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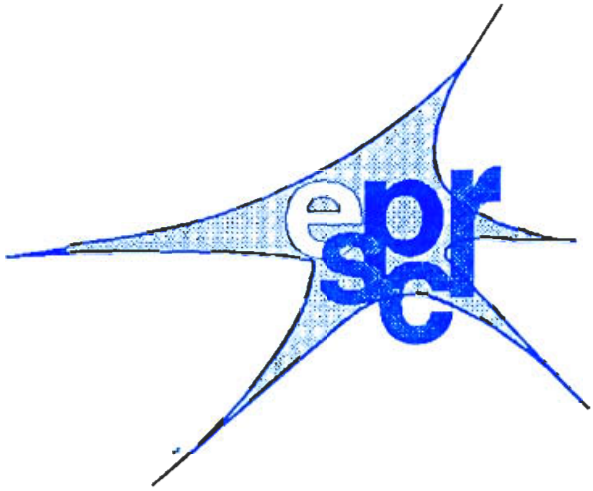

EUROPEAN SOCIETY FOR PIGMENT CELL RESEARCH *BULLETIN*

N° 38 - December 2000

Editorial Office: G. Ghanem (Editor), C. Meunier, R. Morandini (Production Team), Laboratory of Oncology and Experimental Surgery (L.O.C.E.), Université Libre de Bruxelles, Institut J. Bordet, Rue Héger-Bordet 1, B - 1000 Brussels, Belgium.
Phone: 32-2-541.32.96 Fax: 32-2-534.95.50 E-Mail: gghanem@ulb.ac.be

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HAPPY NEW YEAR 2001

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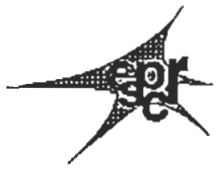

LETTER TO THE EDITOR
DISCUSSION, REVIEW,
SHORT COMMUNICATION, ...

A message from Prof. Dorothy BENNETT, the new ESPCR President

Dear Friends and Colleagues of the ESPCR;

It was very pleasant to see so many of you again at the recent Ulm meeting, although quite a surprise, at short notice, to be arriving there as the incoming president. Those of you who were not there may be interested to know that you also elected Prof. José-Carlos García-Borrón (Murcia, Spain) as Secretary, and Dr Lionel Larue (Orsay, France) as Treasurer.
Many thanks to everyone for your continuing support of the ESPCR. I think the three pigment cell societies of the IFPCS have a terrific amount to offer members. The "pigment cell community" is such a co-operative and friendly group. These have become my favourite meetings, where I have made many friends, learned much useful information, and set up many a valuable collaboration. Many of you have probably had similar experiences and we owe a great deal to the ESPCR's founders, in particular Prof. Giuseppe Prota, its first President from 1985.
We are also indebted to all those who have kept it going over the years. Warmest thanks and acknowledgements to the outgoing Officers, Professors Stan Pavel, Sheila Mac Neil and Ralf Peter, for all of their work over the past three years. They have done much in this period to improve the workings of the Society and make it more transparent and simple. All of them remain on the Council. Thanks too (once again) to Ralf Peter, Peter Kaskel and all of their team for setting up such a successful meeting at Ulm. This is also a rare opportunity to acknowledge and thank Professor Ghanem Ghanem, who so competently and successfully produces this Bulletin and manages our Web pages. When you look at those pages, I know Ghanem will be delighted to receive any comments you may have.
For the future, I hope you will all continue to support the ESPCR for many years (invitations to renew your subscription will be arriving shortly); and perhaps you will consider encouraging other colleagues in the field to join this Society, if they are not yet members. The more first-rate scientists (of all ages) who join in and attend the meetings, the more valuable it becomes for all of us. Our student subscription rate is excellent value at 25 EUR, and all younger members are eligible to apply for ESPCR travel grants. Prof Mauro Picardo has an exciting program lined up for you for September 2001 in Rome and, as you probably know, the ESPCR is host for the joint International Pigment Cell Congress in 2002, with Prof Pavel as principal organizer. It is also a great pleasure to announce here that our 2003 meeting will be organized by Prof Jean-Marie Naeyaert in Gent.
If there is anything you would like to know, any comments or suggestions you would like to make about the ESPCR, or any way in which you can offer to help, please do not hesitate to contact me, or any of the members of Council, whose names are in the Web pages. We will always value and welcome input from members.

Looking forward to seeing you in Rome
Dot Bennett

International Federation of Pigment Cell Societies

Officers: Shosuke Ito (JSPCR, *President*); Stan Pavel (ESPCR, *Vice-President*); Richard A. King (PASPCR, *Secretary/Treasurer*)

COUNCIL MEMBERS: Dorothy C. Bennett (ESPCR); Jose C. García-Borrón (ESPCR);; Sally Frost-Mason (PASPCR); Masako Mizoguchi (JSPCR); James J. Nordlund (PASPCR); Shigeki Shibahara (JSPCR); Vincent J. Hearing (*Ex Officio* member as the Editor of Pigment Cell Research) and Stan Pavel (*Ex Officio* member as Organizer of the 18th IPCC)

A Letter from the IFPCS President to the ESPCR members

At the end of the 20th century, I found this past year a remarkable one for pigment cell biologists. Scientists have made incredible advances in many disciplines of pigment cell biology, and those are now being disseminated to broader fields of biology and medicine. As the President of the IFPCS, I am glad to hear that the annual meetings of the ESPCR (in Ulm, Germany), the JSPCR (in Sapporo), and the PASPCR (in College Station, Texas) were excellent ones covering a broad range of topics in the pigment cell field. I wish to congratulate the Chairs of those meetings: Drs. Ralf U. Peter, Kowichi Jimbow, Lynn Lamoreux, and Estela Medrano for their successful meetings. In addition to the good news, however, we must recall sad news as well: the deaths of two prominent pigment cell scientists, Dr. Fritz Anders who died last December and Dr. Yoshiaki Hori who died last March. Dr. Anders will be remembered not only for his great contribution to the genetics of melanoma but also for the cheerful, yet successful 12th IPCC that was held in Giessen, 1983. Dr. Hori had been among the leaders of the pigment cell field in Japan for many years and served as the Vice-President of the IFPCS from 1996 to 1999. He will also be sorely missed by all who knew him.

The IFPCS has established the following goals for the Federation (also available on the **IFPCS Web page at http://www.cbc.umn.edu/ifpcs)**:

1. To encourage the dissemination of knowledge related to pigment cells by the establishment, sponsorship and support for the publication of books, bulletins, newsletter, journal, reports or other means.
2. To organize a tri-annual international meeting, to honor outstanding contributions in the field by awarding the Myron Gordon award at that meeting, and to select a scientist who has made recent and significant advances in the field to present the Seiji Memorial lecture.
3. To foster and enhance research on pigment cells and pigmentation among the regional Societies and to foster scientific collaboration, cooperation and communication among the regional Societies.

Goal #1 was achieved by establishing an official IFPCS-sponsored journal, ***Pigment Cell Research (http://www.pigment.org)***. The journal is now in the 13th year of publication. I wish to congratulate Dr. Vincent J. Hearing for his success in further raising the reputation of the journal in such a short time after succeeding as Editor at the beginning of this year from Dr. Jiro Matsumoto. To further promote the growth of the journal, it is essential that the numbers of subscribers and submitted papers be increased. I wish to urge all ESPCR members to subscribe to ***Pigment Cell Research,*** to make sure your Institution's library is subscribing, to submit papers to it, and to cite its pertinent references in your publications.

Goal #2 may be the most visible one among the several efforts that the IFPCS has been making; The ***International Pigment Cell Conference (IPCC)*** has been held every three years since 1946 when Dr. Myron Gordon held the first meeting in New York. Since the

inauguration of the IFPCS in Kobe in 1990, the IFPCS and one of the regional Societies have co-organized the IPCC on a rotating basis among the ESPCR, PASPCR, and JSPCR. The 15th IPCC was thus held in London in 1993, the 16th IPCC in Anaheim, California in 1996, and the 17th IPCC in Nagoya last year. I am happy to inform you that the venue of the 18th IPCC is a splendid, five star hotel in Scheveningen on the North Sea coast. The Chair of the next 18th IPCC, Dr. Stan Pavel, and his Organizing Committee, are working hard to welcome you to the Netherlands in September 8-13, 2002. The basic framework of the scientific program is now being planned, and will be finalized after consultation with the International Program Committee; you will receive the first announcement early next year. I wish to urge each of you to start planning to attend this exciting and stimulating Conference and to present your new findings.

Goal #3 is being achieved through related and important initiatives that the IFPCS has taken in the past several years. ***Special Interest Groups*** have been established and are providing substantial benefits to our scientific community, as shown on our Web page. We now have Special Interest Groups in the subdisciplines of **Biology of Melanoma, Pigment Cell Development, Genetics of Pigmentation, Hypo/Hyperpigmentation, Ocular/Extracutaneous Pigmentation,** and **Vitiligo**. The Federation Council has decided to continue these Interest Groups as a mechanism to promote pigment cell research. We expect that some of those Groups will hold their own Satellite symposia at the next IPCC, as they did at the Nagoya IPCC. The Pigment Cell Development group is also organizing an open workshop on April 4-6, 2001 at the NIH, Bethesda, USA. Further information will be available from Drs. Dorothy Bennett and Bill Pavan.

Another initiative to achieve Goal #3 was the establishment of the ***IFPCS Visiting Scientist Award***. The grants, established in 1997, are intended to support investigators from one of the regional Societies who wish to visit the laboratory of an investigator in another regional Society to learn specialized techniques and/or to establish inter-Society collaborations. You will find a full description of that program, the name of generous corporate donors, and the name of awardees on the IFPCS web page. The initial 3-year period of the program will end this year with 9 awardees being selected, but as the program has been quite successful, we hope to continue this program with a renewal of corporate donations.

I sincerely hope that we will see healthy and steady progress in our 3 regional Pigment Cell Societies, ESPCR, JSPCR, and PASPCR at the beginning of the new 21st century. In this respect, I wish to welcome new faces to the IFPCS Council; Drs. Dorothy Bennett (new President of the ESPCR) and JoseGarcía-Borrón (new Secretary of the ESPCR). Finally, I urge each of you to contribute to your Society in any way you can: submitting your abstracts to the regional Society meetings, publishing your papers in ***Pigment Cell Research***, collaborating with other members, and recruiting others scientists and clinicians to join us. Let me take this opportunity to wish each of you and your colleagues a wonderful and successful year 2001, the beginning of the 21st century.

Shosuke Ito
President, **IFPCS**

CURRENT LITERATURE

1. Melanins and other pigments chemistry

NOT AVAILABLE

2. Biology of pigment cells and pigmentary disorders

(Dr. M. Picardo)

Some authors have investigated apoptotic induction mechanisms both in normal and in melanoma cells. For to characterise the mechanism underlying melanocyte death in collagen, **Alanko A and Saksela O** have tested the effects of transforming growth factor β1 (TGF-β1), known to be functionally active in the skin. They found that melanocytes grown in three-dimensional type I collagen became exceptionally sensitive to endogenous or exogenous TGF-β, whereas both dermal nevus cells and melanoma cells were resistant to the apoptotic effect of TGF- β. The authors suggest that TGF-β play a central role in the skin controlling the growth and localization of cells of the melanocytes lineage. **Arita Y at al** have investigated whether apoptosis mediates effects of 12-O-tetradecanoylphorbol-13-acetate (TPA), that stimulate the growth of normal human melanocytes yet inhibit the growth of most melanoma cells. They observed that: 1) in melanocytes cultured with TPA few apoptotic cells and high levels of Bcl-2 were found; 2) removal of TPA induced an increase in the number of apoptotic cells; 3) addition of TPA inhibited strongly the growth in a metastatic cell line, but it did not induce apoptosis. These results show that TPA have a melanocyte growth effect by promoting anti-apoptotic mechanism associated with high levels of Bcl-2. **Vucic D and co-workers** reported the characterisation of ML-IAP, a novel human powerful inhibitor of apoptosis induced by death receptors and chemiotherapeutic agents. They found that the majority of melanoma cell lines expressed high levels of ML-IAP in contrast to primary melanocytes, which expressed undetectable levels. This elevated expression of ML-IAP renders melanoma cells resistant to apoptotic stimuli and thereby potentially contributes to the pathogenesis of this malignancy. **Gull Kin Y and co-workers** characterised apoptosis in UVB-damaged melanocytess by examing changes in the expression of p53 downstream molecules, Bcl-2 and BAX, following UVB irradiation. They found that UVB irradiation induced apoptosis and p53 up-regulation in melanocytes; also Bax was up-regulated and moved to the peri-nucler area, but Bcl-2 was not affected in terms of expression and distribution 24 hour after irradiation. The authors conclude that the increase and re-distribution of Bax is an early event that initiates the apoptotic response to UVB.

Several authors focused on melanogenesis regulation and melanocyte metabolism and phenotype. In the number of August 2000 of Pigment Cell Research **Prota G** reported a scientific profile of his studies on melanin and melanogenesis. **Kippenberger S et al** investigated the effects induced by mechanical stretch in melanocyte proliferation: mechanical strain generated in both keratinocytes and melanocytes an increased mitogen-activated protein kinase (MAPK) activities, but with different kinetics suggesting an activation of different signalling cascades. In human melanocytes **Sasaki M and co-workers** have evaluated tyrosinase gene expression induced by nitric oxide (NO), which has been reported to be involved in UV-induced melanogenesis. After NO treatment they observed up-regulation of tyrosinase mRNA expression, followed by an increase in dopa oxidase activity; immediately after stimulation with an NO donor the cGMP content in melanocytes rose; therefore the blocking of tyrosinase activity by a PKG inhibitor was observed. Taken together, these results indicate that the up-regulation of tyrosinase m-RNA via cGMP pathway may be a primary mechanism for NO-induced melanogenesis. **Horikoshi T et al** demonstrated the inhibitory effect of a topical applied NO synthase specific inhibitor on UVB-induced pigmentation in guinea pig skin; their results indicate that NO production may contribute to the regulation of UVB-induced pigmentation. For to examine the photoprotective role of melanocytes in low phototype caucasian skin, **Cario-André and co-workers** compared the effect of a single dose of UVB irradiation in epidermal reconstructs with and without melanocytes. Comparing the two reconstruct types no difference was detected in cyclobutane pyrimidine dimers (CPD) and 6-4 photoproducts (6-4PP), markers of DNA acute photodamage; more necrotic/apoptotic cells, however, were noted 24 h following UVB irradiation in reconstructs lacking melanocytes. These evidences suggest that even in fair-skinned individuals, melanocytes contribute to protect against short-term effects; in the long term, however, the cumulated DNA damage associated with the reduced apoptosis rate could increase the risk of skin cancer in such low phototype individuals. Melanosomal pH could be an important regulatory factor in control of melanogenesis in mammalian cells, as reported by **Ancans J and Thody AJ**, and by **Puri N et al. Seiberg M** et al showed that serine protease inhibitors, that interfere with protease-activated receptor 2 (PAR-2), induce depigmentation by affecting melanosome transfer and distribution. **AberdamE and co-workers** focused their attention on a neural endopeptidase, neprilysin, reporting that it is highly expressed by human melanocytes and

UVB light induce a down-regulation of its expression and catalytic activity. In addition they showed that alpha-MSH and ACTH were specific substrates for neprilysin and that specific inhibition of neprilysin increases the melanogenic activity of these peptides on human melanocytes. These data indicate that neprilysin inactivation by UVB in melanocytes may contribute to enhance the POMC paracrine loop mediating UV-induced pigmentation. In a European population representative sample, with Northen blot and immunocytochemical analyses, **Dwyer T et al** studied the association of arm naevi with melanin density at the upper inner arm and with melanin type in hair samples. Their findings suggest that human susceptibility to mutations of melanocytes can be estimated by objective biological measures.
Melanoma cell properties and melanoma phenotype and progression index was evaluated in several papers. **El Abdaimi K et al,** demonstrated higher parathyroid hormone-related peptide (PTHrP) in a human amelanotic melanoma cell line (A375) respect to normal human melanocytes; the treatment with a low calcemic dihydroxyvitamin D(3) analog (EB-1089), therefore, induced an inhibition of both cell proliferation and PTHrP expression. The authors indicate a stepwise increase in PTHrP expression when cell progress from normal to malignant phototype and suggest that EB-1089 should be further evaluated as a therapeutic agent in human melanoma. **Barral AM and co-worker** reported that thioredoxin (Trx) and Trx reductase (TrxR), as well as TNF-α, IL1-α and IL1-β, were highly expressed in cultured skin melanocytes and malignant melanoma cell lines; however, TNF-α showed a secretory block in these cells, suggesting a cytoprotective and a possible autocrine role or TNF-α. These data suggest that the resistance to TNF-α-induced cytotoxicity could be correlated with the intracellular expression of Trx and TrxR together with endogenous TNF-α. In melanoma cells, **Hedley SJ et al** showed that, when a T-cell proliferation stimulation was induced by cytokine pre-treatment, the addition of α-MSH reduced this response. These findings could confirm the hypothesis that α-MSH may assist melanoma cells to evade interaction with immune cells. To evaluate histidine decarboxylase (HDC) expression in human melanoma cell lines and primary tissues, **Haak-Frendscho M and co-workers** developed an anti-human HDC polyclonal antibody. They observed that the level of HDC expression was strongly associated with malignancy in skin. Thus, Haak-Frendscho suggested that HDC immunoreactivity may be useful as a clinical correlate for melanoma staging. **Boukerche H and co-workers** isolated a new monoclonal antibody specific for the membrane protein Mr 55,000 expressed by selected melanoma variants with increased metastatic properties. In vivo, this molecule may play a functionally important role in
determining metastasis and, thus, may represent a new metastatic risk marker in human melanoma **Selzer E et al** found expression of erythropoietin receptor (EpoR) in transformed melanocyte cell lines and in human melanoma cell lines, derived from melanoma metastases, but not in normal primary human melanocytes. These observations can support the hypothesis that EpoR may serve as a progression marker for human melanoma and it might be useful in the early diagnosis of melanoma. **Easty DJ and Bennett DC** reported that the high expression amount of protein tyrosine kinases both in benign and malignant melanocytes are likely to play a role in melanoma genesis and progression. Transfecting A-mel 3 hamster melanoma cells with sense- and antisense rhMIA cDNA and analysing subsequent chances in their tumorigenic and metastatic potential, **Guba M et al** provide direct evidence that melanoma inhibitory activity (MIA) play a role in metastasis of malignant melanomas.
Hypopigmentation mechanism and related disorders were examined by some authors. **Lehman AL and co-workers** demonstrated that the underwhite locus is a major determinant of mammalian pigmentation. Analysing in situ immune infiltrates in lesional, perilesional and non-lesional skin biopsies from patients with vitiligo, **van den Wijngaard R et al** demonstrated infiltrating T cells HLA-DR/CD8+ and focal expression of ICAM-1 and HLA-DR in the epidermis at the site of interaction between the immune infiltrates and the disappearing melanocytes. These findings support the hypothesis of a major role for skin-homing T cells in the death of melanocytes seen in vitiligo. **Desmond J and co-workers** re-examined the ultrastructure and immunohistochemistry of white/lesional and 'normal' pigmented skin of patients with vitiligo by focusing particularly on the presence/absence of functioning melanocytes and on the melanocyte-keratinocyte interaction. Even in depigmented skin from patients with old vitiligo hystory, they found rare melanocytes and melanin granules, but both in normal and in white/lesional epidermis an impairment of melanocytes-keratinocytes interaction was observed; a recovery of melanocyte functionality, therefore, was obtained after the removal of hydrogen peroxide. This study supports the concept that vitiligo involves the entire epidermal unit both in depigmented and normal pigmented skin. **van den Wijngaard R et al** investigated the posssible role of melanocyte-expressed apoptosis regulatory molecules in melanocytes disappearance. Their study indicate that the relative apoptosis susceptibility of melanocytes in vitiligo is comparable with that of normal control cells.

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3. MSH, MCH, other hormones, differentiation

(Dr. B. Loir)

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4. Photobiology

(Dr. E. Wenczl)

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5. Neuromelanins

(Dr. M. d'Ischia)

The origin, structure and biological roles of neuromelanin form the subject of various papers appeared in the last months of 2000, which provide interesting contributions toward an understanding of these longlasting issues. In a paper devoted to the structural characterization of human substantia nigra melanin Double et al. (J. Neurochem. 2000, 75, 2583-2589) demonstrate the occurrence in the pigment of a tightly bound proteinaceous component and confirm the existence of aromatic structural units purportedly derived from dopamine, but provide additional evidence against a substantial analogy between neuromelanin and dopamine melanin. The latter conclusion seems now founded on solid conceptual and experimental grounds and poses a serious caveat to the frequent use of dopamine melanin to model neuromelanin in in vitro studies. This would also apply in principle to the paper by Stepien et al. (Biochim. Biophys. Acta 2000, 1523, 189-195) showing that dopamine melanin protects against peroxynitrite induced tyrosine nitration, tryptophan oxidation and Ca-AtPase inactivation. Although the results would argue in favour of a protective role of neuromelanin against peroxynitrite toxicity, the actual relevance to the situation in human substantia nigra neuromelanin is opne to question. Further contributions to the elucidation of neuromelanin structure derive from the 13C NMR analysis of neuromelanin from parkinsonian patients by Aime et al. (Mov Disord 2000, 15, 977-981), which demonstrates the presence of a highly cross-linked protease-resistant lipophilic material by far exceeding the pigmented component, and similar in character to the melanoprotein obtained by enzymatic oxidation of dopamine in the pesence of albumin. The importance of a lipidic component in neuromelanin is also highlighted by Zecca et al. (J. Neurochem. 74, 1758-1765). Sulzer et al. (Proc. Natl. Acad. Sci. USA 2000, 97, 11869-11874) offer evidence for a possible inciting event in neuromelanogenesis, namely an excess of cytosolic catecholamines not accumulated into synaptic vesicles. The origin of neuromelanin from catecholamine oxidation and the

possible neurotoxic events underlying this process are addressed by Linert and Jameson (J. Inorg Chem. 2000, 79, 319-326) who emphaisze the critical role of iron, possibly in unprotected form, in the generation of cell damaging species. Finally, Braak et al. (Acta Neuropathol (Berl) 2000 99, 489-495) analyze the pathological changes that occur in the lower brainstem of patients with Parkinson's disease and suggest that deterioration of supramedullary limbic centres and bulbar brainstem nuclei reduces the limbic influence and affects control of premotor and motor neurones, thus contributing to the motor system dysfunction in Parkinson's disease.

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Neuromelanin biosynthesis is driven by excess cytosolic catecholamines not accumulated by synaptic vesicles. Proc Natl Acad Sci U S A 97(22):11869-74, 2000.

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6. Genetics, molecular and developmental biology

(Dr. F. Beermann)

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7. Tyrosinase, TRPs, other enzymes

(Prof. J.C. Garcia-Borron)

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8. Melanosomes

(Dr. J. Borovansky)

A rising tide of papers dealing with molecular mechanisms both of melanosome intracellular movement (*Vancoille et al, Wu & Hammer)* and their transport in keratinocytes (*Seiberg et al*) has continued. Molecular events critical for melanosome formation and for melanogenesis in them were characterized by *Jimbow et al* and *Ancans & Thody*, respectively.

- Ancans J, Thody AJ..
  Activation of melanogenesis by vacuolar type H^+-ATPase inhibitors in amelanotic, tyrosinase positive human and mouse melanoma cells. FEBS Letters 478(1-2): 57-60, 2000.
  Comments: The activation of melanogenesis by vacuolar type H^+-ATPase inhibitors (bafilomycin A_1, concanamycin A /folimycin/) in melanoma cells *in vitro* was documented. The findings suggest that the melanosomal pH could be an important factor in the control of melanogenesis in mammalian cells.

- Dell'Angelica EC, Mullin C, Caplan S, Bonifacino JS.
  Lysosome-related organelles. FASEB J 14(10): 1265-1278, 2000.
  Comments: An excellent review describing characteristics of lysosome-related organelles (melanosomes, lytic granules, MHC class II compartments, platelet-dense granules, basophil granules, neutrophil azurophil granules and Drosophila pigment granules) and their biogenesis. Close relationship between these organelles is demonstrated by their abnormalities in Chediak-Higashi and Hermansky-Pudlak syndromes.

- Jimbow K, Park JS, Kato F, Hirosaki K, Toyofuku K, Chua C, Yamashita T.
  Assembly, target-signalling and intracellular transport of tyrosinase gene family proteins in the initial stage of melanogenesis. Pigment Cell Res 13(4): 222-229, 2000.
  Comments: A critical review detailing the mechanisms involved in stage I melanosome biogenesis and discussing sorting signals and adapter proteins implicated in the trafficking of proteins into premelanosomes.

- Seiberg M, Paine C, Sharlow E, Andrade-Gordon P, Constanzo M, Eisinger M, Shapiro SS .
  The protease-activated receptor 2 regulates pigmentation via keratinocyte-, melanocyte interactions. Exp Cell Res 254: 25-32, 2000.

- Seiberg M, Paine C, Sharlow E, Andrade-Gordon P, Constanzo M, Eisinger M, Shapiro SS.
  Inhibition of melanosome transfer results in skin lightening. J Invest Dermatol 115: 162-167, 2000.
  Comments: Modulation of keratinocyte-melanocyte interaction via the protease-activated receptor 2 (PAR2), expressed on keratinocyte, affects melanosome transfer. The activation of PAR2 by SLIGRL (a

PAR2 specific peptide agonist) in vivo and melanin uptake by keratinocytes in vitro, whereas RWJ-50353 (a serine protease inhibitor), inhibited the PAR2 activation and induced depigmentation.

- Vancoillie G, Lambert J, Mulder A, Koerten HK, Mommaas AM, Van Ooostveldt, Naeyaert JM.
Cytoplasmic dynein colocalizes with melanosomes in normal human melanocytes. Brit J Dermatol 143: 298-306, 2000.
Comments: Using RT-PCR techniques and immunofluorescence and immunogold electron microscopic approach in normal human melanocytes the authors demonstrated a colocalization of dynein heavy chains 1 and 2 with melanosomes; dynein intermediate chain 74 was present on the melanosomal surface forming a link with □-tubulin.

- Wu X, Hammer III JA.
Making sense of melanosome dynamics in mouse melanocytes. Pigment Cell Res 13(4):241-247, 2000.
Comments: A review from series "Innovative Technology" focused on the techniques used in visualizing melanosome dynamics within wild type and *dilute* mouse melanocytes which helped to localize molecular motors (e.g. myosin Va).

9. Melanoma experimental, Cell culture

(Dr. N. Smit)

A. Melanoma cytotoxicity, experimental

- Albino AP, Juan G, Traganos F, Reinhart L, Connolly J, Rose DP et al.
Cell cycle arrest and apoptosis of melanoma cells by docosahexaenoic acid: association with decreased pRb phosphorylation. Cancer Res 60(15):4139-4145, 2000.
Comments: The fact that pRb became hypophosphorylated after exposure to DHA suggests a cross-talk mechanism between fatty acid metabolism and the pRb pathway. Determining the mechanism by which PUFAs can inhibit melanoma growth will be an important first step in the rational use of PUFAs as antitumor agents.

- Dutkiewicz R, Albert DM, Levin LA.
Effects of latanoprost on tyrosinase activity and mitotic index of cultured melanoma lines. Exp Eye Res 70(5):563-569, 2000.
Comments: The intraocular pressure-lowering drug latanoprost, a phenyl-substituted analogue of prostaglandin F2 alpha (PGF2 alpha), increases iris pigmentation in a small number of patients. Given that latanoprost induced tyrosinase activity, but did not increase the mitotic index in any of the human melanoma lines studied, this suggests that the in vivo iris pigmentation side effect of latanoprost may not result from increased cell division, but from elevated tyrosinase activity.

- Franchi J, Coutadeur MC, Marteau C, Mersel M, Kupferberg A..
Depigmenting effects of calcium D-pantetheine-S-sulfonate on human melanocytes. Pigment Cell Res 13(3):165-171, 2000.
Comments: When added to a culture medium, calcium D-pantetheine-S-sulfonate (PaSSO3Ca), at doses indicating no cytotoxicity, causes a visually recognizable, reversible loss of pigment in both normal adult melanocytes (HNM) and M4Be melanoma cells. It is likely that the compound exerts its depigmenting effects in human pigment cells through the modification of glycosylation of tyrosinase and TRP1.

- Friebe M, Mahmood A, Spies H, Berger R, Johannsen B, Mohammed A et al. '
3+1' mixed-ligand oxotechnetium(V) complexes with affinity for melanoma: synthesis and evaluation in vitro and in vivo. J Med Chem 43(14):2745-2752, 2000.

- Gembitsky DS, De Angelis PM, Reichelt KL, Elgjo K.
An endogenous melanocyte-inhibiting tripeptide pyroGlu-Phe-GlyNH2 delays in vivo growth of monoclonal experimental melanoma. Cell Prolif 33(2):91-99, 2000.
Comments: The melanocyte-inhibiting tripeptide (MTP) pyroGlu-Phe-GlyNH2 is present in tissue cultures of non-transformed melanocytes and melanoma cells and influences melanocyte growth in vitro. The results indicate that MTP temporarily delays in vivo tumour growth.

- La Porta CA.

nPKCdelta a new therapeutic marker for melanoma metastasis? (Review). Int J Mol Med 5(5):467-471, 2000.
Comments: The possible use of nPKCdelta as a therapeutic target is reviewed and discussed. Motivated by recent results, we propose a model in which nPKCdelta modulates melanin synthesis as well as metastasis.

- Lentini A, Autuori F, Mattioli P, Caraglia M, Abbruzzese A, Beninati S.
Evaluation of the efficacy of potential antineoplastic drugs on tumour metastasis by a computer-assisted image analysis. Eur J Cancer 36(12):1572-1577, 2000.
Comments: The main objective of this computerised procedure was to evaluate how the tumour cell is affected in the host by the drug under investigation. The use of the method is exemplified by an analysis of the antitumour activity of some methylxanthines.

- Maeshima Y, Colorado PC, Kalluri R.
Two RGD-independent alpha vbeta 3 integrin binding sites on tumstatin regulate distinct anti-tumor properties. J Biol Chem 275(31):23745-23750, 2000.

- Mitjans F, Meyer T, Fittschen C, Goodman S, Jonczyk A, Marshall JF et al.
In vivo therapy of malignant melanoma by means of antagonists of alphav integrins. Int J Cancer 87(5):716-723, 2000.

- Molinari A, Toccacieli L, Calcabrini A, Diociaiuti M, Cianfriglia M, Arancia G.
Induction of P-glycoprotein expression on the plasma membrane of human melanoma cells. Anticancer Res 20(4):2691-2696, 2000.
Comments: Flow cytometry, laser scanning confocal microscopy and cytotoxicity studies demonstrated that the activity of the drug extrusion system was related to both surface P-glycoprotein (P-gp) expression and resistance to doxorubicin. In conclusion, P-gp, but not multidrug resistance-related protein (MRP1), might play a pivotal role in the pharmacologically-induced MDR phenotype of melanoma cells.

- Raisova M, Bektas M, Wieder T, Daniel P, Eberle J, Orfanos CE et al.
Resistance to CD95/Fas-induced and ceramide-mediated apoptosis of human melanoma cells is caused by a defective mitochondrial cytochrome c release. FEBS Lett 473(1):27-32, 2000.
Comments: In the present study, five of 11 melanoma cell populations were shown to release cytochrome c from mitochondria, which activates caspase-3 and finally results in DNA fragmentation upon treatment with the agonistic monoclonal antibody CH-11. In contrast, this apoptotic pathway was not activated in the remaining six melanoma cell populations. Interestingly, the susceptibility of melanoma cells to CD95L/FasL-triggered cell death was clearly correlated with N-acetylsphingosine-mediated apoptosis. Our results are in line with a defect upstream of mitochondrial cytochrome c release in resistant cells.

- Redondo P, Bandres E, Solano T, Okroujnov I, Garcia-Foncillas J.
Vascular endothelial growth factor (VEGF) and melanoma. N-acetylcysteine downregulates VEGF production in vitro. Cytokine 12(4):374-378, 2000.
Comments: Vascular endothelial growth factor (VEGF), the most potent angiogenic factor identified to date, is associated with growth and metastasis of solid tumours, including melanoma. N-acetylcysteine inhibits VEGF production in three human melanoma cell lines. This antioxidant might have therapeutic applications in metastatic melanoma in combination with other cytotoxic drugs.

- Roberts JE, Wiechmann AF, Hu DN.
Melatonin receptors in human uveal melanocytes and melanoma cells. J Pineal Res 28(3):165-171, 2000.
Comments: In uveal melanoma cells, the expression of RNA encoding the Mel1b receptor suggests that the growth inhibiting effect of melatonin on uveal melanoma cells is related to activation of the melatonin Mel1b membrane receptor.

- Sengupta S, Ray S, Chattopadhyay N, Biswas N, Chatterjee A.
Effect of retinoic acid on integrin receptors of B16F10 melanoma cells. J Exp Clin Cancer Res 19(1):81-87, 2000.

- Smith CC, Yu YX, Kulka M, Aurelian L.
A novel human gene similar to the protein kinase (PK) coding domain of the large subunit of herpes simplex virus type 2 ribonucleotide reductase (ICP10) codes for a serine-threonine PK and is expressed in melanoma cells. J Biol Chem 275(33):25690-25699, 2000.

Comments: Endogenous H11 RNA and the H11 phosphoprotein are expressed in melanoma cell lines and primary melanoma tissues at levels higher than in normal melanocytes and in benign nevi. Melanoma cell proliferation is inhibited by treatment with antisense oligonucleotides that inhibit H11 translation, suggesting that H11 expression is associated with cell growth

- Stratton SP, Dorr RT, Alberts DS.
The state-of-the-art in chemoprevention of skin cancer. Eur J Cancer 36(10):1292-1297, 2000.
Comments: A transgenic murine melanoma model has been developed for evaluating potential agents in vivo. Agents at various stages of study include the green tea catechin epigallocatechin gallate (EGCG), the limonene derivative perillyl alcohol, the ornithine decarboxylase inhibitor alpha-difluoromethylornithine (DFMO), selenium, retinoids and salicylates. New chemopreventive agents that can be used to complement sunscreens may result in decreased incidence, morbidity and mortality of skin cancer

- Strickland FM, Muller HK, Stephens LC, Bucana CD, Donawho CK, Sun Y et al.
Induction of primary cutaneous melanomas in C3H mice by combined treatment with ultraviolet radiation, ethanol and aloe emodin. Photochem Photobiol 72(3):407-414, 2000.
Comments: 20-30% of the mice treated with a combination of UV radiation and ethanol vehicle and 50-67% of the UV-irradiated animals given aloe emodin in ethanol vehicle developed primary cutaneous melanin-containing tumors. The diagnosis of melanoma was established using Fontana silver stain for melanin. Our findings have led to the development of the **first facile murine model** for the induction of primary melanoma.

- Theron T, Binder A, Verheye-Dua F, Bohm L.
The role of G2-block abrogation, DNA double-strand break repair and apoptosis in the radiosensitization of melanoma and squamous cell carcinoma cell lines by pentoxifylline. Int J Radiat Biol 76(9):1197-1208, 2000.
Comments: These data suggest that radiosensitization by pentoxifylline is not a consequence of G2-block abrogation alone, but that inhibition of DSB repair plays a role in certain cell types.

- Zhou R, Bansal N, Leeper DB, Glickson JD.
Intracellular acidification of human melanoma xenografts by the respiratory inhibitor m-iodobenzylguanidine plus hyperglycemia: a 31P magnetic resonance spectroscopy study. Cancer Res 60(13):3532-3536, 2000.
Comments: The synergetic effects of MIBG and hyperglycemia result in significant acidification of the tumor and a decrease in tumor bioenergetic status, and the effects are largely selective for tumors in comparison with normal tissues.

B. Melanoma gene and immunotherapy

- Clark PR, Stopeck AT, Ferrari M, Parker SE, Hersh EM.
Studies of direct intratumoral gene transfer using cationic lipid-complexed plasmid DNA. Cancer Gene Ther 7(6):853-860, 2000.
Comments: Animals received i.t. injections of VR1103, a DNA plasmid encoding the gene for human interleukin-2 (IL-2), either alone or complexed with the cationic lipid N-(1-(2,3-dimyristyloxypropyl)-N,N-dimethyl-(2-hydroxyethyl) ammonium bromide/dioleoyl phosphatidylethanolamine (DMRIE/DOPE). The results indicate that the formulation and dosage of cationic L:D complexes, but not injection technique, play a key role in determining the level of i.t. transgene expression.

- Perricone MA, Claussen KA, Smith KA, Kaplan JM, Piraino S, Shankara S et al.
Immunogene therapy for murine melanoma using recombinant adenoviral vectors expressing melanoma-associated antigens. Mol Ther 1(3):275-284, 2000.

- Shiose S, Sakamoto T, Yoshikawa H, Hata Y, Kawano Y, Ishibashi T et al.
Gene transfer of a soluble receptor of VEGF inhibits the growth of experimental eyelid malignant melanoma. Invest Ophthalmol Vis Sci 2000; 41(9):2395-2403, 2000.
Conclusions: Adenovirus-mediated gene transfer of a soluble form of VEGF receptor (flt-1) gene inhibited the growth of the experimental eyelid malignant melanoma. This method may be useful as an antiangiogenic therapy for eyelid malignant melanoma.

- Slingluff CL, Jr., Colella TA, Thompson L, Graham DD, Skipper JC, Caldwell J et al.

Melanomas with concordant loss of multiple melanocytic differentiation proteins: immune escape that may be overcome by targeting unique or undefined antigens. Cancer Immunol Immunother 48(12):661-672, 2000.

- Villa R, Folini M, Lualdi S, Veronese S, Daidone MG, Zaffaroni N.
Inhibition of telomerase activity by a cell-penetrating peptide nucleic acid construct in human melanoma cells. FEBS Lett 473(2):241-248, 2000.

C. Culture systems to study melanocytes

- Alanko T, Saksela O.
Transforming growth factor beta1 induces apoptosis in normal melanocytes but not in nevus cells grown in type I collagen gel. J Invest Dermatol 115(2):286-291, 2000.

- Catala M, Ziller C, Lapointe F, Le Douarin NM.
The developmental potentials of the caudalmost part of the neural crest are restricted to melanocytes and glia [In Process Citation]. Mech Dev 95(1-2):77-87, 2000.
Comments: we have analyzed the developmental potentials of neural crest cells arising from the caudalmost part of the neural tube in avian embryo in in vitro culture and by means of heterotopic transplantations in vivo. We show here that neural crest cells arising from the neural tube located at the level of somites 47-53 can differentiate both in vitro and in vivo into melanocytes and Schwann cells but not into neurons.

- Chen YF, Chang JS, Yang PY, Hung CM, Huang MH, Hu DN.
Transplant of cultured autologous pure melanocytes after laser-abrasion for the treatment of segmental vitiligo. J Dermatol 27(7):434-439, 2000.

- Dekker SK, van Doorn R, Kempenaar J, Gruis NA, Vermeer BJ, Ponec M.
Skin equivalent: an attractive model to evaluate early melanoma metastasis. Melanoma Res 10(2):127-140, 2000.
Comments: In contrast to the submerged co-cultures, all four cell lines formed sharply demarcated tumour cell nests within the epidermal compartment of the skin equivalent model, with the morphology highly mimicking the in vivo situation. The air-exposed skin equivalent model was shown to be suitable for studying differences in growth patterns and potential invasive behaviour.

- Dupin E, Glavieux C, Vaigot P, Le Douarin NM.
Endothelin 3 induces the reversion of melanocytes to glia through a neural crest-derived glial-melanocytic progenitor. Proc Natl Acad Sci U S A 97(14):7882-7887, 2000.

- Graeven U, Rodeck U, Karpinski S, Jost M, Andre N, Schmiegel W.
Expression patterns of placenta growth factor in human melanocytic cell lines. J Invest Dermatol 115(1):118-123, 2000.

- Hsu M, Andl T, Li G, Meinkoth JL, Herlyn M.
Cadherin repertoire determines partner-specific gap junctional communication during melanoma progression. J Cell Sci 113(Pt 9):1535-1542, 2000.
Comments: We examined gap junctional capability of melanocytic cells from various stages of tumor progression in coculture models using dye transfer assays. Normal melanocytes coupled with keratinocytes by gap junctional formation, whereas melanoma cells did not. Our data suggest that (1) melanocyte transformation is associated with loss of the pre-existing gap junctional activity with keratinocytes but a concomitant gain of communication with a newly juxtaposed cell type, the fibroblasts, (2) the specificity of gap junctional formation during melanoma development is determined by the cadherin profile on the melanocytic cells.

- Hsu MY, Meier FE, Nesbit M, Hsu JY, Van Belle P, Elder DE et al.
E-cadherin expression in melanoma cells restores keratinocyte-mediated growth control and down-regulates expression of invasion-related adhesion receptors. Am J Pathol 156(5):1515-1525, 2000.
Comments: In a skin reconstruction model, ectopic E-cadherin expression inhibits invasion of melanoma cells into dermis by down-regulating invasion-related adhesion receptors, MelCAM/MUC18 and beta3 integrin subunit, and by induction of apoptosis.

- Iyengar B.
Melatonin and melanocyte functions. Biol Signals Recept 9(5):260-266, 2000.
Comments: Melanocyte melatonin positivity increases with dark incubation and is higher with a pulse of UV exposure after dark incubation with melatonin. This increase is associated with a doubling of melanocyte number after dark incubation and a further doubling upon exposure to a pulse of UV.

- Jordan SA, Jackson IJ.
MGF (KIT ligand) is a chemokinetic factor for melanoblast migration into hair follicles. Dev Biol 225(2):424-436, 2000.

- Kim NS, Cho JH, Kang WH.
Behavioral differences between donor site-matched adult and neonatal melanocytes in culture. Arch Dermatol Res 292(5):233-239, 2000.

- Le Poole IC, Boissy RE, Sarangarajan R, Chen J, Forristal JJ, Sheth P et al.
PIG3V, an immortalized human vitiligo melanocyte cell line, expresses dilated endoplasmic reticulum [In Process Citation]. In Vitro Cell Dev Biol Anim 36(5):309-319, 2000.
Comments: Vitiligo melanocytes Ma9308P4 were transfected with HPV16 E6 and E7 genes using the retroviral construct LXSN16E6E7. Successful transformants were selected using geneticin and subsequently cloned to ensure genetic homogeneity. The resulting cell line PIG3V has undergone more than 100 cell population doublings since its establishment as a confluent primary culture, whereas untransfected melanocytes derived from adult skin senesce after a maximum of 50 population doublings.

- Meije CB, Mooi WJ, Le Poole IC, Van Muijen GN, Das PK.
Micro-anatomy related antigen expression in melanocytic lesions. J Pathol 190(5):572-578, 2000.

- Nataf V, Le Douarin NM.
Induction of melanogenesis by tetradecanoylphorbol-13 acetate and endothelin 3 in embryonic avian peripheral nerve cultures. Pigment Cell Res 13(3):172-178, 2000.

- Sarangarajan R, Zhao Y, Babcock G, Cornelius J, Lamoreux ML, Boissy RE.
Mutant alleles at the brown locus encoding tyrosinase-related protein-1 (TRP-1) affect proliferation of mouse melanocytes in culture [In Process Citation]. Pigment Cell Res 13(5):337-344, 2000.

- Zuasti A, Martinez-Liarte JH, Solano F, Ferrer C.
Melanization stimulating factors in the integument of the Mugil cephalus and Dicertranchus labrax. Histol Histopathol 15(4):1145-1150, 2000.
Comments: Media conditioned by exposure to dorsal and/or ventral skin, stimulates the melanization of Xenopus laevis neural crest cells throughout a 3 day assay period. Similarly conditioned culture media tested on B16-F10 murine malignant melanocytes, revealed a considerable influence in enzymatic activities: dopachrome tautomerase (DCT), tyrosine hydroxylase and dopa oxidase.

ANNOUNCEMENTS & RELATED ACTIVITIES

Calendar of events

Also available in more details from address: http://www.ulb.ac.be/medecine/loce/espcr.htm

2001 5th World Conference on Melanoma
Venice, Italy, February 28 - March 3

Contact: Dr. Mario SANTINAMI
Secretary General
5th World Conference on Melanoma
Casa di Cura S. Pio X
Via F. Nava 31
I - 20159 Milano
Phone/Fax: 39-02-69516449
E-Mail: info@melanoma2001.org
Website: www.melanoma2001.org

2001 VIth International Conference on Solar Energy and Applied Photochemistry Cairo, Egypt, April 3-8

Contact: Prof. M.S.A. ABDEL-MOTTALEB
Photoenergy Center
Faculty of Science, Ain Shams Unversity
Abbassia, Cairo, Egypt
Phone: 002012 216 9584 (cellular)
Faxes: 00202 244 7683 or 00202 484 5941
E-mail: solar@phoenergy.org
solar@link.com.eg
Website: http://www.photoenergy.org/solar2001.html

2001 International Workshop on Molecular Mechanisms of Tanning
Nice, France, April 27-29

Contact: Prof. J.P. ORTONNE, Dr. R. BALLOTTI
IWMMT Congress Office – Maryse Clappier
Hôpital l'Archet 2 – Service de Dermatologie
BP 3079
F- 06202 Nice Cedex 3
Phone: 33 (0)4 92 03 61 19
Fax: 33 (0)4 92 03 65 32
E-mail: maryse.clappier@unice.fr

2001 11th International Conference on Second Messengers and Phosphoproteins, Melbourne, Australia, April

Contact: Gayle McMurray
Administrative Officer
St Vincent's Institute of Medical Research
Phone: 9288-2480
Fax: 9416-2676

E-mail: g.mcmurray@medicine.unimelb.edu.au
Web page: http://www.secondmessengers.com

2001 Xth PASPCR Meeting
Minneapolis, MN, June 14-17
Contact: Dr. Richard KING
University of Minnesota
Depts. of Medecine and Pediatrics
Box 485 UMHC
420 Delaware Street
USA - Minneapolis, MN 55455
E-Mail: king@mail.ahc.umn.edu

2001 Xth Meeting of the ESPCR
Rome, Italy
Contact: Dr. Mauro PICARDO
Istituto Dermatologica San Gallicano
Via San Gallicano 25/A
I – 00153 ROMA
E-mail: picardo@crs.ifo.it

2001 15th Japanese Society for Pigment Cell Research Meeting (JSPCR)
Sendai, Japan, December 1-2
Contact: Prof. S. SHIBAHARA
E-mail: shibahar@mail.cc.tohoku.ac.jp

2002 XVIIIth International Pigment Cell Conference : The Hague, The Netherlands
Contact: Dr. Stan PAVEL
University Hospital Leiden
Dept of Dermatology
PO Box 9600
NL - 2300 RC LEIDEN
Tel: 31-(71) 526 1952
Fax: 31-(71) 524 8106
E-mail: SPavel@algemeen.azl.nl

2003 XIth Annual Meeting of the PanAmerican Society for Pigment Cell Research
September 3-7, Wood's Hole, MA
Contact: Dr. Jean BOLOGNIA
E-mail: jeanbolognia@gm.yale.edu

2003 XIth Meeting of the ESPCR: Gent, Belgium
Contact: Prof. J.M. NAEYAERT

2004 XIIth Meeting of the ESPCR: Paris, France
Contact: Dr. Lionel LARUE

2005 XIVth International Pigment Cell Conference : Bethesda, USA
Contact: Dr. V. HEARING

New Members

The ESPCR is delighted to welcome the following colleagues to membership and hope that they will play a full and active part in the Society.

Dr. R. BUSCA
Dept. of Biology and Physiopathologie
Faculty of Medicine
INSERM U385
Avenue de Valombrose
06107 Nice, France

Dr. Rosa CICERO
Dipartimento di Biochimica Medica e Biologia Medica
Facoltà di Medicina e Chirurgia
Università degli Studi
Policlinico
Piazza Giulio Cesare
70124 Bari, Italy

Dr. S. COMMO
L'Oréal
Avenue Eugène Schueller 1
92117 Clichy, France

Dr. E. HEALY
University of Southampton
Dept Dermatopharmacology
Mail point 825, Level F, South Block
Southampton General Hospital
Tremona Road
UK - SOUTHAMPTON SO16 6YD

Dr. M. HOOGDUIJN
Dept. of Biomedical Sciences
University of Bradford
Richmond Road
BD71DP Bradford, UK

Dr. C. JIMENEZ-CERVANTES
Dept. of Biochemistry and Molecular Biology
School of Medicine
University of Murcia
Apto 4021, Campus de Espinardo
30100 Espinardo, Spain

Dr. J. LAMBERT
Dept. of Dermatology
University Hospital
De Pintelaan 185
9000 Gent, Belgium

Dr. L. MONTOLIU
Departamento de Biologia Molecular y Celular
Centra Nacional de Biotecnologia
Campus de Cantoblanco (CNB-CSIC)
28049 Madrid, Spain

Dr. R. PANDHI
Institute Med. Edu.
Dermatology
Sector -12
IND - 160012 CHANIGARH

Dr. S. PRINCE
Marie Curie Research Institute
Eukaryotic Transcription Unit
The Chart
Oxted Surrey RH8 0TL, UK

Dr. F. SHAMSI
Dept. of Optometry and Visual Science
Cardiff University
Redwood Building
PO Box 905 - King Edward VII Avenue
Cardiff, CF103YJ
Wales, UK

Dr. J. SIMON J.
Duke University
Department of Chemistry
USA - DURHAM, NC 27708

Dr D.J. TOBIN
University Bradford
Richmond Road
UK - Bradford BD7 1DP

Dr. F. VAN NIEUWPOORT
Dept. of Dermatology
Leiden University
Wassenaarseweg 12
2323 Al Leiden
The Netherlands

Dr. L.J. WAINWRIGHT
Unilever Research
Skin Hait Biology
Colworth Lab.
UK - BEDFORD MK44 1LQ

ESPCR financial report 9/1999- 9/2000

1. Number of members 9/2000:	203
new members in 1999:	13
new members in 2000:	8
cancelled members in all:	55
cancelled members 1999:	8
cancelled members in 2000:	10
members paid the annual subscription 1999:	132
members paid the annual subscription up to 9/2000:	107
2. Costs 2000 –10/2000	
international society of pigment cell research	
bulletin and web costs (Prof. Ghanem)	1784,22 DM
bank charges	74,52 DM
travel costs and office expenses	10692,38 DM
(travel awards, ESPCR future executive meeting, mail)	
office expense	562,92 DM
together	13114,04 DM
Costs 1999 –1/2000	
international society of pigment cell research	4181,29 DM
bulletin and web costs (Prof. Ghanem)	1413,00 DM
travel stipends (paid on 26.10.1999)	2000,00 DM
bank charges	186,47 DM
office expense (Prof. R. U. Peter)	291,00 DM
reimbursement Prof. Dr. R.U. Peter	5000,00 DM
(ESPCR 2000, Ulm):	
Bank charges:	24,96 DM
together:	13096,72 DM
3. Subscriptions	
subscription fees paid 1999	10490,20 DM
subscriptions fees paid 2000	12963,58 DM
4. Balances of account	
31.12.1998	23.785,26 DM
30.12.1999	21.178,74 DM
29.08. 2000	26.691,20 DM

Prof. R.U. Peter
Treasurer

INTERNATIONAL WORKSHOP ON MOLECULAR MECHANISMS OF TANNING
Plaza Concorde Hotel - 12, avenue de Verdun - Nice - France
April 26 – 29, 2001

Organisers: *Prof. J.P. Ortonne – Dr R. Ballotti (Service de Dermatologie CHU Nice ; Inserm U 385)*

This workshop will gather specialists to discuss of aspects of the biological effects of ultraviolet radiation on normal skin with special emphasis on photo-induced melanogenesis.

Topics: The melanocyte system ; Transcriptional control of melanogenesis ; Role of the camp and PKC ; signalling pathways in melanocyte differenciation ; Melanosome biogenesis and transport ; Effects and ; signalling of **UV** ; **UV** and melanogenesis ; Photoprotection and future strategies to modulate melanogenesis and melanin photoprotection.

Invited speakers : : Heinz **ARNHEITER** (USA) ; Philippe **BAHADORAN** (FRANCE) ; Corinne **BERTOLOTTO** (FRANCE) ; Roser **BUSCA** (FRANCE) ; Benoît **DERIJARD** (FRANCE) ; Mark **ELLER** (USA) ; David **FISHER** (USA) ; Gary **FISHER** (USA) ; Colins R. **GODING** (UK) ; John **HAMMER** (USA) ; Vincent J. **HEARING** (USA) ; Meenhard **HERLYN** (USA) ; Nancy **JENKINS** (USA) ; Jean **KRUTMANN** (GERMANY) ; Nicole **LE DOUARIN** (FRANCE) ; Thomas **LUGER** (GERMANY) ; James **NORDLUND** (USA) ; Hee-Young **PARK** (USA) ; William J. **PAVAN** (USA) ; Giuseppe **PROTA** (ITALY) ; Johnatan **REES** (UK) ; Alain **SARASIN** (FRANCE) ; Rainer **SCHMIDT** (FRANCE) ; Thomas **SCHWARZ** (GERMANY) ; Miri **SEIBERG** (USA) ; Shigeki **SHIBAHARA** (JAPAN) ; Richard **SPRITZ** (USA) ; Anthony **THODY** (UK).

The number of participants is limited to 150. The workshop will start on Friday 27 april 2001 at 8.00am and will finish on Sunday 29 April at noon. Information for registration and abstract submission (for poster presentation only) can be obtained from the congress office :

IWMMT Congress Office
Maryse Clappier
Hôpital l'Archet 2
Service de dermatologie
BP 3079
06202 Nice cedex 3
Tel: 33 (0)4 92 03 61 19
Fax: 33 (0)4 92 03 65 32
E-mail : maryse.clappier@unice.fr

Download information, scientific programme, registration and accommodation forms from:
http://www.ulb.ac.be/medecine/loce/espcr/announce.htm