter. All council members agreed that the Newsletter was valuable and should be continued. Hearing asked Orlow to chair the Membership Committee. King reviewed the draft of the membership brochure that is being prepared and comments and suggestions for its improvement were made. Nordlund, representing the Awards Committee, reported that a conference call was held to discuss the 1995 Gelb Lectureship. Shirley Tilghman will be the 1995 Gelb Lectureship recipient. Hearing reviewed the Career Achievement Award for 1995 and indicated that nominees had not been included in the Nominating Committee's activities. Potential candidates were reviewed and the Council will decide by written ballot. Honorary Membership for 1995 was reviewed. Nominations were reviewed, and it was noted that the Rule and Regulation describing honorary membership did not address the issue of society membership. This issue was tabled until this Rule and Regulation could be further reviewed.

Frost-Mason gave an update on the 1995 Kansas City meeting. A call for the abstracts will be distributed in mid-February, and abstracts will be due at the end of March. Major symposia will be held in the morning and two concurrent sessions in the afternoons.

Under old business, King reviewed the amendment to the Young Investigator Award Rule and Regulation which required that candidates for Young Investigator Awards be nominated at the time of abstract submission. The reasons for proposing this amendment were reviewed. This procedure will be tried for the Kansas City meeting and its benefits assessed after the meeting. Under new business, King proposed a new Rule and Regulation: an election that ends in a tie vote by the membership would be decided by a written ballot of the nine-person Council that is in existence in the year of the election. This was unanimously approved.

Quevedo with H. Swartz has proposed to hold the 1997 meeting in Providence, Rhode Island, on June 15-18. Their proposal letter and tentative budget for the meeting were reviewed. This proposal was unanimously approved by the council.

The minutes have been prepared by R. King, Secretary-Treasurer

## Meeting Report : by Roger Cone

**Annual Meeting of the Federation of American Societies for Experimental Biology (FASEB)**
**Atlanta, GA April 9-13, 1995**

A session entitled "Melanocortin Receptor Function and Signalling: Neural, Pigmentary and Immunomodulatory Functions", organized by Dr. Jeffrey Tatro (New England Medical Center, Boston), brought together several of the investigators interested in this receptor family at the recent FASEB meeting in Atlanta. While a good deal of the data presented concerned the actions of α-MSH and melanocortin receptors in physiological processes other than pigmentation, a deeper understanding of the melanocortin receptors expressed in the brain and elsewhere (MC2-R, MC3-R, MC4-R and MC5-R) is likely to add to our knowledge of the structure and function of the MSH receptor on the melanocyte. Dr. Tatro began the session with a review of his work on the distribution of MSH binding sites in the brain, and with the assertion that - traditionalists beware! - the melanocyte MSH receptor be hereafter called the MC1 receptor to distinguish it from the three recently discovered receptors that bind α-MSH and other melanocortin peptides. These have been named, in the order of their cloning, the MC3, MC4 and MC5 receptors. It's probably not a bad idea, and in any event, the official names for the gene loci for these receptors are MC1R-MC5R in man and mC1R-mC5R in the mouse.

Linda Roselli-Rehfuss (ICRM, Montreal) presented an overview of the cloning and characterization of the 5 melanocortin receptors and then presented new data on the expression of POMC and MSH-R mRNAs in a panel of human and murine melanoma cell lines. Many melanomas continue to express the MSH receptor (oops! MC1-R) and if POMC is also expressed and processed into melanocortin peptides then there is the possibility of a cAMP autoregulatory loop in these cells that could in various ways affect tumor progression. While expression of immunoreactive POMC peptides is seen in some melanomas and full-length 1.1kb POMC mRNA has been demonstrated in primary melanocytes, Roselli-Rehfuss sought to identify POMC mRNA in melanoma cell lines. Only the truncated 800bp POMC mRNA was identified by Northern hybridization in the Cloudman and B16 melanoma cell lines, however RT-PCR was then used to demonstrate the presence of the full-length 1.1kb transcript in both cell lines. Dr. Ira Gantz (University of Michigan) presented a comparative analysis of the pharmacology of all 5 human melanocortin receptors, and in agreement with previous reports demonstrated that in fact, unlike the murine MC1-R, the human MC1-R is equally responsive to α-MSH and ACTH. Dr. Gantz also presented work performed in collaboration with Dr. Greg Barsh (Stanford) on the pharmacology of baculovirus-produced human agouti signalling protein (ASP). Similar to mouse agouti, human agouti is a potent antagonist of both the human MC1-R ($IC_{50}$= $2x10^{-10}$M) and MC4-R ($IC_{50}$=$1.8x10^{-10}$M). Interestingly, Dr. Gantz also showed data demonstrating that human agouti might antagonize each of the other melanocortin receptors at 100-1000 times higher doses, although a pure preparation of bona fide mammalian agouti forms may ultimately be necessary to resolve the fine points of agouti pharmacology.

Dr. Victor Hruby (University of Arizona) presented a very thorough discusion of the evolution of models of the active structure of α-MSH, based on many years of structure-activity studies in his laboratory. Two exciting new series of α-MSH compounds were also presented. One series consisted of a polyvinyl alcohol backbone containing multiple fluoroisothiocyanate moities and multiple copies of an α-MSH analogue conjugated via either disulfide or thioether linkage. These multivalent ligands appear to be useful for detection of levels of MSH receptor protein undectable by conventional radioligand binding assay. A second series of compounds were characterized in collaboration with my laboratory. These consisted of cyclic lactam analogues of α-MSH containing modifications of the D-Phe residue at position 7. In particular, introduction of a D-paraiodophenylalanine or D-naphthylalanine at position 7 resulted in extremely potent antagonists for the MC3 and MC4 melanocortin receptors. These compounds were full agonists of the mammalian MSH (MC1) receptors but are probably illustrative of the types of compounds one would need to make to produce antagonists of the MC1-R.

Finally, Robert Starr and Jim Lipton (U.T Southwestern) presented data suggesting expression of the MC1 receptor mRNA in the RAW macrophage cell line. While pharmacological data was not presented to confirm the presence of functional MC1 receptors in these cells, effects of $10^{-8}$M α-MSH on nitric oxide synthase were documented, suggesting one possible mechanism for some of the reported anti-inflammatory effects of peripherally administered α-MSH.

**Meeting Report :** **by Zalfa Abdel-Malek**

**Annual Meeting of the Society for Investigative Dermatology**
**Chicago, IL May 24 - 28, 1995**

**GENERAL PLENARY PRESENTATIONS:**

Lerner M. et al. - Numerous MSH antagonists were synthesized and screened by determining their inhibition of frog skin darkening *in vivo* and the darkening of cultured frog melanophores. The most potent antagonist was a tripeptide D-Trp-Arg-Leu which also acted as an antagonist of α-MSH on the human MCIR.

Böhm M. et al. - Transfection of murine melanocytes with a deletion mutant adenovirus oncogene E1A which codes for a protein that binds p105, p107 and p60 cyclin A, but not p300 resulted in the same changes caused by the wild type E1A which sequesters all of the above proteins. These changes include autonomous growth, loss of dendricity and pigmentation due to the suppression of tyrosinase, TRP-1 and pmel-17, but not TRP-2 or the p gene product. Melanocytes transfected with EIA mutant that only sequesters p300 did not become autonomous and exhibited significant reduction in their growth rate.

Im S. et al. - Response of melanocytes to UVB light, unlike their response to UVC light, does not involve tyrosine phosphorylation and activation of Raf-1 or ERK2, yet induces the prolonged expression of c-fos, p53 and $p21^{Waf-1/SDI-1/Cip1}$. Prolonged expression of the latter protein inhibited the phosphorylation of the retinoblastoma tumor suppressor gene product, and thus blocked UVB irradiated melanocytes in $G_1$ phase of the cell cycle.

Kupper TS and Sarkar S - The chromosomal region 9p21 harbors melanoma tumor suppressor gene(s) that is often deleted in melanoma. One gene that has been already identified is $p16^{INK4}$. Additionally, in 7 of 10 melanomas, p15 deletion was observed. The expression of p15 was found to be upregulated by TGFβ, leading to inhibition of cell growth.

Eller et al. - UV-induced DNA damage and/or its repair may be a signal for melanogenesis mimicked by pTpT. Introduction of the restriction enzyme Pvu II into S91 melanoma cells as well as treatment with methylmethane sulfonate increased melanogenesis by increasing the mRNA and protein levels of tyrosinase.

**CONCURRENT SESSION PRESENTATIONS:**

Grimes PE et al. - Depigmented and uninvolved skin of 20 patients with vitiligo, and 18 control subjects were examined for the presence of viral (HIV, HTLV, herpes simplex, varicella zoster and cytomegalovirus) genomes by PCR screening of paraffin-embedded sections. Cytomegalovirus DNA was found in the depigmented and uninvolved skin of some patients with vitiligo but not in any of the controls.

Hann SK et al. - Antibodies to the VIT90, VIT75 & VIT40 antigens are thought to be involved in the pathogenesis of vitiligo. 10 patients who had at least two of the above antibodies were included in the study. After 2-4 months of treatment with 0.3 mg/kg daily prednisolone, 9 out of 10 patients repigmented, and 7 out of the 9 had a significant reduction of their antibody levels.

Wagner SN et al. - Pmel17 encodes HMB45 antigen which is expressed at very low levels in normal melanocytes and has highest expression level in metastatic melanoma cells. Pmel17 nonamers have epitopes recognized by cytotoxic T lymphocytes, and thus may be utilized as an antigen for immunotherapeutic strategies.

**CONCURRENT ORAL SESSION:**

Gruis NA et al. - P16 19 base deletion was observed in 13 out of 15 Dutch families. Two family members were homozygous for the deletion, suggesting the presence of functionally redundant genes that can fulfill the role of p16 in its absence. The occurrence of familial atypical multiple mole-melanoma without the p16 19 base deletion questions the role of p16 as the melanoma gene.

Puig S et al. - Loss of heterozygosity at 9p in many cutaneous malignant melanoma tumors. The extent of the deletions correlated with metastic potential.

Dietrich et al. - High surface expression of CD44 on primary melanomas was associated with increased metastatic risk and poor survival.

Meyer et al. - P16 mRNA is evident in human melanocytes nevi, primary melanoma cells, and established melanoma cell lines with significant pattern of altered expression.

Fujita et al. - Mutant N-ras and H-ras overexpression in a melanoma cell line with low invasive potential and lack of growth in soft agar induced colony formation, anchorage-independent growth, and invasive potential on matrigel.

Hara M. et al. - Media conditioned by cultured human keratinocytes, particularly UV irradiated keratinocytes, stimulated the dendricity of melanocytes. Antibodies against endothelin-1 blocked this effect, while endothelin-1 treatment increased melanocyte dendricity. Kinesin, found to be associated with β-tubulin and melanosomes, was required for dendrite formation and melanosomal transfer.

Andersen W. et al. - Amyloid precursor protein is synthesized by human melanocytes and melanoma cells. Its synthesis is increased when melanocytes are plated onto laminin, and in response to UV light. Conditioned melanocyte medium contains β-amyloid fragment which was toxic to melanocytes at high concentrations, and increased dendricity at low concentrations.

Yaar M. et al. - NGF protects melanocytes from UV induced apoptosis by inducing BCL-2, and $p21^{sdi-1}$ expression. UV increases p75 NGF receptor expression. In the presence of NGF, p75 receptor expression protects from apoptosis, while increased p75 receptor expression in the absence of NGF enhances apoptosis.

Von Tschirschnitz et al. - α3β1 integrin on keratinocytes mediates adherence to epiligrin. α3 is a major integrin mediating melanocyte-keratinocyte attachment and cell matrix contact of melanocytes to extracellular matrix. α3 inhibits melanocyte proliferation while stimulating keratinocyte growth.

Abdel-Malek Z et al. - Human melanocytes respond to a single irradiation with UV light with a dose dependent inhibition of proliferation due to arrest in $G_1$ phase of the cell cycle and increased cell killing. Melanocytes from skin types I & II differ from those derived from skin types IV-VI in their inability to increase melanin synthesis decreased ability to resume proliferation after UV irradiation, and in their prolonged induction of p53 expression. Also, lightly pigmented melanocytes encounter more cyclobutane pyrimidine dimers at each of the UV doses used than their heavily pigmented counterparts.

Park et al. - PKCβ causes serine-threonine phosphorylation of tyrosinase. Only 5 serine/threonine residues in the cytoplasmic domain, out of a total of 60 residues found in tyrosinase were phosphorylated by PKCβ. Depletion of PKCβ by chronic TPA treatment reduced tyrosinase activity but did not alter the expression of tyrosinase at the mRNA or protein levels. TPA treatment increased the protein level of TRP-2.

Scott G & Hiang H - $p125^{FAK}$ was equally expressed in human melanocytes, in the human melanomas SK28 line (which grows as monolayer) and in SK1 line (which grows in suspension). Tyrosine phosphorylation of $p125^{FAK}$ occurred in human melanocytes and SK28, but not in SK1 melanoma cells. β1-integrin-activating antibody enhanced $p125^{FAK}$ phosphorylation only in human melanocytes. Paxillin, the substrate for $p125^{FAK}$ was expressed by the above cell types but did not become phosphorylated in any in response to β1 integrin activating antibody.

Halaban et al. - The melanocyte mitogens endothelin-1, hepatocyte growth factor and stem cell factor induce phosphorylation of ser133 within the kinase induced domain of CREB. Synergistic interaction of these mitogens prolonged the phosphylation of CREB and the activity of the MAPK2 cascade. CREB phosphorylation was mostly due to the activation of $p90^{RSK}$ and to a lesser extent $p70^{S6K}$. The activity of the latter was required for melanocyte proliferation.

Alvarez-Franco et al. - Transfection of immortalized normal black mouse melanocytes with recombinant CREB derivatives carrying mutations in the transactivating domain (dominant negative CREB S133A), reduced melanin content, and the levels of tyrosinase and TRP-2 without altering the levels of TRP-1, Pmel17 or pink-eye dilution proteins.

## MINISYMPOSIUM PRESENTATIONS

Boissy RE et al. - Melanocytes were cultured from an African American patient with oculocutaneous albinism who exhibited the brown phenotype, and his normal fraternal twin brother. Melanocytes of the patient contained mostly amelanotic melanosomes, exhibited DOPA positive reactivity,

normal tyrosine hydroxylase activity when assayed in cell lysates, decreased dopa oxidase activity and lacked the expressed of TRP-1 mRNA or protein. The patient was found to be homozygous for a single bp deletion in exon 6.

Medrano EE et al. - Cells from human melanocytic origin express the human h-ski oncogene product in this order: adult melanocytes < nevus cells < neonatal melanocytes = atypical melanocytes < primary melanomas = metastatic melanoma. H-ski protein is phosphorylated by melanocyte specific mitogens, and its expression is decreased 4-8 hours after UV irradiation. Overexpression of h-ski in melanoma cells accelerated the UV induced cell death.

## POSTER PRESENTATIONS

Imokawa G et al. - Proliferation of human melanocytes is stimulated by medium conditioned by human fibroblasts, particularly fibroblasts derived from old donors. This mitogenic activity was blocked by tyrosine kinase inhibitors and was mostly due to the presence of hepatocyte growth factors and stem cell factor. The release of both factors from young, but not old fibroblasts was induced by IL-1.

Lee JH and Park KC - Medium conditioned by UV irradiated human keratinocytes elicited an increase in the proliferation of cultured human melanocytes, as determined by MTT assay and $^{3}$H-thymidine incorporation. This conditioned medium reduced dopa oxidase activity and melanin content.

Rallis TM et al. - C-jun mRNA expression was not different among human melanocytes, nevus cells, and melanoma cells, maintained under serum conditions, serum starved conditions, or in the presence of retinoic acid. c-fos mRNA was not detectable in any of the above cell types, however after serum starvation, c-fos expression increased in nevus cells and 1 out of 3 melanoma cell lines.

Kubilus J et al. - Incorporation of normal human melanocytes into a model of the human epidermis resulted in the proper localization of these cells which were dopa-positive in the basal layer. UVB exposure of these cultures exhibited an increase in viability retention of 5-10% compared with melanocyte free cultures.

Slominski A et al. - Cyclophosphamide or dexamethasone cause disruption of follicular melanogenesis. Only cyclophosphamide causes toxic damage to melanocytes, inhibits melanosome formation and induces pathogenic transfer of melanosomes into the internal and outer root sheath and proximal matrix. However, cyclophosphamide increased while dexamethasone decreased TRP-2 activity.

Norgauer J et al. - Two important steps in metastasis, the avidity of the vitronectin receptor and actin network organization in melanoma cells are regulated by phosphatidylinositol 3-kinase. Inhibition of this kinase abolishes the interaction of $\alpha V\beta 3$ integrin receptor with vitronectin and causes the breakdown of actin stress fibers.

Piepkorn M - Human keratinocytes produced more sulfated glycosaminoglycans than did melanocytes from the same donor. Keratinocytes mostly produced heparin sulfates while melanocytes produced chondroitin sulfates. With increased cell density growth inhibition, chondroitin sulfate proteoglycan synthesis decreased in both cell types, but only keratinocytes demonstrated increased heparin sulfate proteoglycan.

Farooqui J et al. - A potent epidermal melanogenic inhibitory protein has been purified from human skin grafted onto athymic nude mice. This protein has a molecular weight of 14 KDa, a pI of 7.5 and is highly homologous to the fatty acid binding proteins that play a role in the storage and transport of essential fatty acids from the liver to the skin.

Suzuki I et al. - The melanotropins $\alpha$-MSH and ACTH stimulate human melanocyte proliferation and melanogenesis. Comparison of the effects of $\alpha$-, $\beta$-, $\gamma$-MSH and ACTH revealed that $\alpha$-MSH and ACTH have the highest affinity to the MC1 receptor, followed by $\beta$-MSH then $\gamma$-MSH. This order of affinity correlated with the ability of these peptides to increase cAMP formation, tyrosinase activity and melanocyte proliferation.

Seline PC et al. - Expression of E-cadherin which may function to maintain the integrity of the epidermal-melanin unit, was compared in primary melanomas or metastatic lesions, using human melanocytes as a control. It was found that melanoma cells express E-cadherin inversely to disease progression.

Horikawa T et al. - Melanocyte movement induced by melanocyte specific mitogens occurs via different signaling pathways from those of proliferation. Movement induced by bFGF or

endothelin-1 was inhibited by pertussis toxin, and recovered upon the addition of db cAMP. Dibutyryl cAMP which is mitogen to melanocytes, inhibited melanocyte movement. The effect of endothelin-1 on movement was not inhibited by the tyrosine kinase inhibitor genistein.

Norris D et al. - Melanization was found to augment apoptosis in melanocytes induced by ionophore, UV light and oxidative stress. Comparison of two mouse melanocyte cell lines, Melan-A which is very melanotic, and Melan-C which lacks functional tyrosinase, revealed that Melan-A demonstrates increased apoptosis and necrosis in comparison to Melan-C in response to the above inducers.

Böhm M et al. - Melanocytes and melanoma cells contain neuronal type nitric oxide synthase which colocalizes with NADPH-diaphorase which serves as an obligatory electron donor in the synthesis of nitric oxide. NADPH-diaphorase in melanocytes could be stimulated by norepinephrine, and inhibited by $N^G$-methyl-L-arginine. Similar levels of nitric oxide synthase were detected in human melanocytes and melanoma cells.

Ao Y et al. - Treatment of S91 melanoma cells with α-MSH results in increased tyrosinase mRNA level within 24 hours, an effect that is due to increased transcription rate, and not to increased half life of tyrosinase specific mRNA. Depletion of PKC by phorbol ester treatment inhibited the α-MSH induced increase in tyrosinase mRNA and reduced basal tyrosinase mRNA as well.

Ali W et al. - Immunohistochemical staining of melanocytes in the keratinocytic tumors basal cell carcinoma and squamous cell carcinoma revealed the following: increased immoreactiity to NKI/beteb and HMB 45 antibodies in hyperpigmented basal cell carcinomas. No consistent differences could be detected with anti-α-MSH or anti-stem cell factor between these two tumor types. However, increased bFGF expression was detectable in pigmented basal cell carcinomas.

Yaar et al. - Fibroblasts and keratinocytes enhance melanocyte survival and function in the skin. UV irradiation of dermal equivalent model containing fibroblasts and melanocytes resulted in a significant reduction of melanocyte death, in comparison to melanocytes irradiated in monolayer, increased migration of melanocytes toward keratinocytes and increased melanocyte dendricity and increased melanogenesis.

Jimbow et al. - Two cell lines of vitiligo melanocytes from hyperpigmented skin adjacent to the non-treated vitiligo leukoderma showed a 3-4 fold increase in TRP-1 expression, and abnormal protein-protein interaction with calnexin due to altered melanosomal glycoprotein intermediates, in comparison to normal melanocytes. These abnormalities might lead to loss of TRP-1 function and to apoptosis in response to oxidative stress.

Searles GE and Jimbow K - Melanocytes and melanomas express the integrin subunits β, α3, α2 and α5. Radical growth phase tumors had the highest integrin expression levels, and expression decreased with tumor progression. Radical phase tumors had poor adhesion to all substrates tested, a characteristic that was restored by bromodeoxy uridine treatment. The levels of expression of integrins induced by bromodeoxyuridine was characteristic of the expression in more advanced tumor progression phase, while expression induced by α-MSH was similar to the expression seen in less advanced tumor progression phase.

LePoole IC et al. - A melanocyte cell line was developed by introducing the E6 and E7 genes of human papilloma virus type 16 into a pure primary melanocyte culture. This cell line grows for more passages than expected of normal melanocyte cultures, express E6 and E7 mRNA, has a 50% increase in proliferative rate and higher expression of several melanoma markers than the parental cell line, and also exhibits clonal growth in soft agar.

Kawa et al. - In cultured neural crest mouse cells, stem cell factor (SCF) seems to play a role in the appearance of c-kit positive cells (melanocyte precursors). The presence of SCF during the first 5 days in culture is required to induce c-kit positive cells. The effects of SCF on Dopa positive (which appear between days 6-9 in culture) and mature melanocytes were markedly enhanced by the presence of cholera toxin, which increased the expression of tyrosinase in these cells.

Roy et al. - E-cadherin expression was compared in compound melanocytic nevi, intradermal nevi, primary invasive malignant melanomas and metastatic melanoma. In nevi, there was a loss of expression with increasing depth of infiltration, while invasive malignant melanomas showed more E-cadherin reactivity. Metastatic melanomas had greater loss of E-cadherin than primary melanomas.

Koppula S et al. - Three MSH receptor alleles have been characterized using genomic DNA extracted from skin samples of patients with different skin types. The first allele corresponds to the originally published sequence; the second contained a Valine to Methionine substitution at position 92, and is present in skin types I and II predominantly in individuals with blue yes and blond hair. The third allele contained an Aspartic acid to Glutamic acid substitution at position 84 (D84E). Functional analysis of the latter two alleles is currently ongoing.

## Members in the News

**Roger Cone** presented a Symposium lecture at the FASEB meeting recently held in Atlanta (cf meeting report above).

**Aaron Lerner** received the first D. Martin Carter Award for excellence in mentorship.

**Estela Medrano** co-chaired the concurrent oral session on Pigment Cell Biology at the annual meeting of the Society for Investigative Dermatology. She also gave a general plenary, as well as a minisymposium presentation.

## Positions - Wanted and Available :

**Predoctoral Position** - Opportunity available to do graduate studies towards a doctoral degree at the University of Cincinnati College of Medicine. Graduate program is through the Department of Cell Biology, Neurobiology, & Anatomy. Dissertation project would focus on molecular biology of the melanocyte physiology and pigmentary diseases. For information contact: Raymond E. Boissy, Ph.D., Department of Dermatology, University of Cincinnati College of Medicine, 231 Bethesda Avenue ML-592, Cincinnati, Ohio 45267-0592; (513)558-6242 [TEL]; (513)558-0198 [FAX]; boissyre@ucbeh.san.uc.edu [eMAIL].

**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 Update : **by Vincent Hearing**

The INTERPIG database is underway! Entries have been drifting in from members of the ESPCR, JSPCR and PASPCR listing their various reagents, cDNAs, antibodies, cell lines and other research materials that are useful to pigment cell researchers. A copy of the database entry sheet is enclosed again in this Newsletter and please take a moment to fill out that form with any items that you have developed over the past years that would be useful to other researchers in the field. Later this year we will be providing printouts of those items entered - the usefulness of this database

depends entirely upon the willingness of all of us to take the time to input our data into the database. Please take a moment to fill out your entries today. Mail or FAX them to V Hearing at the address listed on the form.

## Bibliography :

The Bibliography published in this issue covers the period February, 1995 through April, 1995. 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.

### MELANINS, MELANOGENS & MELANOGENESIS


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