DALTONIANA

NEWSLETTER
OF THE INTERNATIONAL RESEARCH GROUP ON COLOUR VISION DEFICIENCIES

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November 1990 No. 69

IRGCVD News

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LETTER

Dear Colleagues

We are writing to solicit collaborators to join an ongoing study of the molecular genetics of tritanopia. Our study is aimed at determining the genetic alterations responsible for inherited tritanopia. Several families analyzed to date carry mutations in the gene encoding the blue-sensitive cone pigment. We are interested in expanding this study and would like to include tritanopes from families with more than one affected member. We would also be interested in male subjects who present with achromatopsia/monochromacy with a functioning red- or green-sensitive cone system (such subjects are likely to be tritanopes with a concurrent protanopia or deuteranopia).

For our analysis we would require a small sample of peripheral venous blood from the subject and appropriate family members.

All collaborators will be authors on the resulting publications.

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Johns Hopkins University School of Medicine
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THE SECOND W.S. STILES LECTURE

will be delivered by

PROFESSOR M.F. LAND, FRS

Professor of Biological Sciences, University of Sussex

IMAGES IN THE EYES OF ANIMALS

5.30 p.m. Thursday, 15th November, 1990

CHAIRMAN

Professor S. Zeki
Professor of Neurobiology, University of London

DARWIN LECTURE THEATRE
UNIVERSITY COLLEGE LONDON
GOWER STREET
WC1E 6BT

ADMISSION FREE WITHOUT TICKET

LITERATURE SURVEY

Limits of binocular fusion in the short wave sensitive ('blue') cones. H R WILSON, R BLAKE and J POKORNY. Vision Res, 1988, 28, 555-562.

Stereoscopic depth perception is possible when the short wave sensitive (SWS or 'blue') cones are isolated using a yellow adapting field. We have measured the maximum disparity that can be fused (the diplopia threshold) as a function of the separation between pairs of dots or lines. Under all conditions these diplopia thresholds are the same for the isolated SWS cones as for the entire visual system. In addition, SWS diplopia thresholds vary as a linear function of dot or line separation, so that they exhibit disparity scaling. Further experiments show that disparity scaling is dependent upon the presence of low spatial frequencies in the stimulus and not upon the retinal eccentricity of stimulation. These data indicate that the SWS cones provide information to the disparity processing system through more than one low spatial frequency channel but not through high frequency ones - **The Authors.**

Le devenir des familles protan + deutan de Franceschetti et Klein (1949-1956) une génération après. (The progeny of the two protan + deutan families described by Franceschetti and Klein (1949-1956) one generation later). A ROTH, D KLEIN, F PACCOLAT, D HERMÉS, M PELIZZONE, J L MANDEL and R FEIL. Ophtalmologie, 1989, 3, 275-278.

The progeny of the couple of which the husband was a protanope (Franceschetti, 1949) was examined (3 generations) in 1986 and 1987. This couple had 4 children of which 3 sons were deutan and 1 daughter, a double carrier was phenotypically normal. This girl, in her turn and in exemplary fashion, had 3 children: 1 daughter, being a simple carrier was phenotypically normal, 1 son was protan, and 1 son was deutan. The study of genomic DNA of 3 normal subjects reveal the presence of two genes responsible for green and one gene responsible for red. The genomic DNA of a protanomalous subject shows a modification of the gene for red while that of two deuteranopes shows absence of genes responsible for green. The descendents of the second couple, in which the husband was a deuteranope, (Fransceschetti and Klein, 1956) were all female and, therefore, were all phenotypically normal - **J. Vola.**

Eye movement perimetry in glaucoma. G E TROPE, M EIZENMAN AND E COYLE. Can J Ophthalmol, 1989, 24, 197-199.

Present-day computerized perimetry is often unreliable owing to the need to maintain central fixation over long periods while repressing the normal response to presentation of peripheral stimuli. We tested a new method of perimetry that does not require prolonged central fixation. During this test eye movements were encouraged on presentation of a peripheral target. Twenty-three eyes were studied with an Octopus perimeter, with a technician monitoring eye movements. The sensitivity was 100% and the specificity 23%. The low specificity was due to the technician's inability to accurately monitor small eye movements in the central 6 field. If small eye movements are monitored accurately with an eye tracker, eye movement perimetry could become an alternative method to standard perimetry - **I.A. Chisholm.**

Failure of vitamin E to protect the retina against damage resulting from bright cyclic light exposure . M L KATZ and G E ELDRED. Invest Ophthalmol Vis Sci., 1989, 30, 29-36.

Cumulative light-mediated damage to the retina over a long time period may be involved in the development of age-related retinopathies. Light is thought to produce retinal damage by initiating autoxidative reactions among the molecular components of the retina. Animal experiments were therefore conducted (1) to confirm that long-term differences in cyclic

light intensity affect the rate of age-related photoreceptor cell loss from the retina; and (2) to determine whether the antioxidant, vitamin E, is an effective inhibitor of damage to the retina by bright cyclic light. Vitamin E deficiency did not enhance the effect of bright cyclic light in reducing photoreceptor cell densities. Thus, it appears unlikely that retinal damage by cyclic light occurs via an autoxidative mechanism - **I.A. Chisholm.**

A new test of luminous efficiency for babies. D MAURER, T L LEWIS, P CAVANAGH and S ANSTIS. Invest Ophthalmol Vis Sci, 1989, 30, 297-303.

The minimum motion method was used to measure the luminous efficiency of red and green and of yellow and blue for "normal" 1- to 3-month-old babies, and for one 3-month-old boy destined to be color-deficient because of a deutan mother. Subjects watched a display which created apparent motion, the direction of which depended on the relative luminance of the colors. To determine the equiluminant points, we observed the optokinetic nystagmus elicited by the display as the relative luminance of the colors was varied. The equiluminant points of the normal mothers and their infants were similar to each other but different from those of the deutan mother and her son. This method demonstrates the early maturation of input from red and green cones into achromatic pathways - **I.A. Chisholm.**

Investigation of the source of the blue field entoptic phenomenon. S H SINCLAIR, M AZAR-CAVANAGH, K A SOPER, R F TUMA and H N MAYROVITZ. Invest Ophthalmol Vis Sci, 1989, 30, 668-673.

The cellular source of the blue field entoptic phenomenon was investigated in two microvascular preparations using video-microscopy with lighting conditions similar to those under which the entoptic phenomenon is visualized within the human eye. Microvascular flow was simultaneously videotaped under transmission illumination of 430 nm and under unfiltered illumination. In a second animal model, alternating observations were made using transmission illumination at 430 nm and epi-illumination fluorescence microscopy with leukoytes rendered fluroescent. In both prepartions, low magnification video-microscopy using 430 nm illumination produced a field of particles, which were brighter than the background, flowing within a network of dark vessels. The appearance of the particles and their movement simulated the blue field entoptic particle motion. Under higher magnification, the particles were demonstrated to be leukocytes. The results of this study of two microvascular preparations strongly suggest that in the human eye the blue field entoptic phenomenon is produced by leukocytes flowing within the macular retinal microvasculature - **I.A. Chisholm.**

Adult vernier thresholds do not increase with age; vernier bias does. **J V ODOM, R J VASQUEZ, T L SCHWARTZ and J V LINBERG. Invest Ophthamol Vis Sci, 1989, 30, 1004-1008.**

Vernier acuity and vernier bias were examined in persons aged 20 to 79 years using a method of adjustments. Vernier bias (mean error) showed a sharp increase between 35 and 45. Vernier acuity (standard deviation or precision of alignment) did not vary significantly with age. These different results indicate the importance of separate evaluation of acuity and bias. Vernier acuity is little affected by minor optical changes that occur with age. Therefore, normal vernier acuity in older persons suggests that the neural substrates which underlie fine-grain discrimination of object location are unaffected by aging over the range investigated - **I.A. Chisholm.**

Autosomal dominantly inherited macular dystrophy with preferential short-wavelength sensitive cone involvement. G H BRESNICK, V C SMITH and J POKORNY. Am J Ophthalmol, 1989, 108, 265-276.

An apparent tritan-like color vision defect was detected in five members of a family, spanning three generations, showing an autosomal dominant inheritance pattern. The

defect was associated with mild macular pigmentary changes, poor foveolar reflexes, and slightly reduced visual acuity. Results of various color vision tests, including PIC plates, arrangement tests, anomaloscopes, and investigation utilising the Wald-Marré technique, indicated preferential involvement of the short-wavelength sensitive cone system, with relative preservation of the middle- and long-wavelength sensitive cone systems. Both anomaloscope testing with larger (8-degree) fields and short-wavelength sensitive electroretinography indicated some short-wavelength sensitive cone system involvement beyond the central macula. The abnormal macular findings and mild reduction in visual acuity distinguish this condition from congenital tritanopia; the normal optic disks distinguish it from autosomal dominant optic atrophy - **I.A. Chisholm.**

An electroretinographic and molecular genetic study of X-linked cone degeneration by E REICHEL, A A BRUCE, M A SANDBERG and E L BERSON. Am J Ophthalmol, 1989, 108, 540-547.

A 15-year-old boy was found to possess near normal visual acuity in company with protanopia. His mother and a maternal aunt also had normal visual acuity, but diminished cone electroretinograms with predominant loss of red cone function. His maternal grandfather and a great uncle had a visual acuity of 20/200, a deficiency in color vision, signs of macular degeneration, and diminished cone electroretinographic responses. Genomic DNA isolated from these patients was analyzed with a red cone pigment gene DNA probe that disclosed deletion in the red cone pigment gene. A defect in a gene encoding for a cone photoreceptor protein may lead to a cone photoreceptor degeneration - **I.A. Chisholm.**

Fast and slow oscillations of the electro-oculogram in Best's macular dystrophy and retinitis pigmentosa. R G WELEBER. Arch Ophthalmol, 1989, 107, 530-537.

Fast oscillations (FOs) of the electro-oculogram are fluctuations in the standing potential of the eye that are greatest in response to stimulation by dark and light periods of approximately 1.25 minutes each, in contrast to the slow oscillations (SOs), which are greatest in response to dark and light periods of approximately 12.5 minutes each. The FOs and SOs were measured in four families with Best's macular dystrophy who were found to have marked loss of the SOs with relatively preserved normal and near-normal FOs. A patient with autosomal recessive retinitis pigmentosa demonstrated FOs of abnormal phase and markedly subnormal SOs. Six patients with early RP had greater attenuation of FOs than SOs - **I.A. Chisholm.**

Cone-rod dystrophy. K YAGASAKI and S G JACOBSON. Arch Ophthalmol, 1989, 107, 701-708.

Three patterns of visual dysfunction were identified in patients with autosomal recessive cone-rod dystrophy using rod and cone electroretinography and light- and dark-adapted static threshold perimetry. In the first pattern, there was a central rod and cone scotoma with eccentric fixation, mild peripheral retinal dysfunction equally affecting rod and cone systems, and slow progression. The second pattern, which was relatively more severe, also showed a central rod and cone scotoma and eccentric fixation; however, there was more cone than rod dysfunction detected by electroretinography, and function was lost in the peripheral visual field before it was lost in the midperipheral field. A third pattern, which was rapidly progressive, showed central unsteady fixation and no measurable cone function. Patches of rod function were retained in the central and infero-temporal regions of the visual field. Most of the patients studied fit within the three patterns and the patterns were consistent within families - **The Authors.**

The photoreceptors in atypical achromatopsia. R F HESS, K T MULLEN, L T SHARPE and E ZRENNER. J Physiol, 1989, 417, 123-149.

The receptoral mechanisms underlying the vision of two atypical achromats of the complete variety were studied with standard psychophysical procedures. Under scotopic conditions the spectral sensitivity of each achromat was well described by the CIE (Commission Internationale de l'Eclairage) scotopic sensitivity function and the recovery of sensitivity after a retinal bleach showed characteristic duplex behaviour with the time constant of recovery of the slower phase matching that of normal rod vision for both foveal and peripheral stimulation. Their spectral sensitivity was measured under the conditions of chromatic adaptation in order to reveal any residual middle or long wavelength cone activity. Only one photopic spectral response was found and this was adequately described by the spectral sensitivity function of Stiles' Π_3 mechanism of normal vision. Increment threshold measurements as a function of background intensity revealed a double-branched function in the fovea. The lower branch was found to have the spectral sensitivity of the rods; the upper branch that of Stiles' Π_3 mechanism. Stiles-Crawford measurements of directional sensitivity confirmed that the branch with the rhodopsin action spectrum had the directional sensitivity of rods and that the branch with the action spectrum of Π_3 had the directional sensitivity of cones. There was no evidence for hue discrimination under photopic conditions. Regions of apparently normal performance on hue discrimination tests on more careful examination could be explained by luminosity judgements mediated by short wavelength-absorbing receptors. We reject the notion of there being rhodopsin-filled cones in the foveas of these subjects. The foveal and peripheral vision of each of these achromats can be adequately described in terms of the participation of only two types of receptor, namely normally functional rods under scotopic conditions and normally functioning short wavelength-absorbing cones under photopic conditions. They are therefore functional blue mono-cone monochromats, an explanation which was originally proposed by Blackwell and Blackwell (1957) over thirty years ago - **The Authors.**

Human photopic vision with only short wavelength cones: post-receptoral properties. R F HESS, K T MULLEN AND E ZRENNER. J Physiol, 1989, 417, 151-172.

Spatial and temporal contrast sensitivities were investigated in two subjects whose photopic vision has been previously shown to be subserved by only short wavelength cones. Spatial contrast sensitivity was uniformly reduced compared with that of the normal trichromatic observer. Peak contrast sensitivity reached 40 which is a factor of 2-3 better than previous estimates and extrapolated acuity was around 15 cycles deg^{-1}. Central, non-aliased grating acuity was between 6-9 cycles deg^{-1}. This declined with eccentricity such that at 20 deg it was around 1 cycle deg^{-1}.The variation in contrast sensitivity across the visual field was measured for a range of different spatial frequencies. It was found to be of the same form as that for the normal trichromat but reduced in overall sensitivity. Temporal contrast sensitivity was measured for two different spatial frequencies and found to exhibit the spatio-temporal covariation which is typical of normal trichromatic vision. Temporal acuity exhibited a strong dependence on illuminance and reached asymptotic values of around 40-45 Hz. While this is more than a factor of two above most previous estimates for the short wavelength receptors of normal vision it agrees with some more recent estimates obtained using a different technique. Temporal resolution was found to be evenly distributed across the visual field. Similarities were found between the post-receptoral properties of these achromats and the properties of the isolated blue mechanism of normal vision and also the properties of normal luminance contrast processing in general. The present results provide an upper bound on the contribution of the short wavelength mechanism to normal vision and also provide a suitable model of its possible contribution to the processing of luminance contrast in the normal visual system - **The Authors.**

SYMPOSIUM OF THE INTERNATIONAL RESEARCH GROUP ON COLOUR VISION DEFICIENCIES

June 20 - 23, 1991 Sydney, Australia

GENERAL INFORMATION

VENUE Electrical Engineering lecture theatre, University of New South Wales.

ABSTRACT Deadline for submission - February 15, 1991

Special Themes - a) Occupational consequences of colour vision deficiencies,
b) Molecular genetics of colour vision deficiencies, and
c) Spatial aspects of colour vision

SUBMISSION OF ABSTRACT instructions are on pages 9 and 10.

SCIENTIFIC PROGRAMME

Two of the sessions will be devoted to posters and one will be an IRGCVD Business Meeting.

June 21	8:00 am - 5:00 pm	
June 22	9:00 am - 5:00 pm	
June 23	9:00 am - 5:00 pm	
June 24	Joint IRGCVD-AIC Meeting	

SOCIAL PROGRAMME

June 20	2:00 pm - 6:00 pm	Tour of Sydney
	6:00 pm - 10:00 pm	Welcome Reception
June 21	7:00 pm - 11:00 pm	Harbour Cruise
June 22	7:00 pm - 11:00 pm	Australian Dinner

ADDITIONAL ACTIVITIES FOR ACCOMPANYING PERSONS:

June 21	9:00 am - 4:00 pm	Taronga Park Zoo
June 22		Free shopping day
June 23	9:00 am - 5:00 pm	Blue Mountains

METHODS OF PAYMENT FOR REGISTRATION FEES AND ACCOMMODATION

Either bank cheque or bank transfer may be made in Australian Dollars.

i) In the case of a bank cheque, please make it in favour of IRGCVD XIth Symposium and send it together with the completed Registration and Accommodation form.

ii) In the case of a bank transfer, please make it through your own bank to the IRGCVD account at the State Bank, University of New South Wales Branch, A/C 862047-00.
Enclose a copy of the bank transfer with your Registration and Accommodation form.

CANCELLATIONS AND REFUNDS OF REGISTRATION FEES

Written notification should be received by the SECRETARIAT no later than May 15, 1991.

Refunds: Before May 15, 1991: Less 20% for administration costs.
After May 15, 1991: No refunds.

ACCOMMODATION

Arrangements have been made with the most conveniently located motels and University residential colleges. Please see the "REGISTRATION AND ACCOMMODATION FORM" and information on the next page.

For all communications, please mail or fax to:

IRGCVD SYMPOSIUM SECRETARIAT
School of Optometry
University of New South Wales
Kensington
New South Wales 2033
Australia

Phone: Int + 61 2 697 4629. Fax: Int + 61 2 313 6243. Telex: AA26054
e-mail: sdain%usage.csd.unsw.oz.au@murtoa.cs.mu.oz

SYMPOSIUM OF THE INTERNATIONAL RESEARCH GROUP ON COLOUR VISION DEFICIENCIES

June 20 - 23, 1991

AND JOINT IRGCVD-AIC MEETING

June 24, 1991

ACCOMMODATION INFORMATION AND FLIGHT HINTS

				Distance to venue
1.	Barker Lodge Single Twin Share	Tel: Int + 61 3 662 8444 $110 $120	32 Barker St, Kingsford Basis: room only	5 mins walk
2.	Eastside Motel Single Twin Share	Tel: Int + 61 3 663 0631 $85 $95	147 Anzac Pde, Kensington Basis: room only	10 mins walk
3.	Gemini Motor Inn Single Twin Triple	Tel: Int + 61 3 399 9011 $115 $125 $135	65 Belmore Rd, Randwick Basis: room only	20 mins walk
4.	New College Single Students receive an additional discount	Tel: Int + 61 3 697 5437 $42	On Campus Basis: Bed and Breakfast	5 mins walk
5.	International House Single Student Kosher cuisine	Tel: Int + 61 3 697 5436 $40 $25	On Campus Basis: Bed and Breakfast	5 mins walk

PLEASE NOTE: This accommodation has been selected for its vicinity to the University of New South Wales. If you are also attending the AIC meeting (25-28 June) and would prefer not to change accommodation, you will find that the AIC meeting site is reasonably accessible from this locality by public transport and taxis are plentiful and relatively cheap.

FLIGHT HINTS

June is the cheapest time to fly to Australia. To obtain the cheapest fares will require a minimum stay of 14 days. For instance, the cheapest return flight from Britain by QANTAS is less than £900 at present. QANTAS also offers flights within Australia for a very modest fee to its own passengers and offers a cheap within Australia pass to passengers arriving on other airlines. For instance, it is possible to fly into Cairns and visit the Great Barrier Reef, fly to Sydney for the IRGCVD and AIC symposia and then fly to Melbourne for the CIE meeting all within the one ticket cost This is only available on airlines which make internal flights; mainly QANTAS although the Sydney/Melbourne flight is available with British Airways, KLM, Malaysian and Garuda Indonesia. To make your tour even more enjoyable, at least one stopover before you reach or after you leave Australia can be made even on the cheapest ticket. Travelling from Europe these can be in places like Bangkok or Singapore and from North America in Hawaii or Tahiti. In Britain, United States and Canada the company to contact about these flights and other pre- and post-conference tour options is Jet-About Travel (owned by QANTAS). In other countries contact your QANTAS office. Further information on flights and prices will be communicated as it becomes available. If you need specific information and your local airline or travel agent is unhelpful then contact the local organizer.

XIth IRGCVD SYMPOSIUM AND JOINT IRGCVD-AIC MEETING

INSTRUCTIONS FOR SUBMISSION OF ABSTRACTS

Due date for abstracts to be received in Sydney is 15 February 1991

Abstracts must be submitted:

1. on the form provided,
2. on an Apple Mackintosh disk as a Microsoft Word file (any version) or a text file readable into Microsoft Word.
3. on an IBM or IBM compatible disk as text file, or
4. by e-mail.

The Abstract itself should contain about 200 words.

Abstracts submitted on the form MUST be camera ready. No retyping will be done. Abstracts will be reproduced without reduction. Use 1 1/2 line spacing and print no smaller than 10 point (this paragraph is 10 point with 1 1/2 spacing). Since computer and e-mail submissions will be printed in Helvetica font, it is preferred, but not essential, that printed submissions also be in Helvetica font. Dot matrix printing (other than near letter quality) is NOT acceptable because it does not reproduce well. Facsimile (Fax) submissions are NOT acceptable.

ADDRESS FOR CONVENTIONAL MAIL AND DISC SUBMISSIONS AND OTHER ENQUIRIES

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School of Optometry
University of New South Wales
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Kensington
New South Wales 2033
Australia
Phone: Int + 61 2 697 4629 (Universal Time + 10 hours)
Fax: Int + 61 2 313 6243

E-MAIL SUBMISSION OF ABSTRACTS

The following scheme is adapted from that of the American Academy of Optometry with permission of and thanks to its author Dr Larry Thibos.

Abstracts may be submitted by e-mail.

The address for email abstracts is:

sdain@usage.csd.unsw.au.oz

From the USA this has been achieved by !munnari.usage.csd.unsw.oz.au!dain@uunet.uu.net

From Europe this has been achieved by sdain@usage.csd.unsw.au.oz@earn.relay

The method of getting the message to the oz gateway will vary with your network and the country in which you are located. If in doubt or difficulty, mail or fax your email address to Dr Dain who will e-mail you so that you can get the return pathway from the message. Since some email gateways are uni-directional we hope we can get this to work.

It is assumed that the author is otherwise familiar with sending messages by e-mail. Therefore, the following instructions are limited to specific requirements for submitting an abstract to the IRGCVD.

PLEASE ENSURE that the Subject entry identifies the submitted abstract as being for the IRGCVD Symposium or the joint IRGCVD-AIC meeting. The local organiser is also involved with submissions for the AIC meeting in Sydney in June 1991 and so it must be quite clear for which event your abstract is intended.

1. The abstract should be submitted as a text message containing only printable ASCII characters. NO FORMATTING CODES, SPECIAL CONTROL CHARACTERS, OR OTHER NON-PRINTABLE CHARACTERS ARE TO BE INCLUDED!!

   If you are at all uncertain about meeting these requirements, the best test is to send theabstract as an e-mail message to yourself first to see if it is transmitted and received satisfactorily.

2. Each line of your message should contain at most 80 characters, including spaces, and should be terminated with a carriage-return, line-feed.

3. A complete submission will contain 9 items, each of which is introduced by a key word enclosed in angle brackets. The sequence of these key words is <TOPIC> <PRESENTATION> <TITLE> <AUTHORS> <ABSTRACT> <EQUIPMENT> <ADDRESS> <E-MAIL> < >

4. The information typed after each key word is as follows:

   <TOPIC> Type the selected topic area title as in the printed form.

   <PRESENTATION> Type a two letter code, in uppercase, according to your presentation preference. The code is: LO (lecture only), PO (poster only), LP (lecture first, then poster), PL (poster first, then lecture).

   <TITLE> Enter title in CAPITAL LETTERS

   <AUTHORS> Enter the authors' names and any academic or company affiliations, with the presenting author marked by an asterix.

   <ABSTRACT> Enter a one-paragraph, single spaced abstract of about 200 words.

   <EQUIPMENT> Describe any special equipment required for your presentation like video cassette player, tape player, etc.

   <ADDRESS> Give full mailing address of communicating author.

   <E-MAIL> Give e-mail address of communicating author if appropriate.

   < > Marks the end of your message.

5. Remember that e-mail messages are automatically time-stamped when received, so be sure to submit your abstract before the 15 February deadline. The time required for delivery of e-mail can vary from a few minutes to a day or more, depending on network traffic, routing and other factors.

6. An example is given below of an e-mail session in which an abstract is submitted. The initial dialogue for addressing the message will vary with your particular connection to the network and host computer.

SPECIMEN E-MAIL

Mailsend

To: sdain@usage.csd.unsw.au.oz
Subj: IRGCVD 1991 Abstract Submission

<TOPIC> Occupational consequences of colour vision deficiencies
<PRESENTATION> LO
<TITLE> ELECTRONIC SUBMISSION OF ABSTRACTS
<AUTHORS> Fred E Flintstone, Department of Neolithic Anachronisms, American University, Hollywood, CA.
<ABSTRACT> This is an example of how an abstract would appear when submitted by email. The abstract must consist of a single paragraph, single spaced about 200 words total. Avoid the use of obscure abbreviations or acronyms, literature citations, and references to figures or tables. A good abstract will state (1) the purpose of the study, (2) the experimental design, subjects and procedures, (3) the major results, and (4) the principal conclusions.
<EQUIPMENT> IBM-compatible computer, electrical outlet
<ADDRESS> Barney J Miller, School of Indecision, Podunk University, Anytown, NSW 2999.
<E-MAIL> bitnet:FRED@PODUNK
< >

SYMPOSIUM OF THE INTERNATIONAL RESEARCH GROUP ON COLOUR VISION DEFICIENCIES

June 20 - 23 1991

JOINT IRGCVD AND AIC MEETING

June 24 1991

REGISTRATION AND ACCOMMODATION BOOKING FORM

REGISTRATION

NAME Prof/Dr/Mr/Mrs/Miss/Ms .....

ADDRESS .....

.....

.....

.....

REGISTRATION FEES

			(before 15 May 1991)	
Participant*		$AUS420	($AUS340)	$ .....
Non-member Supplement		$AUS 36	----------	$ .....
Student (before May 15, 1991)		--------	($AUS180)	$ .....
Proceedings Volume (Non-member, Student)		$AUS 96	--------	$ .....
Accompanying person(s)	Name(s)			
.....	@	$AUS360	($AUS275)	$.....
JOINT IRGCVD-AIC meeting 24th June		$AUS 50	--------	$ .....

* Full members who are in good standing for the years 1990 and 1991 will receive a copy of the proceedings automatically.

ACCOMMODATION

Arrival Date ..... Departure Date ..... Total number of nights .....

MOTEL

SINGLE/DOUBLE/TRIPLE ..... Indicate requirement

PREFERENCE 1 .....

See accompanying form 2 .....

3 .....

One night's deposit ..... $ .....

COLLEGE

PREFERENCE 1 .....

See accompanying form 2 .....

3 .....

One night's deposit ..... $ .....

TOTAL ..... $ .....

See page 7 for payment instructions

For all communications, please mail or fax to: IRGCVD SYMPOSIUM SECRETARIAT School of Optometry, University of New South Wales, Kensington, New South Wales 2033, Australia

Phone: Int + 61 2 697 4629.Fax: Int + 61 2 313 6243.Telex: AA26054
e-mail:sdain%usage.csd.unsw.oz.au@murtoa.cs.mu.oz

XIth SYMPOSIUM OF THE IRGCVD

SYDNEY 20 - 23 JUNE 1991

ABSTRACT FORM **Due for submission by 15th February 1991. See Instructions.**

1 Please tick the topic area:

- ☐ Molecular genetics of colour vision deficiencies
- ☐ Occupational consequences of colour vision deficiencies
- ☐ Spatial aspects of colour vision
- ☐ Other

2 Please tick your preference for one of the following forms of presentation:

- ☐ Prefer a poster, if not then a paper
- ☐ Prefer a paper, if not then a poster
- ☐ Prefer a paper only
- ☐ Prefer a poster only

3 Title of presentation IN CAPITAL LETTERS

4 Authors' names and affiliations. Put asterix against presenting author, who must be an IRGCVD member.

5 Abstract

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