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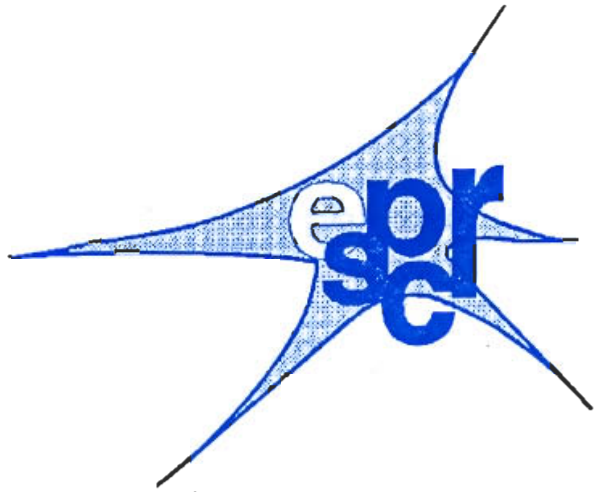

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HAPPY NEW YEAR 1998

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MEETING REPORT

ESPCR 7th meeting
Bordeaux, 9 - 11 October 1997
by Dr. Sheila Mac Neil

This report has been compiled with input from several colleagues who kindly shared their highlights from this meeting - Dorothy Bennett, David Gawkrodger, Marco d'Ischia, Mauro Picardo and Tony Thody.

For those of you who may not have attended a European Pigment Cell Research meeting before, there are perhaps a few facts I should tell you - you will have a good time socially (and the food and drink this year were chosen with particular care - Tony Thody wishes congratulations to be passed on to Jean-Etienne Surleve-Bazeille and Alain Taieb on the excellence of both) - however, you will also need stamina and to keep your wits about you if you are to do justice to a 2½ day meeting which typically includes presentations from 6 guest lecturers, 55 oral communications and 72 posters on a range of topics which span the biochemistry of the melanins, developmental and cellular biology of the melanocytes, their genetics through to their behaviour in vitiligo and melanoma. There are no simultaneous sessions at these meetings and it is tough being a member of the audience at a European Society for Pigment Cell Research meeting.

So, is it worth listening to talks in areas outside of your own field or speciality? The answer is a clear 'yes'. If you are willing to listen and learn, the chances of setting up collaborations to extend and broaden your research are excellent. Many scientific friendships have been forged at these meetings and continue to flourish. A lot of the presentations at the meeting were visible fruit of such collaborations. Not that I am biased, of course, but as the new Secretary for the ESPCR, I would say that this meeting (more than any other that I know of) has just the right chemistry (Professor Prota) to set up enjoyable collaborations - the meetings are the right size - they contain a number of disciplines all focussed on the melanocyte and you can be pretty sure of meeting up with your colleagues on an annual basis. If you have never attended one of these meetings before, don't be put off - we are a very easy society to join, very friendly, newcomers are made very welcome and guest lecturers are chosen with infinite care to provide topical overviews of particular areas of melanocyte biology. That was certainly the case at this meeting where the quality of the guest lectures was excellent throughout. So, enough advertising - here are some of the highlights from this meeting.

GUEST LECTURES

Particularly commended by many colleagues was the guest lecture by Jonathan Rees on genetic approaches to melanoma susceptibility in which he reviewed recent work from Newcastle and other Centres on mutations affecting the melanocortin-1 receptor gene. There are now known to be a considerable number of mutations of this receptor and Professor Rees group is leading the research into looking at the possible relationships between gene variants and inheritance of red hair and between gene variants and the likelihood of developing melanoma. This work is likely to open new vistas not only on the origin of skin tumours but especially on the complex mechanisms affecting skin type and hair colour - these studies favour a gradual shift of interest from the eumelanin to the phaeomelanin pathway as the key to the understanding of melanoma susceptibility - it will be interesting to see how these results will be integrated into the current knowledge of the biochemistry of phaeomelanin pigmentation - Marco d'Ischia.

Other excellent guest lectures were from Seth Orlow on the comparative genetics of oculo-cutaneous

pigmentation (detailing the different stages at which differences in genetics could have an effect on pigmentation encompassing melanocyte migration, melanosome biogenesis and melanosome trafficking and transfer) and from Barbara Gilchrest who addressed the issue of how melanocytes in vivo are undoubtedly under the control of factors produced by adjacent cells which will themselves change in response to UV. Professor Gualde also reviewed the very wide array of approaches to inducing immunogenicity in melanoma that are currently under investigation in a very clear presentation.

Another excellent guest lecture was from Nicole Le Douarin on factors controlling the development of the melanocytic lineage from the neural crest. This was closely followed by a presentation from Laure Lecoins from Professor Le Douarin's laboratory on a novel sequence for an avian endothelin receptor which may help in understanding the evolution of function of these receptors (Dorothy Bennett).

AND NOW TO SOME OF OUR FAVOURITE THINGS ...

With respect to interactions between α-MSH and cytokines, things are looking interesting - on one hand, we learned that inhibition of melanogenesis by TNF-α is mediated through a downregulation of the tyrosinase promoter activity (Englaro et al.) - on the other hand, we learned that α-MSH itself can oppose the action of TNF-α in melanocytes (Hedley & Mac Neil) and melanoma cells (Morandini & Ghanem). The implications of α-MSH opposing the response of melanocytes and melanoma cells to cytokines are potentially quite exciting - time and further work will tell.

α-MSH was also found to increase production of nitric oxide in melanocytes and to potentiate UV-induced nitric oxide production (Alison Graham and Tony Thody) - whether this will relate to any melanogenic actions of α-MSH or other actions of this busy little hormone, time will tell.

How the agouti protein fits into things is slowly emerging. Vincent Hearing showed that agouti protein is capable of upregulating some and downregulating other genes. These genes are likely to be important in control of eumelanin and phaeomelanin synthesis and determining hair colour and may provide further key information on the biochemical pathways responsible - possibly some important missing pieces of our jigsaw - Dorothy Bennett. Dorothy also singled out a talk from Fritz Anders on a new class of oncodeterminants as the most important talk of the meeting - Dorothy explains - that the Xiphorus fish work produced the first oncogene and the first tumour suppressor gene and mammals followed fish on both these occasions. In his talk, Dr. Anders presented evidence of retrotransposons as heritable and amplifiable tumour-suppressor-suppressors. He also pointed out that all of these sequences are found in telomeres and one repeat sequence that he gave in detail was crammed full with methylation sites (of CG). Methylated DNA goes heterochromatic and can silence neighbouring genes by spread of this state - apparently a well known phenomenon. Once again, these little fish in Dr. Anders capable hands may be pointing the way .....

I am indebted to Marco d'Ischia for highlights of the session on the biophysics and biochemistry of melanins. The effect of thiol compounds on melanogenesis was the central theme of two contributions by Smit, Pavel et al. and Potterf Benathan, Hearing and co-workers. While the first paper focussed on the effects of varying concentrations of tyrosine and cysteine on melanogenesis highlighting a role of melanin production on glutathione depletion in melanocytes cultured with high tyrosine and low cysteine concentrations, the second paper addressed the relationship between agouti signal protein, cysteine transport and uptake in melanosomes and cysteinyl dopa formation to reinforce the notion that it is cysteine and not glutathione that is the actual ultimate precursor of cysteinyldopas. In another partially related paper, Dr. Benathan went on to show that tyrosinase could play a protective role against UV irradiation by catalysing the formation of cysteinyldopa conjugates.

Dr. Mars and Professor Larsson used a microautoradiographic technique to conclude that persons with a high content of phaeomelanin in their skin have toxicological risks of melanin-related adverse effects compared to those of dark skinned people.

Similarities and differences in the process of pigment formation between cutaneous melanocytes and melanogenesis in the ink gland of Sepia Officinalis were presented by Dr. Palumbo. Analysis of the

nature of melanins is improving - Wakamatsu Ito and co-workers have used an improved version of a spectrophotometric method for melanin analysis combined with gel filtration HPLC to evaluate the DHICA content of eumelanins and their molecular size.

Also, in what must be the biggest ever group of related posters - 4 related poster presentations by Riley, Land, Pavel, Smit and co-workers on the mechanism of tyrosinase activation and the synthesis and properties of novel indoliumolate derivatives - these workers concluded that the actual product of the action of tyrosinase on tyrosine is dopaquinone and not dopa which is formed only by a reduction of dopaquinone by leucodopachrome. Other significant contributions dealt with the structural modifications of synthetic eumelanins under aerobic conditions by Prota, d'Ischia and co-workers, the characterisation of melanins from tetrahydroisoquinolines by Rosei, Mosca and colleagues and a MALDI/MS study of oligomers of 5-hydroxytryptimine by Allegri, Traldi et al.

Putting the melanocyte back where it belongs ...

A number of groups are now looking at the behaviour of the melanocyte in in vitro models of reconstructed skin. Clearly leading this field is work from Professor Taieb's laboratory in which a number of basic and fundamental parameters about the behaviour of melanocytes from different skin types and from vitiligo skin have been established. These models are obviously going to prove very useful for studies of pigmentation research and may yield a few surprises - a poster by Hedley et al. showed that fibroblasts added to such an epidermal/dermal reconstituted skin tended to reduce spontaneous pigmentation of these composites. To date, the majority of composites have examined melanocyte/keratinocyte interaction in either an artificial collagen matrix or on acellular human dermis. Such composites are now also being used to investigate invasion of melanoma cells and should be a particularly useful tool for studying melanoma cells/ECM interactions (Bizik et al.).

Do melanocytes differ from melanoma cells in their ability to cope with oxidative stress?

Further evidence supporting this hypothesis was presented in a poster by Chau, Meyskens and Buckmeier showing essentially that melanoma cells cope less well than melanocytes with reactive oxygen species suggesting impairment of redox regulation in melanoma. Following on from this, this same group (Meyskens, Buckmeier and Tohidian) showed that the transcription factor NF'B which is activated by a variety of stimuli including inflammatory cytokines and reactive oxygen species appear to be differently regulated in melanoma cells compared to melanocytes. Differences in expression of rel family members between normal human melanocytes and metastatic melanoma were noted which may go some way to explain the differing abilities of the cells to cope with reactive oxygen species.

POSTER AWARDS

A first prize of 2,000FF was awarded to N.P.M. Smit, S. Pavel, C.J. Kooksey, C.A. Ramsden, P.A. Riley for a poster entitled, "The mechanism of tyrosinase auto-activation. Part IV: Methylation of indoluminolates by COMT". (This was the fourth part of a blockbuster poster presentation which represented combined work from scientists in Leiden, London, Manchester and Keel. The breadth of the work accomplished in these four posters was very evident).

Two joint second prizes of 1500FF were awarded to Y. Gauthier, F. Nagy, M.F. Harmand, A. Taieb for a poster entitled, "Transplantation of cultured melanocytes in piebaldism" and to S.A. Gordan, M.A.F. MacKenzie, P.S. Budd and I.J. Jackson for a poster entitled, "Melanoblast proliferation in W,PhRw mutants".

HONORARY ESPCR MEMBERSHIP

The Society took great pleasure in making two Honorary Lifetime (ESPCR) Membership Awards to **Professor Hans Rorsman** and to **Professor Fritz Anders**. The awards were made by Professor Patrick Riley with the unanimous approval of the ESPCR Council members - judging by the applause on the evening, it was clear that Professors Rorsman and Anders have earned the respect and affection of many many people.

In conclusion, apologies to everyone's work that I haven't mentioned - I hope I have given you a flavour of the meeting and to persuade you that you really cannot afford to miss the next one in Prague in September 1998.

1. Melanins and other pigments chemistry

(Prof. M. Peter)

General: A review on pigment cell research containg aspects of structure and biosynthesis, emphasizing the role of DHI and DHICA in protection of epidermal tissues against toxic oxygen radical species as well as in inflammatory and immune reactions and the role of pheomelanin the abnormal susceptibility of red heads to sun damage and skin cancer has appeared (Prota).

Structure and Analysis: Two papers of the Naples group dealt with the structure of *Sepia* melanin which contains a high proportion of degraded 5,6-dihydroxyindole-2-carboxylic acid units (Pezzella et al.), as was also shown by mass spectrometry (Pezzella et al.). Truffle melanin was characterized as an allomelanin by pyrolysis/GC-MS and by solution and solid-state NMR (De Angelis et al.). Another paper decribes melanin from *Tuber melanosporum* (Harki et al.). Spectroscopic studies of melanin were reported in a meeting of the Am. Chem. Soc. (Forest and Simon). X-ray microanalysis was used to determine melanin types in human skin melanosomes (Koerten et al.). The total content of melanins and the proportion of pheomelanin was analyzed in hair of Karakul lambs by means of EPR spectroscopy (Latypov et al.).

Oxidation Chemistry: The oxidative polymerization of 5,6-dihydroxyindoles (d´Ischia et al.) and the role of peroxidase in melanogenesis (Okun) have been reviewed. Memoli et al. found that certain melanin related metabolites inhibit lipid peroxidation. A hydroxyl radical independent hydroxylation/oxidation mechanism - basically different from the Fenton reaction - which involves direct interaction of the peroxide with a dopamine-Fe(III) chelate was discovered in the oxidation of dopamine to 6-hydroxydopamine and rationalized as one of the possible mechanisms involved in degeneration of neurons in Parkinson´s disease (Pezzella et al.). Riley et al. studied side chain variations in *o*-quinones with respect to melanogenesis targeting and cytotoxicity in a model system. Rozanowska et a. reported on photoinduced oxidation of ascorbate in the presence of melanin. Melanins from opioid-peptides were reviewed (Rosei).

A series of papers described conjugates of amino acids with quinonoids, including a review that contains an account of conjugates formed in insect cuticle sclerotization (Andersen et al.). Electrochemically generated dopamine quinone reacts with N-acetylcysteine to give mainly 5-*S*-(*N*-acetylcysteinyl)dopamine at pH 2 or 7 with minor amounts of 2-*S*-(*N*-acetylcysteinyl)dopamine and 2,5-*S*,*S*-di-(*N*-acetylcysteinyl)dopamine (Xu et al.). Imidazole and *N*-acetylhistidine react under similar conditions with the quinones of *N*-acetyldopamine (NADA) and *N*-β-alanyldopamine to give mainly C-6 adducts (Huang et al.). Serotonin yields, upon electrochemical oxidation in presence of glutathione, 4-*S*-glutathionyl-5-hydroxytryptophan and 7-*S*-glutathionyl-tryptophan-4,5-dionewhere the latter is generated from tryptophan-4,5-dione or by migration of a glutathionylresidue from position 4 to position 7. Another product is an unusual tricyclic pyrroloquinoline (Wu and Dryhurst). The quinones of epinephrine or norepinephrineadd cysteine to give 5-*S*-cysteinyl- and 2-*S*-cysteinyl-adducts which, upon further oxidation either lead to bis-addition products or cyclize to give a number of novel dihydrobenzothiazines (Shen and Dryhurst); analogous cyclization products are not found with the quinone of dopamine (Shen and Dryhurst).

Drug and Metal Binding: Quantitative structure-activity-relationships of drug binding were reported in a meeting of the Am. Chem. Soc. (Famini et al.). The mechanism of incorporation of 2-thiouracil into melanin has been elucidated by Napolitano et al. Cocaine binding to Caucasoid and Africoid hair is influenced by melanin and lipids (Joseph et al.) and was compared in melanin granules and human hair in vitro (Potsch et al.). Wang and Casadevall investigated the binding of trifluoperazine and chloroquine in *Cryptococcus neoformans.*

Binding of iron to neuromelanin of human substantia nigra and synthetic melanin was investigated by EPR spectroscopy (Shima et al.). EPR of neuromelanin extracted from normal human midbrains indicates that iron is present as polynuclear oxy-hydroxy ferric aggregates as well as isolated Fe(III) centres, different from ferritin (Aime et al.). The iron binding capacity of melanin could possibly be used for magnetic resonance imaging of melanomas (Enochs et al.). Mouse fibroblasts and human embryonal kidney cells that were transfected with an expression vector containing a complementary DNA sequence that encodes the human tyrosinase gene (pcDNA3tyr) have a higher ^{111}In-binding capacity which can be detected with MR imaging and scintigraphy (Weissleder et al.).

4-Borono-2-fluorophenylalanine, a specific melanogenesis-seeking compound synthesized for use in boron neutron capture therapy of malignant melanomas, is useful for diagnosis of metastatic malignant melanoma by positron emission tomography (Mishima et al.). A resume of the early developments in boron neutron capture therapy of tumors with ^{10}B-boronophenylalanine was given by Sweet. ^{123}I-α-Methyltyrosine (AMT) is taken up in melanoma cells in vitro and in vivo and is accumulated in melanoma metastases. However, owing to its low sensitivity, the clinical use of whole-body AMT scintigraphy cannot be recommended (Boni et al.). Also, gadopentetate dimeglumine-enhanced MR imaging does not differentiate malignant melanomas from benign melanocytic nevi (Maurer et al.). On the other hand, Menif et al. found that MR imaging is accurate in determining the exact location of lesions in choroidal melanoma and their nature owing to the paramagnetic properties of melanin. Furthermore, N-isopropyl-*p*-^{123}I-iodoamphetamine is incorporated into melanin and melanotic melanoma allowing planar scintigraphy in the detection of malignant melanoma while single photon emission tomography revealed the exact tumour localization, even in the case of a metastatic tumour located in the lung (Watanabe

et al.).
Biosynthesis and Inhibition: Mechanisms of phenol-oxidizing enzymes and applications in biosensors were reviewed (Peter and Wollenberger). A mutational analysis of mammalian tyrosinase revealed that two copper binding sites, CuA and CuB, are both required for catalytic activty, and that most likely His363, His367, and His389 are involved in the CuB site (Spritz et al.). A new tyrosine hydroxylase that is not a tyrosinase occurs in the inner ear and possibly also in other extracutaneous tissues (Benedito et al.). The peroxidase m-RNA of *Sepia* ink gland has been cloned (Gesualdo et al.) and the subcellular localization and function of melanogenic enzymes in that gland has been elucidated (Palumbo et al.). Immune response in insects is linked with melanization (Marmaras et al.). The enzymology of prophenol oxidase activation and melanization of microfilariae in mosquito blood was investigated with inhibitors of serine protease (phenylmethylsulfonyl fluoride), phenoloxidase (diethyldithiocarbamate), and dopa decarboxylase (*m*-hydroxybenzylhydrazine) (Liu et al.). Butterflies contain an develomentally regulated pupal melanization reducing factor which is discussed in three papers by Starnecker and by Starnecker and Bückmann. Different types of fungal melanin are synthesized depending on the growth medium (Eagen et al.). Miranda et al. have investigated L-tyrosine 3-monooxygenase and L-DOPA oxidase in relation to melanogenesis in black and white truffle. A bioassay for mycotoxins is based on a melanin precursor producing ciliate (Gonzalez et al.).
Cellular thiols coregulate the activities of tyrosinase and glutathione peroxidase in opposite directions in melanoma cells: high levels of CysSH regulate down tyrosinase while GPO increases. These interdependent processes could provide melanoma cells with protection against oxidative stress at low as well as at high thiol concentration (Benathan). Insulin inhibits the biosynthesis of melanin and the formation of 5-S-cysteinyldopa and that of melanin in human Swift melanoma cells via the inhibition of tyrosinase activity (Benathan and Labidi). Eller et al. report that DNA-damage enhances melanogenesis.
Natural products chemistry continues to yield new inhibitors melanogenesis. Certain polyene natural products named fusarins inhibit "melanin" biosynthesis from dihydroxynaphthalene (Eilbert et al.). The structures of the natural products MR566A and MR566B, produced by *Trichoderma harzianum*, were elucidated as 1-(3-chloro-1,2-dihydroxy-4-isocyano-4-cyclopenten-1-yl)ethanol and 1-(1,2,3-trihydroxy-3-isocyano-4-cyclopenten-1-yl)ethanol, respectively (Lee et al.). The structure of novel oxazole, MR93B, was elucidated as 4-[(1Z)-3-hydroxy-2-hydroxymethyl-1-propen-1-yl]oxazole (Lee et al.). DL-α-tocopheryl ferulate inhibits melanogenesis (Funasaka et al.). A screening test for insecticides, based on inhibition of tyrosinase and quinone methide isomerase, was developed by Londershausen et al.
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2. Biology of pigment cells and pigmentary disorders

(Dr M. Picardo)

Prof. Riley in an interesting paper published in Dermatology suggests a hypothesis on the mechanisms controlling melanocyte proliferation and naevogenesis in epidermis based on the concept that melanocyte growth is regulated by the transfer of melanosomes to competent acceptor keratinocytes. The hypothesis sounds interesting and could explain both the low rate of proliferation of epidermal melanocytes in vivo and the origin of acquired benign pigmented moles. Several papers have been published on the mechanism of control of melanogenesis. Schallreuter et al. have evaluated the role of pteridines in control of UV-induced pigmentation. The authors report that UVB irradiation increase epidermal TNFα and 6-bipoterin levels, GTP-cyclohydrolase 1 and phenylalanine hydroxylase activities suggesting that UVB can control the supply of tyrosine in the epidermis and that this mechanism could be involved in de novo melanin synthesis. Interestingly, they found that this metabolic cascade was stimulated more in fair than in dark skin types and, even if he number of the examined subjects is too low to give a statistically significant differences, these results are intriguing. Suzuki and coworkers presented data showing that the agouti signalling protein blocked the binding of α-MSH to the MCR1 and inhibits the effect of α-MSH on cell proliferation and tyrosinase activity in normal human melanocytes. These results indicate a similar functional activity of agouti protein in mouse and human melanocytes and suggest a potential physiological role of the protein in human pigmentation. On the same subject Sakari et al. showed that in black melan murine melanocytes, treatment with agouti protein inhibits eumelanin production and the formation of eu melanosomes. Moreover the treatment

produced a time and dose response decrease in melanogenic gene expression On the contrary, treatment with α-MSH stimulated the expression of the same gene. The results obtained suggest that, in vitro, agouti protein can induce eumelanogenic cells to show features of pheomelanic cells. Imokawa et al. presented adjunctive data on the possible role of Endothelin-1 in stimulating epidermal pigmentation. ET-1 treatment induced an increase of tyrosinase and TRP-1 mRNA expression which was inhibited by H7 and exposure to UV light in vivo produced an increase of the ET-1 mRNA expression. These data support the hypothesis that ET-1 is one of the mediators of UVB pigmentation in vivo. Kishikawa et al reported the electron microscopic analyses of dermal end epidermal melanocytes obtained from Ota's nevus and cultured in vitro.

Several papers have been published on the biology of melanoma cells. Rosdahl et al. studied the metabolism of retinol and retinoic acid in culture human melanocytes and melanoma cells. These authors found the expression of mRNA for cellular retinoic acid and retinol binding proteins; the cellular concentration of retinol and its metabolites were 5 times higher in melanocytes than in melanoma cells and the authors conclude that the different concentration and metabolism of retinoids between normal and malignant melanocytes may play a role in the regulation of cell differentiation and growth. Okano-Mitani e coll. described that human melanoma cells can generate LTB4 and LTC4 from LTA4 and that the process is catalysed by two enzymes the LTA4 hydrolase and LTC4 synthetase. Ahmed et al. have evaluated the expression of fibroblast growth factor receptors in naevus-cell naevus and in melanoma. The results suggest that tumour-derived bFGF could be involved in melanoma formation through a paracrine mechanism. Dr. Benathan, continuing his interesting studies on the intracellular metabolism of thiols in melanocytes and melanoma cells, reported that cellular thiols regulate the activities of tyrosinase and glutathione peroxidase in opposite direction. When reducing or excluding the extracellular concentration of cysteine low levels of intracellular glutathione, increased tyrosinase activity and decreased glutathione peroxidase activity were detected. On the contrary higher concentration of cysteine decreased tyrosinase and increase glutathione peroxidase activities. The conclusion of the author was that melanogenesis could be a mechanism compensating a deficit in GSH-dependent antioxidant defences in melanoma cells. Spielholz et al. have evaluated the uptake of dehydroascorbic and ascorbic acid in normal melanocytes and melanoma cells. They found that both melanocytes and melanoma cells can transport vitamin C either in the reduced or in the oxidized forms. However melanoma cells transport dehydroascorbic acid at a rate 10 times faster than melanocytes and that can accumulate vitamin C at concentrations 100 times greater than the corresponding extracellular concentration. They concluded that the differential capacity of melanoma cells to transport the oxidized form of vitamin C can reflect the increased expression of transporters associated with the malignant phenotype. Matsuyoshi et al. reported that even if in melanoma cells the expression of E- and P-cadherin are lower than in melanocytes they showed a stronger homophilic adhesion. Using PCR-RT the authors identified 6 novel and two known cadherins fragments in melanoma cells. Sakai et al. presented an interesting review on the melanosomal proteins and, these structures being specific to melanocytes, could be considered as a source for specific immunetherapies of melanoma. Lowes and coworkers have evaluated the cytokine mRNA expression in 20 primary melanomas and found an association between Th1 cytokines and spontaneously regressing melanoma, suggesting that the intra lesional secretion of these cytokines could be responsible for the regression of the lesion. On vitiligo, Kemp at al. reported the detection of auto-antibodies tyrosinase in 10% of the tested patients with vitiligo using a novel radio immune assay with ^{35}S-labelled recombinant human tyrosinase. The method seems to be sensitive, reproducible and capable of detecting conformational epitopes. Le Poole et al have investigated the expression of integrins in lesional perilesional areas and in cultured vitiligo melanocytes. They did not find any significant differences with respect to controls. Rather, they observed that near to the lesions vitiligo skin contains increased amounts of tenascin in basal epidermis and papillary dermis. The authors suggest that the anti-adhesive effect of this extracellular matrix molecule may contribute to the pigment cell loss in vitiligo. Finally our group have presented data showing a decrease of catalase activity in vitiligo melanocytes in culture and a correspondent increase of vitamin E concentration. Moreover, we have reported that the increased sensitivity to the toxic effect of cumene hydroperoxide in vitiligo melanocytes appeared to be dependent on the alteration of the antioxidants suggesting that the imbalance of the antioxidant system could be the basis for the melanocyte degeneration in the disease.

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Melanocyte cultures
(Dr N. Smit)

Several new papers have appeared which describe some fascinating aspects of melanocyte cultures derived from many different origins. In the paper by Goding and Fisher a meeting review is given from a melanocyte workshop that took place

April 11-14, in Oxted, UK this year. One of the main topics of this review is the development of melanocytes from the neural crest. The importance of the microphtalmia and c-kit genes in the melanocyte development are specifically discussed. Other new reports also deal with the topic of the differentiation of neural crest cells from murine and quail origin (Opdecamp et al, Rao et al and Stone et al). The influence on melanocyte development by endothelin-3 and the c-kit ligand, steel factor are described. Other important studies that may shed a light on pigment regulation by MSH and the agouti protein (ASP) in melanocyte cultures from murine and human origin come from the papers by Sakai et al in EMBO Journal and Suzuki et al in the JID, respectively. The first paper shows that equimolar concentrations of MSH and ASP have similar effects as MSH alone. The effects of ASP alone resulted in a down regulation of the melanogenic enzymes, tyrosinase, TRP1 and TRP2 and also of the activities of tyrosinase and dopachrome tautomerase (TRP2). The results using the human agouti protein and human melanocytes show that the function of ASP in mice and humans is similar. Using B16F1 mouse melanoma cells Graham et al (Pigment Cell Res 10 (5) 298-303, 1997) found that ASP had an antagonizing effect on the action of MSH. This was not observed in the study by Sakai et al. Using the method of Ito and Fujita (Anal Biochem 144, 527-536, 1985) for measurement of eumelanin and pheomelanin only a limited change in the ratio of both types of melanin were found in the mouse melanocyte system (Sakai) whereas for the melanoma cells (Graham) ASP had a stronger effect on the reduction of eumelanin than that of pheomelanin. This may be a demonstration of the large differences one may encounter when using melanocytes or melanoma cells as the model system for studying pigmentation. In this respect the use of both normal and malignant melanocytes in several studies offer interesting information on the differences in the regulation of various aspects of metabolism in these cells (e.g. Easty et al, Halaban et al, Meyskens et al, Shattuck-Brandt et al, Rosdahl et al and Yayon et al). In the study by Meyskens et al also a difference is demonstrated between melanocytes originating from Caucasians and African Americans, suggesting that the latter cells are more efficient in dealing with ROS.
In three different papers the effects of Ca2+ on melanocyte cultures are part of the investigations. Yanase et al show a dose dependent stimulation of melanocyte growth by Ca2+ in culture medium from 0.2 mM to 1.0 mM. In the study by Hedley et al it is described that the intracellular calcium levels strongly influence the attachment of uveal melanocytes to extracellular matrix proteins. In the Barred Plymouth Rock avian melanocytes also a stimulatory effect of Ca2+ on the number of cells with increased pigmentation has been found by Bowers et al.

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4. Photobiology and photochemistry

(Dr M. d'Ischia)

A significant number of papers dealing with the photochemistry and photobiology of melanin pigmentation focused from different perspectives on the putative relationships between UV exposure, pigmentary traits and susceptibility to short term and long term skin damage, including melanoma. Breitbart et al. (Acta Dermato-Venereol 1997, 77, 374-378) investigated 513 patients with melanoma and 498 controls to show that both the history of sunburn and intensive sun exposure are important for the development of melanocytic naevi and, hence, for melanoma. The characteristics of cutaneous pigmentation are also related to atypical melanocytic naevi. Using a portable reflectance spectrometer, Damian et al. (Br. J. Dermatol. 1997, 136, 714-718) measured skin pigmentation in terms of melanin index and demonstrated a good correlation between skin pigmentation and MED. Bessou et al. (J.Invest.Dermatol. 1996, 107, 684-688) analyzed skin phototypes ex vivo to validate their model of epidermal reconstruction with melanocytes, and demonstrated that in all reconstructs the intensity of melanin transfer correlated with the in vivo situation and was induced by UVB. Rubegni et al. (Photochem. Photobiol. 1997, 65, 347-351) specifically addressed the relationship between skin colour, as determined with a colorimeter, and phototype assessed according to Fitzpatrick, whereas Hill et al. (Photochem. Photobiol. 1997, 65, 983-989) studied the photobiology of mouse melanocyte lines with different pigment genotypes, to conclude that the pigment does not protect against UV-induced DNA damage or cell death, and that monocromatic results cannot predict polychromatic responses.
A new hypothesis on the genesis of cutaneous melanoma and a new 7-phase model of malignant transformation of melanocytes were proposed by Dimitrov (European J. Dermatol. 1997, 7, 461-463). These were based on a principle, evolved from the rules of non-equilibrium thermodynamics, of a critical point in local energy storage at a certain level of structural organization of cells in which UV radiation plays a crucial role in the development of malignancy. Hansen et al. (Photochem. Photobiol. 1997, 65, 550-555) found a biphasic transcriptional respose of metallothionein to UV radiation in human melanoma cells, which suggests an early depressed response to acute UV damage possibly switching to a positive protective response at later times. Finally, the risk of melanoma in psoriatic patients treated with PUVA was evaluated by Stern et al. (New England J. Med. 1997, 336, 1041-1045) over a period of 15 years while Kinley et al. (Photochem. Photobiol. 1997, 65, 486-491) investigated the photoprotective properties of epidermal melanogenesis induced by furocoumarins and UVA. From both studies, it could be concluded that PUVA therapy does not contribute significantly to increase skin photoprotection and may increase the risk of melanoma in the long term.

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5. Neuromelanins

(Dr. Marco d'Ischia)

The ability of neuromelanin to cause iron accumulation in pigmented dopaminergic neurons and the possible involvement in neuronal degeneration processes is undoubtedly one of the most active issues in studies of the etiology of Parkinson's disease. Two related papers (Aime et al., Biochim. Biophys. Acta 1997, 1361, 49-58; Shima et al., Free Radic. Biol. Med. 1997, 23, 110-119) specifically address the mechanism of this binding by means of EPR spectroscopy. It is concluded that iron is present in human neuromelanin as polynuclear oxy-hydroxy ferric aggregates as well as isolated ferric centres, and that after binding to melanin, iron can change its location and/or state, as indicated by analysis of spectra at g=4.3, attributable to Fe3+. Along a different line of thought, Galazkafriedman and Friedman (Acta Neurobiologiae Experimentalis, 1997, 57, 217-225) reviewed published experimental results of iron in Substantia nigra and highlighted existing controversies concerning the actual amount of iron, its redox state and the iron binding compounds. While the evidence presented is taken to argue against a large accumulation of divalent iron in post mortem substantia nigra, the authors do not rule out the possibility that iron is indeed involved in the pathogenesis of Parkinson's disease.

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(Dr. F. Beermann)

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8. Melanoma and other pigmented tumours

Experimental melanoma therapy (Dr. N. Smit)

Glutathione is considered as highly important for detoxification reactions in melanoma. In the paper by Palomares et al it is shown that Il-2 has an antagonistic effect on the toxicity of cyclophosphamide towards B16F10 melanoma cells which may be a result of the increases in GSH levels in the IL-2 treated melanoma cells. Hasegawa et al describe that the tyrosinase dependant toxicity of 4-S-cysteaminylphenol (4-S-CAP) is due to the formation of the dihydro-1,4-benzothiazine-6,7-dione product (BQ). This compound was found to be highly reactive towards GSH and a BQ-GSH adduct is formed rapidly in the cells and consequently GSH levels decreased.
Hypoxia treatment of four melanoma cell lines was investigated in order to measure possible changes in radiation sensitivity shortly after or after a longer period of reoxygenation (Danielsen et al). Only minor changes of the radiation sensitivity of the melanoma cells after hypoxia were found. Using a novel oxygen sensor Zilberstein et al were able to measure oxygen consumption rates in melanoma tumors in mice as a result of illuminations during Photodynamic therapy (PDT). Also the reoxygenation during the dark periods could be measured. PDT using 5-aminolaevulinic acid as a precursor of photodynamically active porphyrins was investigated by Abels et al. No complete remissions of A-Mel-3 melanomas in hamsters could be achieved with the treatments. The induction of porphyrins was concluded to be less effective than other photosensitizers such as Photofrin or synthetic porphycenes.

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Malignant melanoma and levodopa: Is there a relationship? Two new cases and a review of the literature. J Am Acad Dermatol 37(2 Suppl. Part 2):332-336, 1997.

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Comments: One approach to overcome these limitations would be to design universal vaccines consisting of standardized gene-transduced neoplastic cell lines or mixtures of gene-transduced cell lines, to be combined with autologous tumor samples if available. We developed and analyzed several different retroviral vectors for their ability to stably express exogenous genes at high levels in a panel of melanoma cell lines. One vector, DCCMV, was found to perform consistently better than the other vectors. The DCCMV vector, which is an extreme example of the double-copy class of retroviral vectors, was subsequently used to generate melanoma cell lines constitutively secreting human interleukin-6 or a soluble form of the human interleukin-6 receptor for potential use in a phase II clinical vaccine trial for the treatment of melanoma patients.

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Melanoma clinical
(Prof. R. Peter, Dr G. Krähn)

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Clinical and prognostic significance of the expression of the c-erbB-2 and c-erbB-3 oncoproteins in primary and metastatic malignant melanomas and breast carcinomas. Anticancer Res. 17(2B):1319-30, 1997.
Comment: The problems of immunohistochemical detection of receptor tyrosine kinases have been addressed in this paper. C-erbB2 is most likely not suitable as a marker for differentiation between benign and malignant pigmented skin lesions. C-erbB3 has not yet been extensively studied in melanoma. It certainly requires further studies on the DNA and RNA level to promote it as a target for future therapies.

- Mooy CM, De Jong PT.
Prognostic parameters in uveal melanoma: a review. Surv Ophtalmol. 41(3):215-28, 1996.
Comment: This paper summarizes the differences between cutaneous and uveal melanomas with respect to penotypical and genotypical characteristics, an aspect underestimated for too long. For both ophtalmologists and dermatologist an informative review.

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Anti-cell death activity promotes pulmonary metastasis of melanoma cells. Oncogene. 14(24):2971-7, 1997.
Comment: An important aspect of the potential of metastasation in melanoma. Though still on the in vitro or animal model level this might be a promissing approach in determining the risk of metastases in vivo.

ESPCR COUNCIL MEETING - WEDNESDAY, 8 OCTOBER 1997 - BORDEAUX

1. The meeting was held in the Conference Centre La Cité Mondiale, Bordeaux on Wednesday, 8 October 1997 from 2.30 - 4.30 pm. The agenda was distributed at the meeting.

2. Attendance at the meeting - Dr. Pavel, Dr. Larsson, Professor Riley, Professor Thody, Dr. Mac Neil, Dr. Borovansky and Professor Taieb (by invitation).

3. Dr. Larsson opened the meeting and Dr. Pavel gave apologies for absence from Dr. Peter, Professor Prota, Dr. D'Ischia, Professor Pehamberger and Dr. Parsons (Professor Ghanem was, at that time, travelling by hire car from Brussels to Bordeaux in a valiant effort to overcome a French rail strike on that day).

4. Dr. Pavel then circulated minutes of the last ESPCR Council Meeting which had been held in Anaheim in October 1996. The Council members read the minutes and approved them - the President signed the minutes.

5. Dr. Pavel gave the Secretary's report. He reported that he had sent out 45 personal letters persuading people to take up membership and had a 25% response rate resulting in something like 12 new members since last year. He emphasised that we should continue doing this. He said that we have approximately 200 members but only 150 paid over 1996 and only 120 have paid up so far in 1997.

The Council decided that if the fee is not paid for 2 years then members will get a letter requesting payment within 3 months (of back fees as well as current fees). If someone has not paid then at 3 years they will be automatically deleted from the membership.

The Secretary also said that he had contacted organisers of other meetings like the Melanoma Meeting in Australia and the Pigmentation Meeting in Bali and given them copies of our membership list.

6. The Treasurer's report was then given by Dr. Larsson in the absence of Dr. Peter. The Council approved the Treasurer's report and it was agreed to publish the 1996 financial report in the bulletin.

7. The next item should have been the Editor's report for the ESPCR bulletin, however Professor Ghanem was not present because of the rail strike as explained in 3. Others present did say that there were lots of positive reports on the website and that the ESPCR bulletin had focussed on meeting reports and it was now possible to visit the Japanese site on the web and you can download the whole bulletin from the website directly. It is also possible to publish photographs in the bulletin from now on. (It later transpired that the increased cost of the bulletin was due to opening up the website. The annual cost of maintaining the website will be very low).

8. The next item was election of ESPCR Officers. The Election Committee comprised Guiseppe Prota, Patrick Riley and Bengt-Larsson who chaired the Committee. Ninety-one ballot forms were received (of which 2 were spoiled). Out of the 89 valid ballot forms, there was one nominee for President (Stan Pavel) who received 88 out of 89 votes, one nominee for Treasurer (Ralph Peter) who received 82 out of 89 votes and 3 nominees for Secretary: Jan Borovansky who received 25 votes, Marco d'Ischia who received 18 votes and Sheila Mac Neil who received 44 votes.

9. Next was an update on the next ESPCR meeting which is in Prague. The organiser (Dr. Jan Borovansky) said that the dates will be 23 September - 26 September 1998. Professor Taieb suggested that Dr. Borovansky could also invite the EORTC members who will be present during the Melanoma Symposium on the Friday morning. Dr. Borovansky accepted the kind offer.

Under Any Other Business, the location of the next International Pigment Cell Society Meeting in Europe was discussed. The next international meeting will be in Nagoya in 1999 and the next one in Europe will be 2002. So far, there is one

offer from Ralph Peter from Ulm in Germany. It was agreed that we should mention this at the ESPCR General Assembly and also put it in the bulletin requesting any further offers for running the meeting in 2002. The Council will discuss the site of next year in Prague and the final decision will probably be made by the Council of the IFECS during the Nagoya meeting.

10. The next item under Any Other Business was the fate of the journal, Pigment Cell Research. In the Japanese Society, every member has a mandatory subscription to the journal but in America and Europe, the subscriptions are voluntary. Europe has the lowest percentage subscription. There was considerable discussion concerning the fate of the journal - apparently, the publishers are able to keep it afloat for another year but only on the understanding that the Society does something to increase the membership. Various options were discussed - this will be taken further under the second ESPCR Council meeting held on Friday.

PRELIMINARY MINUTES OF ESPCR GENERAL ASSEMBLY - THURSDAY, 9 OCTOBER 1997 - BORDEAUX

This meeting was held in the Conference Centre La Cité Mondiale. It was held on the second day of the two and a half day ESPCR Meeting. Approximately 25 members of the ESPCR attended the General Assembly.

1. The chair was taken by the President (Dr. B. Larsson).

2. The minutes of the ESPCR Council Meeting in Anaheim, October 1996 were read, approved and signed by the President.

3. The Secretary (Dr. Pavel) gave his report in which he said that he had written 45 personal letters to colleagues to increase the membership, resulting in some 12 new members. He stated that we have approximately 200 members, of whom 150 have paid up their subscription for 1996 and only 120 have so far paid for 1997. He reported that in a meeting by the Council, it was agreed that those members which had not paid for 2 years would be given a warning letter requesting payment withinn 3 months. A third year of non-payment would result in automatic disqualification from the Society.
He also said that he had contacted the organisers of other meetings making them aware of our membership list ie. the Melanoma Meeting in Australia and the Pigmentation Meeting in Bali.

4. In the absence of the Treasurer, Dr. Peter, Dr. Larsson presented the Treasurer's report for 1996 and said that this would be published in the ESPCR bulletin.

5. Dr. Ghanem gave a report on the ESPCR bulletin. He said that there had been lots of positive reports about the bulletin since it has been available on the website. It is now possible to visit the Japanese site via the web. He stated that, of late, he has focussed on getting meeting reports into the bulletin. It is also possible to now publish photographs in the bulletin. He stated that the cost of opening the website has been met in 1997 but the continued cost of running it on the website will be very low.

There was some discussion concerning the desirability of keeping hard copies of the bulletin for those people who requested it for a few years. There was a general feeling that more and more people would access the bulletin via the website.

6. The new Officers elected to the Society are: Dr. Pavel as President, Dr. Mac Neil as Secretary and Dr. Peter as Treasurer.

7. Dr. Borovansky kindly showed some slides of the venue for the 1998 ESPCR Meeting in Prague and invited members to visit Prague on the occasion of the next ESPCR meeting.

8. Under Any Other Business, the location of the next IPCC Meeting in Europe which will be in 2002 was raised. At present, there is an offer from Dr. Ralph Peter to host the meeting in Ulm. A final decision on the location will be made during the Nagoya meeting. The opportunity to organise the IPCC in Europe in 2002 will be announced in the Pigment Cell Bulletin.

9. The fate of the journal Pigment Cell Research was discussed in light of the fact that the publishers say that it is not financially viable as it stands and there is a need to increase the number of copies purchased by around 100. Various suggestions were made by the membership including invited reviews, making it more accessible to clinicians, reducing the cost of the subscription and making it mandatory as part of the ESPCR membership, page charges, changing publishers,

etc.

10. The meeting was closed by Dr. Larsson.

ESPCR COUNCIL MEETING - 10 OCTOBER 1997 - BORDEAUX

This meeting was held in the Conference Centre La Cité Mondiale, Bordeaux on Friday, 10 October 1997 from 1.00 - 2.00 pm.
Those present were: Dr. Larsson, Dr. Pavel, Dr. Mac Neil, Professor Riley, Professor Ghanem, Dr. Sarna, Professor Thody, Dr. Borovansky, Dr. Garciá-Borrón, Dr. Peter.

1. The chair was taken by the President (Dr. Pavel) who in his previous role as Secretary, circulated an agenda for the meeting.

2. The first item discussed was communication within the ESPCR Council. Council members were urged to communicate by e-mail or fax where this was not yet available or convenient. To facilitate this, the Secretary (Dr. Mac Neil) said that she would shortly send a communication to all Council members and request confirmation of its safe receipt to confirm accuracy of e-mail addresses, fax numbers, etc.

3. The question of nominating candidates for awards by the Pigment Cell Societies was discussed. The International Society has several awards and will accept nominees for awards from its sister societies. It was agreed that a maximum of up to 3 nominees would be forwarded by the Society. For each award, there was some discussion of the nature of the awards and previous recipients of awards and Dr. Riley offered to provide information on the awards and on the previous recipients of the awards. It was agreed that the bulletin would be used to make ESPCR members aware of the existence of the awards and the need for nominees. This item will be put on the agenda of the Council Meeting in Prague.

4. The next section of the agenda dealt with a number of activities that the ESPCR Council members need to pay attention to in the coming year, namely the need to gain financial support from the Society from industrial sponsors, to pay attention to public relations and make potential attendees at the ESPCR meetings aware of our existence, to systematically recruit new members and to undertake Council elections for 1998.
The Secretary agreed to write to all Council members requesting that they consider carefully potential sources of sponsorship for the meetings and that they also inform the Secretary of any meetings that they are aware of with relevance to the ESPCR meetings which we should target for advance publicity of our own meeting. The issue of positively encouraging new members to join the Society will also be raised with the Council members and once we are aware of the number of new Council members required for 1998, existing Council members will be requested to be active in identifying potential willing candidates. Dr. Borovansky offered to deal systematically with the problem of recruiting of new ESPCR members.

5. The next item on the agenda was the position of the ESPCR bulletin in the life of the Society and it was agreed that it is working well and we can use it to an even greater extent in the future. For example, announcements of achievements and developments and relevant anniversaries of events, etc. can be placed in the bulletin. We can use it to make people aware of the need for ongoing and future sponsors, to remind members to inform us of other relevant meetings and to be aware of the need to recruit new members and propose existing members for Council elections in 1998 and to propose existing members of the Society as nominees for awards of the International Society for Pigment Cell Research.

6. Next, the meeting discussed cooperation with other European Societies - Dr. Sarna suggested a joint symposium with the European Society for Photobiology in Prague and this received warm approval. After some discussion of topics, the topic of phototherapy and photoprotection were identified. Dr. Sarna, on behalf of the European Society for Photobiology, offered a contribution of up to DFL 3,000 to assist the ESPCR to organise a joint symposium on a topic of mutual interest. Two other societies: the European Society of Radiation Biology and the European Society for Dermatological Research are potential societies for joint symposia. Dr. Thody also mentioned a new small society on neuropeptides. There is also the European Immunodermatology Society. Dr. Pavel will contact the European Society for Dermatological Research.

7. Dr. Borovansky announced that the Prague ESPCR Meeting would be organised by Dr. Matous and himself and that arrangements were well underway. With respect to the finances of the meeting, Council members offered a pump-priming budget of up to £5,000 to assist Dr. Borovansky in the running of the meeting.

8. Under Any Other Business, the issue of the fate of the journal Pigment Cell Research was considered. Dr. Pavel

reported that at the Publications Committee, a number of suggestions were made concerning invited reviews obtaining more clinical pigmentation material. There was a strong feeling that the journal should be assisted to survive and that steps should be taken to increase its research rating. However, there is an equally strong feeling that mandatory subscription to the journal at the current subscription price would have an adverse effect on the membership of this Society currently. Dr. Pavel will write a personal letter to all ESPCR members who have not been subscribing to the PCR.

70th anniversary of Prof. J. Duchon

by Dr. J. Borovansky & Dr. S. Pavel

Prof. MUDr J. Duchon DrSc was born 70 years ago on 27th July 1927 in Prague into a family of university professor of agricultural chemistry. His inborn inclination to natural sciences was further deepened during his grammar school studies by Dr. J. Kostir who later became the most charismatic university Professor of biochemistry of Czechoslovakia in the 20th century.

During the years 1946-1952, he studies medicine at the Charles University in Prague and simultaneously spent 5 terms of chemistry at the Faculty of Natural Sciences. Already as a student he began scientific and pedagogical work at the 2nd Department of Medical Chemistry and Biochemistry of the Faculty of General Medicine (later renamed as 1st Faculty of Medicine), Charles University in Prague, where he spent all his fruitful professional career consecutively as a lecturer (1952), associated Professor (1966) and full Professor of biochemistry (1993). He acted also as a head of the 2nd Department of Medical Chemistry and Biochemistry since 1970 till 1995; in 1996 he retired.

The scientific journey of Prof. Duchon was predetermined by his teachers. Prof. A.F. Richter, the first head of Dr. Duchon was a strict but enthusiastic scientist who was laying stress on enough scientic tasks for young lecturers even during summer holidays. Before his departure for a summer leave he took, therefore, a dusty jar labelled "Waelsch horse melanin" and asked Dr. Duchon to analyze it. This moment decided not only about the research program of Dr. Duchon, but also about he research program of Dr. Duchon, but also about the research profile of the whole Department until now. The second teacher of Dr. Duchon, Prof. J. Sula, the founder of biochemical oncology in Czechoslovakia extended the research activities of Dr. Duchon in the area of malignant melanoma.

At the beginning, Prof. Duchon paid a special attention to metabolic changes in melanoma patients. He was first to demonstrate an increased excretion of homovanillic acid (Clin Chim Acta 7, 1962, 443) and vanillactic acid (Clin Chim Acta 18, 1967, 487) in the urine of melanoma patients. In cooperation with Dr. Pechan he amply extended our knowledge on melanogenuria (Ann NY Acad Sci 100, 1963, 1048) and with Dr. Matous identified 5-hydroxy-6-methoxyindole-2-carboxylic acid and 6-hydroxy-5-methoxyindole-2-carboxylic acid in melanoma urine (Clin Chim Acta 16, 1967, 397), the compounds which were lately "rediscovered" as disease progression markers. In 1967-1968 he worked at the Department

of Dermatology, Harvad Medical School in Boston in the lab of Prof. T.B. Fitzpatrick. After return to Prague, he put together a research group engaged in melanosome research (Pigment Cell 1, 1973, 165) which brought the first evidence that melanosomes consist of several proteins (Cas lek ces 111, 1972, 218). Postgraduate students of Prof. Duchon prepared and defended 10 Ph.D. and 3 habilitation theses. Prof. Duchon has published almost 150 scientific papers.

As a university teacher he was permanently engaged also in pedagogical work and wrote several textbook of medical chemistry and biochemistry.

in 1979 he took part in the 1st European Workshop on Melanin Pigmentation in Lyon and organized the 3rd Meeting in Prague in 1981. Ever since he has become an indispensable participant of the meetings organized by the ESPCR who enthusiastically took part in discussions.

We wish Prof. Duchon a long, happy and healthy retirement and we look forward to meeting him at future meetings of the European Society for Pigment Cell Research.

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

1998 **8th ESPCR Meeting: Prague, 23 - 26 September**

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1999 **XVIIth International Pigment Cell Conference: Nagoya Congress Center, Japan, October 30 - November 3**

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2000 **9th ESPCR Meeting: Krakow**

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Dear Colleagues,

As your new Secretary, I am writing to urge you to add some important dates to your diaries for 1998 now.
The next meeting of the European Society for Pigment Cell Research is in Prague, 23 - 26 September 1998. Additionally, it is highly likely that there will be a joint symposium with the European Society for Photobiology at this meeting and there will also be a melanoma meeting the weekend before in Vienna with the possibility of there being some social events linking the Vienna and Prague meeting.
We can all be sure of a warm and friendly welcome in Prague and I look forward to seeing you there.
With respect to supporting the Prague meeting, I would be glad to hear details of any potential sponsors for the meeting.
Secondly, I would like to make a plea to encourage members of your research groups and your colleagues to join the ESPCR - we are a very friendly Society, ideal for introducing younger scientific colleagues to a multi-disciplinary approach to the melanocyte and an excellent Society for fostering collaborative research in Europe.
I look forward to seeing you in Prague.

Yours sincerely

Dr Sheila Mac Neil

Postdoctoral position available

A postdoctoral position is available for up to 3 years to study the biology of melanosomes. Current interests include the biogenesis of the melanosome, the relationship between melanosomes and lysosomes, and the interactions among melanosomal proteins in the synthesis and deposition of melanin. Our approach combines molecular and cellular techniques with the power of mouse genetics. The successful candidate will be a higly motivated recent doctoral graduate from Europe, Canada or the US with experience in either cell biology, molecular biology or both.

Interested candidates may submit a C.V. and the names, addresses and fax/e-mail numbers of 3 references to:

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Postdoctoral Scientists

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Web site: www.jnj.com

ESPCR
JSPCR
PASPCR

A Letter from the President -

What are the function(s) of the IFPCS, or perhaps more accurately put, are there any function(s) of the IFPCS? The answers can be found on the IFPCS Web page (http://lenti.med.umn.edu/paspcr/ifpcs.html), but for those who prefer the old fashioned way, please read on . . .

The IFPCS was established with several main goals in mind :

1. To foster and enhance research on pigment cells and pigmentation among the regional Societies.
2. To foster scientific collaboration, cooperation and communication among the regional Societies.
3. 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 lecture.
4. To provide consultation and information regarding all aspects of pigmentation and related topics.
5. To encourage the dissemination of knowledge related to pigment cells by the establishment, sponsorship and support for the publication of books, bulletins, newsletters, journals, reports or other means.

How are we doing with respect to these goals? Well yes, I would like your feedback, but to be honest, I have my own opinions on each of the above topics and would like to share them with you.

Historically we have actually worked to meet these goals somewhat in reverse order. Goals #4 and #5 were achieved by establishing an official IFPCS-sponsored journal, ***Pigment Cell Research***, about 10 years ago. The journal has grown steadily but is in need of your renewed support. Increasing publication costs are squeezing its production costs and it is essential, if we wish to keep our journal, that we all contribute to its health and vitality by: (1) subscribing to it, and (2) by submitting papers to it for publication. Our Societies have made a strong commitment to support our journal and the hard work has already been done; if we don't renew our efforts now to support it financially and scientifically, we most probably will lose it within the next several years. I would urge each of you to make sure your research group and/or library subscribes to the journal, that you submit papers to it and that you cite its pertinent references where applicable in your publications. A quick and unofficial look at current statistics in these areas is quite revealing. Support of the journal with respect to submitted manuscripts is similar among the regional Societies: Of papers published in

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Pigment Cell Research in 1996 and 1997, 27% originated from members of the ESPCR, 29% were from the JSPCR and 31% were from the PASPCR; the remaining 13% of papers published were from nonmembers. Thus there is comparable support of the journal from the regional Societies and this is great. On the other hand, distribution of subscriptions by Society is not so equitable: there were 33 member subscriptions from the ESPCR, 166 from the JSPCR and 43 from the PASPCR. Since the membership base is comparable among the three regional Societies, we obviously need to stimulate our ESPCR and PASPCR members to subscribe to our journal, not only to make its support more equitable, but to improve its circulation and usefulness. I would urge each of you to take a moment to assess whether your laboratory and/or library is receiving the journal and if not, to correct that for 1998. Having a specialty journal is a tremendous synergistic resource for our Societies and we should all commit to working to preserve it.

Goal #3 is probably the most obvious and publicly visible effort of the IFPCS; the social and scientific success of the ***International Pigment Cell Conferences*** (IPCC) has grown with each meeting, and each IPCC seems to be more exciting and stimulating than the last. Prof. Ito, chair of the next IPCC, and his Organizing and Scientific Committees have already designed the outlines of our next IPCC which will be held in Nagoya, Japan in 1999. I would invite each of you, not only to attend the meeting, but to watch its development over the next 2 years by tuning in at regular intervals to the IPCC Website (**http://lenti.med.umn.edu/paspcr/17ipcc.html**). That site already has been stocked with useful information about the development, format, social and scientific program of the Nagoya IPCC.

Goals #2 and #1 have been the most recent emphasis of the IFPCS. The ***Special Expert Groups*** are now going full speed ahead; check out their activities from their home pages on the Web (**http://lenti.med.umn.edu/paspcr/experts.html**) and sign up to be on one or more of them. Those groups are not only promoting active research and collaborations within their own specialties, but will provide input into the design and scientific program of the next IPCC. We now have Expert Groups in the subdisciplines of: **Biology of Melanoma, Developmental Biology, Genetics of Pigmentation, Hypo / Hyper-Pigmentation, Ocular / Extracutaneous Albinism,** and **Vitiligo**

The IFPCS has established a ***Scholars Travel Stipend*** program to promote travel aimed at establishing international collaborations. I would urge any of you who have thought of travelling to another lab to learn a new technique or to establish a collaboration, but haven't had the resources to do so, to apply for one of these Travel Stipends. Conversely, if you want someone to visit your lab for the same reasons, encourage them to apply. The level of financial support (i.e. $3,000) should be sufficient to cover expenses for 1 – 3 months of travel. Each regional Society has 3 of these grants to award prior to the Nagoya IPCC and one such application has already been funded. You can check on the details of this program, review the names of awardees and the scopes of their projects, and acknowledge our magnanimous corporate donors, at the relevant Web site (**http://lenti.med.umn.edu/paspcr/travel.html**). I would like to express my thanks to the following companies for their financial support of this program and hope that other companies will join their ranks: **Beiersdorf AG, Clairol Inc., Nikko Chemicals, Procter & Gamble Co., Shiseido, Taisho Pharmaceutical Co.,** and **Unilever Research.**

Finally, the IFPCS Council has just agreed to distribute the Newsletters and Bulletins from each regional Society to members of the other regional Societies to further facilitate exchange of information.

In sum, the IFPCS is healthy, interactive and functioning well. Memberships in our constituent regional Societies have been increasing steadily, and there has been a tremendous influx of fresh faces into the Offices and Councils, not only of those Societies, but into the IFPCS itself. The study of pigmentation is now in the forefront of scientific research in a variety of disciplines, and we are all in an advantageous position to further our own research and that of our colleagues. I would like to thank all of you for your confidence and support and particularly to thank each of the IFPCS Council Members who have all worked extremely hard to achieve the progress listed above. I'll look forward to seeing you in Nagoya.

Vince Hearing
IFPCS President

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