RESEARCH REPORT

Lighting and Human Health: A Review of The Literature

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LIGHTING AND HUMAN HEALTH

A REVIEW OF THE LITERATURE

Prepared for:

CANADA MORTGAGE AND HOUSING CORPORATION
700 MONTREAL ROAD
OTTAWA, ONTARIO
K1A 0P7

CMHC Project Manager:

VIRGINIA SALARES

Submitted by:

B.RUSAK, G.A. ESKES, S.R. SHAW

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AUTHORS OF THE REPORT

Benjamin Rusak

Dr. Rusak received a Ph.D. in Psychology from the University of California, Berkeley (USA) in 1975. He is currently a Professor in the Departments of Psychology and Pharmacology and a member of the Institute for Neuroscience at Dalhousie University, Halifax, Nova Scotia. His primary research interests are in the effects of light on mammalian circadian rhythms and the neural mechanisms regulating biological rhythms. His research is supported by grants and contracts from NSERC and MRC of Canada, the U.S. Air Force Office of Scientific Research and Servier, Canada.

Gail A. Eskes

Dr. Eskes received a Ph.D. in Psychology from the University of California, Berkeley (USA) in 1981. She is currently a Psychologist at the Victoria General Hospital (Queen Elizabeth II Health Sciences Centre) in Halifax, Nova Scotia, an Adjunct Professor in the Department of Psychology, a Lecturer in the Department of Psychiatry, and a member of the Institute for Neuroscience at Dalhousie University. Her research interests are in the effects of light on circadian rhythms, the treatment of Seasonal Affective Disorder with light therapy, and neural mechanisms underlying human cognition. Her research is supported by grants from NSERC of Canada.

Stephen R. Shaw

Dr. Shaw received a Ph.D. in visual physiology from the Department of Biology, University of St. Andrew's (Scotland) in 1968. He is currently a Professor in the Departments of Psychology and Biology and a member of the Institute for Neuroscience at Dalhousie University. His primary research interests are in various aspects of sensory physiology, including the neural mechanisms underlying vision and the anatomical and optical properties of eyes. His research is supported by grants from NSERC of Canada.

FOREWORD

This review is the first of a collection of studies which are intended to improve our understanding of the effects of lighting on human health. This review summarizes our current knowledge of the effects of light on people and assesses the implications of this knowledge for practices relating to illumination of homes through windows. This review deals with natural light, which is either diffuse light from the sky (daylight) or direct radiation from the sun (sunshine), rather than artificial light.

Due to the comprehensiveness of this review, it is published as a report by itself. It is presented in such a way that the reader can read the synopsis which precedes each section and then refer to the detailed review of that section.

The executive summary of this literature review and the other studies dealing with lighting and windows are published in the separate report - **Energy Efficient Windows, Lighting and Human Health**, CMHC, 1996.

Virginia Salares
Technical Policy & Research Division
Canada Mortgage & Housing Corporation
700 Montreal Road
Ottawa, Canada K1A 0P7

Tel. (613) 748-2032
FAX (613) 748-2402

AVANT-PROPOS

Cette revue est la première d'une collection d'études destinées à améliorer notre compréhension des effets de l'éclairage sur la santé humaine. La revue résume nos connaissances actuelles entourant les effets de la lumière sur les gens et évalue les conséquences de ces connaissances pour les pratiques se rapportant à l'éclairage des maisons par les fenêtres. La revue traite de la lumière naturelle, lumière diffuse du ciel (lumière du jour) ou du rayonnement soleil direct (ensoleillement) plutôt que de lumière artificielle.

Vu le caractère exhaustif de la revue, elle est rédigée comme un rapport proprement dit. Elle est présentée de manière à ce que le lecteur puisse prendre connaissance du résumé qui précède chaque section et se référer à la revue détaillée de la section voulue.

Le résumé de cette documentation et les autres études consacrées à l'éclairage et aux fenêtres font l'objet d'un rapport distinct, Fenêtres éconergétiques, éclairage et santé humaine.

Virginia Salares
Division des politiques et de la recherche en matière technique
Société canadienne d'hypothèques et de logement
700, chemin de Montréal
Ottawa, K1A 0P7
Canada

Téléphone : (613) 748-2032
Télécopieur : (613) 748-2402

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EXECUTIVE SUMMARY

A. How Light Affects Human Physiology

This review examines the effects of lighting on human health, with special emphasis on natural illumination and its transmission through window glass. Light in the environment affects human health via three routes. Two of these routes involve the eyes, while one involves the skin. First, light in the visible portion of the spectrum affects receptors in the eyes which convey this information to the brain. Parts of the brain analyse this information and give rise to the conscious awareness of light that we experience as *vision*. The second route by which light affects human physiology also starts in the eyes, but involves different parts of the brain. Light information conveyed by the second route has profound effects on our physiology, of which we are not consciously aware; these are the non-visual or *photic* effects of light. They include effects on mood, synchronization of our daily behavioural and physiological rhythms to the cycle of day and night, and regulation of patterns of hormone secretion. The third route involves the effects of light mediated by the skin, rather than the eyes. These include influences on the skin itself (for example, tanning and allergic responses of the skin to light), as well as important effects on general physiology, such as altering immune system function and stimulating the production of vitamin D.

B. Conclusions

1. The human visual system can adapt to a wide range of background illumination intensities and colours. We can, therefore, perform ordinary visual tasks (reading, writing, sewing, *etc*) at a great variety of light levels above a minimal threshold intensity.

2. Photic (non-visual) effects of light mediated by both the eyes and skin may not show similar adaptation to levels of background illumination. These effects may depend to a larger extent on the absolute light levels experienced. Reduced lighting intensities (such as those found indoors) that are compatible with normal, conscious visual function may not be adequate to meet the photic requirements for maintenance of normal daily rhythms, mood and arousal.

3. Light can alter states of physiological arousal, probably via the autonomic nervous system and by regulation of the hormone melatonin. The potency of these photic effects and the degree of variability among people in their sensitivities to them have not been studied extensively.

4. The use of tinted and coated window glasses can screen or filter some wavelengths of light as well as reducing general illumination levels. As a result, room occupants may respond by increasing the use of supplementary artificial lighting indoors. The potential physiological effects of the particular artificial systems available then become indirect consequences of window tinting.

5. Certain populations are at higher risk than others for experiencing inadequate lighting. Aging populations are particularly vulnerable because there is a dramatic reduction of light transmission through the eyes during normal aging, and with age-associated abnormalities of the eyes (e.g., cataracts, glaucoma, diabetic retinopathy). In addition, more sedentary lifestyles and chronic illnesses can further reduce natural light exposure for some seniors, particularly during winter months and in inner-city environments. Poor diets may further exacerbate the problem by providing inadequate dietary vitamin D levels. The re-emergence of rickets in some inner-city populations in North America, and evidence of bone demineralization in the institutionalized elderly (resulting from vitamin D deficiency) testifies to the need for more appropriate diets and light exposure in order to maintain adequate vitamin D levels.

6. The evidence that light therapy is an effective treatment for one significant form of clinical depression, seasonal affective disorder (SAD), indicates that light can have profound effects on mood at least in this substantial subset of the population. The characteristics that distinguish such light-sensitive people from others are unknown.

7. Field studies of the effects of different lighting systems, including so-called "full-spectrum" fluorescent lighting, altered spectral characteristics resulting from window tinting, and windowless environments are generally either lacking or so poorly conducted as to not permit any firm conclusions.

8. There are suggestions in the literature that artificial indoor lighting may contribute to the experience of some symptoms associated with "sick-building syndrome", although the issue has not been studied extensively.

C. Recommendations

1. Further research is needed on several aspects of the physiological effects of light on normal people. These include studies of physiological arousal caused by light exposure and of the effects of light on daily rhythms, the characteristics of effective light, and the underlying physiological mechanisms mediating the effects of light.

2. A great deal more information is needed about the range of variation in pattern and intensity of daily light exposure experienced by people in the home, the workplace and outdoors. The changes in exposure with season of the year and at different stages of life need to be investigated.

3. Populations at higher risk for inadequate exposure to light, including seniors and people who are institutionalized, require particular study and attention. More adequate light exposure and improved diets may be needed to maintain health in these populations.

4. Identification of sub-populations with unusual sensitivities to light exposure or to inadequate lighting should be pursued, along with investigations into the mechanisms underlying such sensitivities.

5. Appropriately designed, executed and analysed field studies of the effects of different lighting environments on human health and behaviour are needed in order to assess claims that have been made based on previous inadequate studies.

SCOPE OF THE REVIEW

The goal of this literature review is to summarize our current knowledge of the physiological and behavioural effects of light on people. The context of this review is the desire to assess the implications of our knowledge in this area for practices relating to illumination of homes through windows. Each subsection of the review is preceded by a short synopsis (in bold, italic type) which summarizes the main points of that section in non-technical language. Readers may wish to consult these synopses in order to assess which sections they will be interested to read in further detail.

The review first describes the physical characteristics of light reaching the earth from the sun and reaching the tissues of the human body. Issues discussed include the nature of the spectrum of electromagnetic energy we call light, the physical properties of light from different sources, the effects of glass acting as a light filter, and appropriate methods for measuring light intensity and spectral properties.

The second topic is the structure and function of the visual system, which mediates most of the effects of light on human physiology. This section describes the anatomy of the human eye, the light filtering properties of the eye media, and the nature of the retinal photoreceptors. This is followed by a review of the nature of colour vision and the physiological mechanisms which are responsible for our perceptions of colour. Two final sections address the central neural mechanisms underlying vision and the mechanisms that code for brightness and contrast sensitivity.

The third topic is the effects of light on physiological processes. These include both toxic and beneficial extraretinal effects of exposure to light of different wavelengths, as well as retinally mediated effects on circadian rhythms, pineal gland function and aspects of autonomic arousal.

The fourth topic is the effects of light on mental health. The major subject reviewed is the use of light in the treatment of seasonal affective disorder (SAD) and possible mechanisms underlying its efficacy. In addition, the possible effects of light on mood in forms of non-seasonal depression and on sleep disorders are reviewed.

The final topics reviewed relate to windows and illumination in the home and workplace. Subjects reviewed include attitudes toward windows and windowless environments, effects of window tinting, the influence of room colour and full-spectrum lighting sources on mood and health, and the interaction of aging with characteristics of illumination sources.

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SOMMAIRE

A. Les effets de la lumière sur la physiologie humaine

Le présent ouvrage traite des effets de l'éclairage sur la santé humaine, principalement de l'éclairage naturel et de sa transmission à travers le verre de fenêtres. La lumière présente dans l'environnement exerce des effets sur la santé humaine de trois façons : deux d'entre elles par le biais des yeux et, l'autre, de la peau. Pour la première de ces trois façons, les rayons de la partie visible du spectre touchent les récepteurs oculaires, qui transmettent cette information au cerveau. Certaines parties du cerveau analysent cette information et rendent la personne consciente de la lumière, produisant ce qu'on appelle la *vision*. La deuxième manière dont la lumière affecte la physiologie humaine passe également par les yeux, mais le traitement de l'information se fait par différentes parties du cerveau. L'information transmise de cette deuxième manière au sujet de la lumière affecte considérablement notre physiologie sans que nous nous en rendions compte; c'est ce qu'on désigne par effets non visuels ou *photiques* de la lumière. Il en résulte des modifications dans notre humeur, dans la synchronisation de nos rythmes comportementaux et physiologiques quotidiens liés au cycle du jour et de la nuit, et dans la régulation des régimes de sécrétion hormonale. La troisième façon fait intervenir la peau plutôt que les yeux comme médiateur. Il se produit alors des modifications de la peau elle-même (p. ex. le bronzage et les réactions allergiques de la peau à la lumière) ainsi que d'importantes conséquences sur la physiologie générale, notamment des changements dans le fonctionnement du système immunitaire et une production accrue de vitamine D.

B. Conclusions

1. Le système visuel humain peut s'adapter à une large gamme d'intensités et de couleurs dans l'éclairage ambiant. Par conséquent, nous pouvons exécuter nos tâches visuelles habituelles (lecture, écriture, couture, etc.) en nous accommodant d'un large spectre d'éclairage au-dessus d'un seuil d'intensité.

2. Les effets photiques (non visuels) de la lumière perçue par les yeux peuvent ne pas correspondre au niveau d'éclairage ambiant perçu par la peau. Ces effets peuvent dépendre davantage du niveau absolu d'éclairage perçu. Les moindres intensités de la lumière (p. ex. : celles que l'on perçoit à l'intérieur des habitations) qui ne sont pas compatibles avec les fonctions visuelles normales et conscientes peuvent ne pas convenir aux exigences photiques de préservation des rythmes quotidiens normaux, de l'humeur et de la stimulation.

3. La lumière peut modifier les états de stimulation physiologique, probablement par l'intermédiaire du système nerveux autonome et par la régulation de l'hormone appelée mélatonine. L'ampleur de ces effets photiques et le degré de variabilité dans la sensibilité des personnes à ces effets n'ont pas été étudiés en profondeur.

4. Le verre teinté ou réfléchissant utilisé pour les fenêtres peut filtrer ou bloquer les rayons d'une certaine longueur d'onde, ainsi que réduire les niveaux généraux d'éclairage. C'est pourquoi les occupants d'une pièce peuvent avoir la réaction d'augmenter l'éclairage artificiel. Les effets physiologiques potentiels des systèmes artificiels particuliers ainsi utilisés peuvent donc constituer des répercussions indirectes du verre teinté.

5. Certaines parties de la population courent un risque particulièrement élevé de manquer de lumière. Les aînés surtout sont vulnérables car le processus normal de vieillissement et les troubles oculaires qui lui sont associés (p. ex. : les cataractes, le glaucome et la rétinopathie diabétique) réduisent considérablement la transmission de la lumière par l'intermédiaire des yeux. En outre, les styles de vie plus sédentaires et les maladies chroniques peuvent réduire davantage l'exposition à la lumière naturelle chez certains aînés, surtout durant les mois d'hiver et dans les environnements urbains plus densément peuplés. Les régimes alimentaires déficients peuvent exacerber le problème par suite de niveaux inadéquats de vitamine D. Le retour du rachitisme dans certaines populations des centre-villes en Amérique du Nord et la déminéralisation osseuse constatée chez les aînés en établissement (par suite d'une carence en vitamine D) témoignent du besoin de régimes alimentaires et de périodes d'exposition à la lumière plus appropriés pour assurer des niveaux adéquats de vitamine D.

6. L'efficacité, déjà constatée, de la photothérapie dans le traitement d'une forme importante de dépression clinique, le trouble affectif saisonnier, indique les effets considérables que peut avoir la lumière sur l'humeur, du moins chez cette grande partie de la population. Toutefois, on ne sait pas encore quelles sont les caractéristiques distinctives des personnes sensibles à la lumière.

7. Les études empiriques concernant les effets de différents systèmes d'éclairage, notamment de l'éclairage fluorescent «lumière du jour», des caractéristiques spectrales modifiées par suite de la teinture du verre et des environnements sans fenêtre, sont habituellement déficientes ou si mal exécutées qu'elles ne permettent pas de tirer de fermes conclusions.

8. Dans les ouvrages publiés, on laisse croire que l'éclairage artificiel intérieur puisse contribuer à certains symptômes associés au «syndrome des bâtiments malsains», quoique la question n'ait pas été étudiée en profondeur.

C. Recommandations

1. Il faudra effectuer des recherches supplémentaires sur plusieurs aspects des effets physiologiques de l'éclairage sur les gens normaux. On devrait étudier particulièrement la stimulation physiologique résultant de l'exposition à la lumière, les effets de l'éclairage sur les rythmes quotidiens, les caractéristiques d'un éclairage efficace et les mécanismes physiologiques fondamentaux de médiation de la lumière.

2. Il est nécessaire de disposer d'une somme beaucoup plus considérable de renseignements concernant l'évantail d'habitudes et d'intensité de l'exposition quotidienne à la lumière, tant à domicile qu'au travail et dans l'environnement extérieur. Les changements dans l'exposition selon la saison et l'étape de la vie doivent aussi être étudiés.

3. Les populations qui courent un risque élevé d'exposition insuffisante à la lumière, notamment les aînés et les personnes en établissement, doivent faire l'objet d'attention et d'études particulières. Une exposition plus adéquate à la lumière et un régime alimentaire plus adapté pourraient être nécessaires pour préserver la santé de ces gens.

4. Il faudrait repérer les sous-populations affectées d'une sensibilité inhabituelle à la lumière ou susceptibles d'exposition insuffisante à la lumière, et en étudier les mécanismes fondamentaux.

5. Il faut entreprendre des études pratiques comprenant une conception, une exécution et une analyse adéquates des répercussions des différents environnements lumineux sur la santé et les comportements humains afin d'évaluer les assertions antérieures fondées sur des études inadéquates.

PORTÉE DE L'ÉTUDE

Cet examen bibliographique a pour but de résumer nos connaissances actuelles des effets physiologiques et comportementaux de la lumière chez les humains. Il s'insère dans un désir d'évaluer les implications de nos connaissances en la matière, notamment des pratiques d'éclairage des logements par les fenêtres. Chaque section de l'étude est précédée d'un bref résumé (en caractères gras et en italique) dans lequel on récapitule les points saillants de cette section en langage de tous les jours. Les lecteurs pourraient consulter ces résumés pour déterminer quelles sections de l'ouvrage ils désirent lire plus soigneusement.

Le premier sujet traite des caractéristiques physiques des rayons solaires qui parviennent à la Terre et touchent les tissus du corps humain. On y expose la nature du spectre de l'énergie électromagnétique appelée lumière, les caractéristiques physiques de la lumière provenant de différentes sources, l'action de filtre orthochromatique qu'exerce le verre et les méthodes appropriées de mesure de l'intensité et des caractéristiques spectrales de la lumière.

Le deuxième sujet constitue une étude de la structure et de la fonction du système visuel, qui assure la médiation de la plupart des effets de la lumière sur la physiologie humaine. On y décrit l'anatomie de l'oeil humain, les caractéristiques de filtre orthochromatique du médiateur qu'est l'oeil et la nature des photorécepteurs rétiniens. Cet exposé est suivi d'une étude de la nature de la chromatopsie et des mécanismes physiologiques responsables de nos perceptions de la couleur. Les deux dernières sections portent sur les mécanismes nerveux centraux qui servent de fondement à la vision, et sur les mécanismes de codage des perceptions d'intensité et de contraste.

Le troisième sujet traite des effets de la lumière sur les processus physiologiques. Ces processus comprennent les effets extrarétiniens, tant nocifs que bénéfiques, de l'exposition à la lumière de différentes longueurs d'ondes ainsi que les effets qui s'exercent par le biais de la rétine sur les rythmes circadiens, sur la fonction de la glande pinéale et sur les aspects de la stimulation autonome.

Le quatrième sujet concerne les effets de la lumière sur la santé mentale. On étudie principalement l'utilisation de la lumière pour traiter les troubles affectifs saisonniers et les mécanismes qui pourraient appuyer son efficacité. En outre, on examine les effets possibles de la lumière sur l'humeur, dont la dépression non saisonnière et les troubles du sommeil.

Les derniers sujets concernent les fenêtres et l'éclairage au domicile et au travail. Ces sujets comprennent notamment les attitudes concernant les environnements avec et sans fenêtres, les conséquences de la teinture du verre, les conséquences de la couleur de la pièce et des sources d'éclairage *en spectre continu+ sur l'humeur et la santé, ainsi que le rapport entre le vieillissement et les caractéristiques des sources d'éclairage.

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LIGHTING AND HUMAN HEALTH

I. INTRODUCTION

> *"...[T]he direct and obvious effects of light on the human visual system and the skin are well understood but the deeper effects on general physiological responses have barely been explored yet. It may well be that when they are, a different orientation will be given to lighting equipment and design."*(Boyce, 1981)

Electromagnetic radiation from the sun is the source of energy for most life processes on earth, as well as the only significant natural source of illumination. The physical features of the sun's radiation and its measurement are reviewed in Section II. This radiation interacts with human physiology via several routes, including both the eyes and other body tissues. The *retina* is a part of the eye that is specialized to detect a portion of the spectrum of electromagnetic radiation that we call light. The optic tracts originating in the retina convey information about light in the environment to a variety of targets in the brain. This information can be divided into two major types. First, reception of light energy in the retina initiates a cascade of well-studied information flow through the classical visual system of mammals. The primary optic tracts convey input to the lateral geniculate nuclei (LGN) of the thalamus, which projects in turn to the visual cortex. At each level, visual information is analysed and modified to yield the perceptual phenomena we experience as *vision*. These include the features of shape, colour, shading, movement, size, depth, and the associated percepts of objects, persons and events; this topic is reviewed in Section III.

Second, retinal photoreception initiates patterns of activation in other parts of the visual system which are not typically experienced as visual percepts, but which can have profound effects on our physiology and behaviour. These are referred to as *non-visual* or *photic* effects of light to distinguish them from the processes usually considered to comprise vision. Some retinal projections break away from the optic tracts to terminate in visual centres of the brain which regulate reflex eye movements, of which are we are not consciously aware. These structures include the pretectal area of the midbrain, which uses light information to regulate the size of the pupils of the eyes, thereby controlling light entry. Other retinal projections reach the superior colliculus nearby, and form part of the input to circuits that initiate rapid, reflexive eye and neck movements, allowing objects to be tracked visually. A smaller but important projection reaches the suprachiasmatic nucleus of the hypothalamus, and is concerned with regulating daily and seasonal biological rhythms of bodily activity, as well as influencing motivational states. This projection and its role are discussed in section IV.C.

A third route for light effects on human physiology is via the skin. Although clothing, hair and skin pigmentation reduce the degree of exposure of tissues other than the eyes to light, exposed parts of the body are affected both on the surface and in depth. Light radiation can penetrate to surprising depths through exposed skin surfaces; one study documented considerable penetration of light through a skull as thick as that of a sheep (Ganong *et al,* 1962). An important question is what targets are reached by light falling outside the eyes and what physiological effects it can exert on those targets; this topic is reviewed in Section IV.B.

II. LIGHT AND LIGHTING

A usefully broad introduction to this topic and its definitions is found in the IES Lighting Handbook (1981), while the standard detailed reference source on colour and many other topics is still Wyszecki and Stiles (1982).

A. Spectral Characteristics of Light

1. The spectrum of light

Synopsis: Electromagnetic energy from the sun reaches the earth in a filtered form that still contains a broad range of wavelengths. These include high-energy wavelengths that are not visible to us (the ultraviolet (UV) range), medium-energy radiation that forms the visible spectrum, and low-energy radiation in the infrared range that is experienced as heat.

Like any hot body, the sun radiates energy into space as electromagnetic radiation, the broad range of which forms the electromagnetic spectrum. The radiation is best thought of as wave-like as it travels at the speed of light towards the Earth. In biology and disciplines related to the human environment, the different points along the spectrum are usually characterized according to the *wavelength* of the radiation; i.e., the distance in meters between successive peaks in the propagating wave. Its *frequency* is preferred in other sciences and is equally definitive for the present purpose: the product frequency (n) times wavelength (l) is a constant in a given medium (usually air); namely, the velocity of light, C ($C = n\,l$). In a different medium like glass, C and l vary while n remains constant. Certain wavelengths in the original spectrum are attenuated by selective filtering during transmission through the atmosphere. Of what remains at the Earth's surface, largely in the range 290-2500 nm (nanometres, billionths of a meter), a smaller range can be detected by humans via the eyes and visual system as light. This wavelength band, the *visible spectrum*, extends from about 380 to 700 nm and gives rise to corresponding sensations ranging from violet to deep red, respectively, when narrower bands of wavelengths within this range are isolated.

The specialized visual pigments in the photoreceptors of the eye (see section III.C.3) become progressively much less efficient at catching the longer wavelengths, so the range above about 700 nm (the *infrared* or IR spectrum) cannot be seen, although there is no sudden cut-off. Some IR radiation is absorbed as heat increasing molecular motion in skin pigmentation, raising its temperature. If the energy dose is sufficiently large, this warming can be detected by temperature-sensitive nerve endings in the skin. This effect is enhanced by certain snakes to "see" IR radiation from warm-blooded prey, but there is no comparably sensitive human ability.

At shorter wavelengths in the near *ultraviolet* (UV) spectrum below about 380 nm, all human visual pigments again have started to lose their absorbing power, while the lens and ocular media of the eye progressively absorb UV, stopping much of it from reaching the retina. Short wavelengths also cannot normally be seen, although some animal groups with smaller eyes (insects, some birds) do use part of the UV spectrum, and aphakic (lacking a lens) human patients from whom a lens has been removed surgically also can sense wavelengths shorter than normal. When light radiation interacts with matter it can be more usefully considered to consist of packets of energy called photons (or, interchangeably, *quanta*), where the energy of each photon assumes an exact value that is inversely related to the associated wavelength (Abrahamson

and Japar, 1972; Mauzerall, 1972). Short-wave UV photons have individual energies that are large enough to rupture otherwise stable chemical bonds at the site of absorption in the skin, making prolonged doses of UV radiation potentially dangerous, capable of damaging the DNA of skin cells and of causing skin cancers (see IV.A.1). For simplicity, the ultraviolet spectrum is sometimes sub-divided arbitrarily into the ranges UVA (315-400 nm), UVB (280-315 nm) and UVC (100-280 nm). A great deal more is known about the physiological effects of wavelengths in the visible range than of those in the UV and infrared ranges.

2. Wavelength, "colour" and brightness

Synopsis: Colour is a sensation experienced as the result of activation of certain classes of retinal photoreceptors by selected wavelengths of the visible light spectrum at daylight intensities. The wavelengths of light reflected from a surface also influence our perceptions of brightness.

In conditions of normal interior lighting or daylight viewing, the so-called *photopic* range, the cone system of the retina dominates our vision and the *scotopic* (night-time) rod system of the eye is inoperative (see Figure 4A). Using the cones and the neural network to which they connect in the retina and then the brain, humans can distinguish between different wavelengths of light. Each wavelength range produces a different effect along a continuum that results in the most vivid of our senses, that of colour. Strictly, *colour* should refer to the sensation, while *wavelength* is reserved for the corresponding physical length measurement, but often the first is used loosely as a graphic short-hand to describe the second ("red end of the spectrum", "blue cones", instead of more properly "long wavelength part of the visible spectrum", "cone type sensitive to short wavelengths near 430 nm").

The apparent brightness of surfaces is partly dependent on the wavelength composition of the reflected light, as well as the absolute level of illumination. The photopic luminosity function V(l) describes the relative apparent brightness associated with individual wavelengths, in a way thought to reflect the relative contributions of the three cone systems, in which the blue system is relatively weak (see III.C.3, Figure 4).

B. Characteristics of Light Sources

1. Blackbody radiators and incandescent sources

Synopsis: A tungsten filament lamp can be characterized with reference to its "colour temperature", a value which describes its emission spectrum in terms of its resemblance to an idealized equivalent blackbody radiator. A higher colour temperature describes a light source with its energy distribution shifted more toward the blue end of the spectrum.

When a solid is heated, the energy emitted as a function of wavelength depends upon both the material being heated and on the temperature attained, but an "ideal" radiator is material-independent and its emission can be defined by knowing just one variable, the absolute temperature. Such a spectrum is often called full or Planckian (after Planck who derived a formula for it), or most often, *blackbody*, from the simplest ideal that absorbs all incident radiation. Blackbody radiation is of conceptual use in illumination engineering because some light sources like tungsten filament lamps closely approximate a true blackbody radiator held at a slightly different (lower) temperature: specifying the *colour temperature* is then accurate enough to describe the spectral emission of the lamp by reference to the temperature of the equivalent blackbody. Practical values of blackbody radiation can be got either from Planck's equation itself, or from compiled tables for the visible range (Moon, 1948); see Table 1.6 in Wyszecki and Stiles (1982). For example, the common household tungsten filament lamp powered normally will approximate a blackbody held at about 2000-3400°K (degrees Kelvin or Absolute): its output rises gradually with wavelength throughout the visible region, reaching a maximum in the IR at about 1500 nm if run at 2000°K, falling smoothly after that (Jenkins and White, 1976). The rising output towards the red of these lamps imparts a yellowish cast to objects illuminated by them compared to average daylight, and becomes even more orange if a dimmer circuit is used. Thus colour photographs taken indoors in tungsten light but using outdoor "daylight film", appear too yellow, although we compensate perceptually for this (*colour constancy*; see IV.D.2).

If the lamp's filament temperature is made to rise (by changing the working voltage or its length or diameter), the peak of the blackbody-like radiation shifts to progressively shorter wavelengths, so the illumination appears not only brighter but also more blue. Tungsten evaporates much more quickly from a very hot filament, thereby shortening filament life, causing tungsten to condense on the glass and darkening it. Tungsten-iodine bulbs are made to operate at the higher temperatures, because the evaporated tungsten forms an iodide that is recycled back to the filament, extending its life and reducing deposition on the quartz envelope. These bulbs are commonly used in automobile headlamps, in some projector lamp units, and occasionally in desklamps, but not usually otherwise in interior lighting.

2. Fluorescent sources

Synopsis: Fluorescent light sources emit both a continuous light spectrum and sharp spectral lines. Their spectra can be characterized by their correlated colour temperatures. Fluorescent light typically appears whiter than yellowish incandescent sources and can be modified to emphasize selected ranges of wavelengths.

The idealized form of light emission from a blackbody represents a smooth change of energy output with wavelength, a *continuous spectrum*. In addition to this, when individual atoms in materials become sufficiently excited at high temperature in the gas phase, they emit large amounts of energy at signature wavelengths, some in the visible range, forming their characteristic *line spectra*. Practical examples are powerful arc discharge lamps used in sports fields and in movie theatres (xenon, white), and gas discharge lamps used for some highway lighting (sodium, orange-red; mercury-vapour, green). Mercury-vapour lamps are also used in their modified low-pressure household form as fluorescent tubes, where an inorganic phosphor coated on the inside of the glass envelope captures the otherwise useless and potentially dangerous major mercury line in the UV (253.7 nm) and re-emits this energy as light in the visible range of the spectrum. Overall, the resultant output is more spectrally balanced in the visible range than that of a tungsten filament, although added to this are the large energy spikes in the visible range, from four mercury spectral lines at 405, 436, 546 and 578 nm. These fluorescent sources do not resemble a radiating blackbody at all closely, but a computational procedure has been established to define the colour temperature of the nearest equivalently coloured blackbody, its *correlated colour temperature.*

Most types of fluorescent tube appear whiter than tungsten lamps, and although they are more expensive initially per watt, they are four times more efficient converters of electrical power and have long lives, so they are more economical. The output spectrum can be modified somewhat in manufacture by choosing different phosphors or mixtures, to make the output appear "cooler", "warmer" or "softer" as these variants are described commercially. The spectra of these basic types of tubes (Figure 1A) are illustrated in Table 1.12 and Fig. 1.16 in Wyszecki and Stiles (1982). Other modifications are occasionally used, e.g. for stimulating plant growth, and for UV-sterilization in hospital units (UV output with no phosphor coating). A minor but controversial, expensive variant is sometimes misleadingly termed "full spectrum" (Figure 1B). It has a phosphor mix that has a lower efficiency and a relatively poor green-yellow emission, but therefore possesses a higher relative blue emission and a higher correlated colour temperature, and, in at least one brand, enhanced UV emission. Claims have been made for its superiority in indoor lighting, but these are so far poorly founded, based on inappropriately controlled tests (see VI.C.2).

Both tungsten and fluorescent lamps are nearly always driven directly from the household AC electricity supply, which in North America has the current reverse direction at 60 Hz (60 times per second). Each tungsten filament therefore emits a fluctuating light output at 60 Hz, but the thermal inertia of the filament coil itself damps this modulation to only a few percent of the time-averaged output. This in combination with the photopic human visual system's increasing insensitivity as the flicker frequency rises above about 40 Hz, means that the fluctuations are not visible. The situation is much worse for fluorescent strip lights for which

the optical output modulates deeply within one cycle, but these emit at double the frequency (120 Hz) where human sensitivity to flicker is even lower.

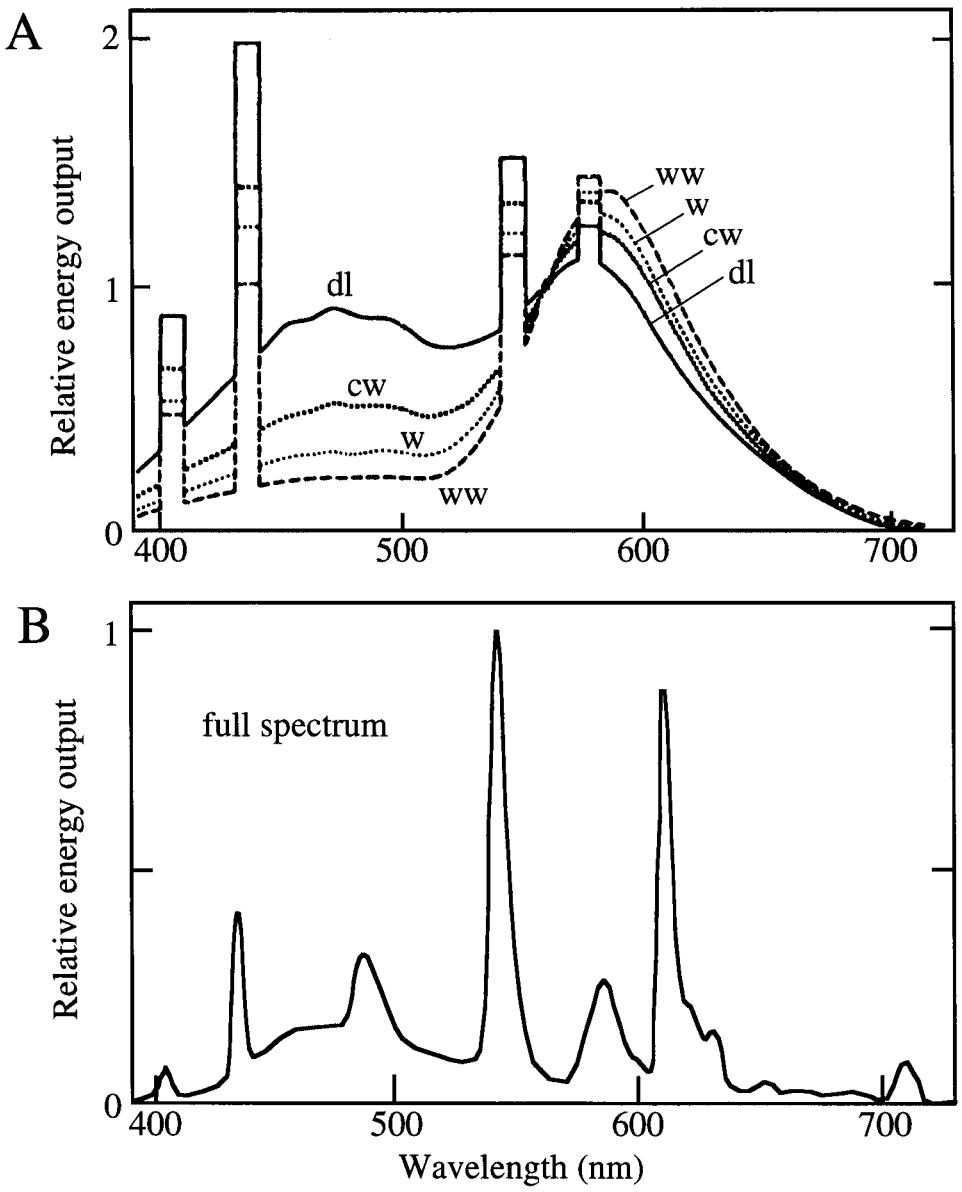

Fig. 1. *A*. Relative spectral energy distributions of four types of fluorescent tube, warm white (ww), white (w), cool white (cw) and daylight (dl). The curves were sampled over intervals of 10 nm and normalized at 560 nm (after Wyszecki and Stiles, 1982). *B*. Output of a full spectrum lamp, sampled at finer intervals and normalized at the highest peak of the line spectrum (redrawn from data of Boyce, 1994)

3. Sunlight and skylight

Synopsis: The spectrum of sunlight is modified by the earth's atmosphere, and skylight is characterized by added short wavelengths. Polarizing artificial light to mimic the natural polarization of skylight, especially in the UV range, is probably of no value since the human eye cannot detect the polarization of light.

The sun's spectrum above the atmosphere is a fair approximation to a blackbody with a colour temperature of about 6200°K. Added inflections particularly in the blue region from elemental line spectra in the sun, giving it a maximum energy output at about 470 nm (blue-green). Extensive tabulated values are given by Moon (1940). At sea level, the radiant flux that arrives is reduced throughout the spectrum by attenuation within the atmosphere, due to scattering and absorption. The effects depend upon the elevation of the sun and the consequent path length through the atmosphere, the so-called air mass m defined as a multiple of the path length with sun directly overhead (m=1). The standard path length used by the IES is m=2, equivalent to the sun at 60° from the zenith (Wyszecki and Stiles, 1982). Attenuation becomes more pronounced at shorter wavelengths where atmospheric scattering by gases including ozone and inclusions (water vapour, dust) becomes more prominent, i.e., illumination at ground level appears red-shifted. Water vapour and oxygen introduce prominent absorption bands at long wavelengths and into the infrared.

As shorter wavelengths are selectively scattered out of the direct path from the sun (mostly Rayleigh scattering, inversely proportional to the fourth power of the wavelength) the sky lateral to this direct line benefits, and some of this short-wave-enhanced light then re-scatters down to reach us combined as daylight. Skylight therefore appears much bluer than the sun, does not resemble blackbody emission, and has an even higher correlated colour temperature than the sun. The actual spectral content in skylight varies greatly with the sun's elevation and atmospheric conditions. To exemplify this range, the output has been tabulated for five representative phases of daylight with correlated colour temperatures from 4,800 to 10,000°K (Judd *et al,* 1964). The luminance of the sun's disk and the sky differ enormously, by a factor of about 300,000:1. Although "sunlight" (above the earth's atmosphere) and "daylight" (filtered sunlight reaching the earth's surface) are different, the term "sunlight" is used in much of the scientific and popular literature to refer to outdoor illumination on the earth's surface, and this term is used in this report.

Parts of the sky are weakly plane-polarized in a rather elaborate pattern that varies across the sky and with time of day, since it relates to the particular position of the sun. Polarization is strongest in the direction 180° away from the sun, at shorter wavelengths into the UV. It has long been known that insects can analyse the plane of polarization and that bees sometimes use it as a sky compass when the sun is overcast. Among vertebrates, a few fish and birds have since been found that are sensitive, but the disposition of the visual pigment in the fluid lipids of the rods and cones of humans is such that we cannot usefully resolve the plane of polarization unaided. Partly on the grounds that it is supposedly "natural", there has been advocacy for use of expensive so-called "full-spectrum" fluorescent tubes in luminaires that are designed to polarize the light output down into the near UV (Karpen, 1994). This trend appears to be without sound scientific foundation since the eye's lens removes nearly all UV, while skylight overall is for the most part not strongly polarized, and humans cannot normally detect

the plane of polarization anyway. Some improvement in readability of text on shiny paper held at a particular angle of incidence might be expected in polarized light (analogous to removal of road glare), but would likely be offset by reduced efficiency of the "full-spectrum" source, compounded by the light losses inherent in polarizing its output. Re-adjusting the tilt of the page under standard lighting would be a more intelligent solution.

4. Effects of glass windows and optical filters

Synopsis: Ordinary glass windows have little effect on wavelengths in the visible spectrum or the infrared range, while they exclude UV wavelengths below 380 nm rather sharply. Elements in the human eye also act as selective wavelength filters. Measuring the filter properties of window glass accurately requires careful attention to the angles at which measured light reaches the glass.

Light sources are often modified by interposed filters, including windows. Thin panes of common glass in windows and in the envelopes of lamps attenuate a small amount rather uniformly across the visible spectrum, depending on the thickness. They therefore modify the spectrum of sunlight, skylight and lamp light very little in this range. They start to absorb ultraviolet significantly below about 380 nm, however, and increasingly attenuate this radiation towards shorter wavelengths. As light enters through the windows of a house, the glass acts as a rather sharp "cut-on" filter because of attenuation of the short wavelengths, compared to the very gradual effect at the other end of the spectrum. Most untinted glasses transmit infrared well, out to wavelengths of a few micrometers, and do not cut off suddenly. Although this IR radiation is invisible, it can contribute significantly to the heat balance of a dwelling, especially where this is a deliberate design feature.

Filter transmission characteristics are most easily measured from UV to near IR, on pieces of the filter placed normal to the measuring beam in an automated commercial spectrophotometer, usually relative to a comparison beam passing through air. The measuring beams are collimated (parallel ray paths), so the result is valid for light incident at right angles (normal) to the glass surface. Certain types of thin-film elements in optical filters do not behave simply as the angle of incidence is varied, because of optical interference, and much of the radiation reaching windows arrives at non-normal incidence. It is important to make sure that this is not a complicating factor when assessing new materials like coated glasses, by running spectrophotometric checks at different angles of incidence, or by using a different measuring system incorporating a good (Lambertian) diffuser before the glass, to allow integration across all practically significant angles of incidence.

Filter transmission characteristics are sometimes given using a linear scale of the *transmittance* T (percent transmission, relative to the measurement in air with no filter present) of the filter as a function of wavelength, λ (T_λ). Sometimes transmittance is given normalized. More usually a logarithmic scale of *optical density* (OD_λ) against wavelength is employed, sometimes called *absorbance*, where

$$OD_\lambda = \log_{10} (100/T_\lambda)$$

The OD scale is practical to use because the overall OD of several filters in series is simply the sum of the individual densities at the wavelength in question, while transmittances are multiplicative and so less intuitive. A logarithmic scale also gives a much more readable print-

out for examining blocking zones where a filter transmits below a few percent of the incident light, which will plot unusefully close to zero on a linear spectrophotometer scale; some filter manufacturers even use a double-log scale for this reason. Optical elements in the eye, notably the lens, macular pigment and visual pigment all behave in some respects like optical filters, and so their transmission characteristics also are often expressed conventionally in units of optical density (often abbreviated to "density").

C. Measurement of Light

1. Energy or quanta? Radiometric and photometric units

Synopsis: There are two commonly used systems for measuring light intensity. The radiometric system measures the energy of the light without reference to human or any other species' sensitivity to the light. The photometric system relates light measurement to the relative effectiveness of light of different wavelengths for the human eye at daylight intensities. Illuminance is the photometric measure, expressed in lux, which is used to describe lighting levels in the home and workplace. In general, people prefer illumination sources with a higher colour temperature at higher light intensities. This preference may have implications for lighting choices in situations where window coatings alter both the intensity and spectral properties of light entering a room.

The results above are conventionally measured and quoted in *radiometric* units. These would be derived originally from some measuring device like a thermopile that is blackened to make it absorb different wavelengths uniformly, and that has been calibrated indirectly with reference to the original internationally recognized standard. Most modern instruments use a more sensitive silicon photodiode that is not spectrally flat, but is either calibrated by wavelength or preceded by a compensating optical filter. The most sensitive devices at short wavelengths are still photomultiplier devices. The result from each is an electrical output that can be interpreted as radiant energy delivered over a time interval, and over some area or solid angle, for example, Watt meter^{-2}, identical to Joule sec^{-1} m^{-2}. There is great variation in spectral output between the different sources above and even some between lamps of similar manufacture, so the *total* energy count does not necessarily provide useful information about the stimulating power for human vision. The energy of light sources is therefore most useful when tabulated per unit wavelength interval, usually in bands 10 nm wide.

Photoreception in animals, including humans, is not a direct energy-absorbing process like that in a thermopile, which causes increased thermal motion of atoms. Instead it depends upon the capture of individual photons (quanta) by single visual pigment molecules in the cones. Each capture alters the electronic structure leading to a change in conformation of this protein molecule, and this starts the visual process (Abrahamson and Japar, 1972); see III.B.2. Therefore, to estimate the relative effectiveness of radiation at two different wavelengths, what is needed to express stimulating power is not the *relative energy* but the *relative numbers of photons*. These are simply related because the energy delivered when one photon is captured is $E = hC/l$, where h is Planck's constant, so a spectral energy scale can be converted into a relative numbers-of-photons scale simply by dividing by the associated wavelength. A source emitting equal energy at 380 nm and 760 nm, for example, would contain twice the number of photons

at 760 nm. Most visual studies since the 1960s when this became widely appreciated in vision quote results on a quantal absorption basis, not an energy basis. In addition, visual pigments absorb fairly selectively at different wavelengths. In estimating the effectiveness of different wavelengths of illumination in humans, the spectral sensitivity of the particular photoreceptor subsystem has to be taken into account (see III.C.3).

It is also possible to construct a measuring system based (originally) on human visual comparisons made relative to some viewed standard source, usually "white" (originally a standard tallow candle, or its light reflected from a surface). This has generated another complicated and parallel system of *photometric* units for comparing the brightness of sources and surfaces. It is important to recognize that these units do not translate directly one-for-one into the *radiometric* (energy based) units, since they necessarily incorporate the human photopic spectral sensitivity function V(l) in the result (Figure 4A), itself a non-simple byproduct of the responsivities of different cone types, and somewhat variable between people. It is possible but tedious to convert one type of unit into the other, if the spectral sensitivity function is known individually, or can be taken from tabulated representative values. Photometric units are not directly useful in most animal studies, where the spectral sensitivity function is usually different from that of humans. Radiometric units are "objective" in the sense of not depending upon human or animal sensitivities, so these have come to be the units used in careful quantitative work on vision. Both the IES Lighting Handbook (IES lighting handbook, 1981) and Wyszecki and Stiles (1982) have extended sections on photometry.

People pursue their various activities under a wide range of lighting conditions, from almost complete darkness to tropical sunlight at noon. More restrictively, we can ask what illumination level is adequate or optimal, which in turn depends on the tasks to be performed. Optimal lighting ranges have been defined for a wide variety of tasks outdoors, in the workplace and in the home. These are categorized from lowest to highest on a scale from A through H, or are tabulated numerically as recommended illuminances (IES lighting handbook, 1981). *Illuminance* is a measure of the illumination delivered to a surface, a photometric quantity that expresses the concentration of luminous flux incident on the surface, in units of *lux*. In the home, recommended illuminances fall mostly in the categories C through F, thereby varying by a factor of about 20, depending on the nature of the critical seeing task. The lowest recommended residential range involving any visual discrimination task would be about 100 lux at a dinner table surface, increasing to 2000 lux locally for fine sewing on a dark fabric surface. Safe illuminances for outdoor activities extend from 5-20 lux at the lower end for visually non-taxing activities like gardening, up to 500-1000 lux for some fast action sports. Visual contrast sensitivity improves as illuminance increases, and much higher illuminances are recommended, such as 10,000-30,000 lux, for critical industrial inspection tasks involving fine detail, or for exacting surgical operations in hospitals.

In artificial light when choices are offered, observers indicate a preference for an increased colour temperature of the lighting as average illuminance increases. At each illuminance, source colour temperatures below each preferred optimum were perceived as cold and drab, while those above the optimum were felt to render objects overly colourful and distracting. This could have implications for the inhabitants' long-term perception of well-being in buildings or houses fitted with energy-efficient coated window glass, if the glass type significantly changes the perceived spectral composition of room light. The opposite result has

been found where exacting tasks of colour selection must be performed in textile manufacture, however. At higher illuminances, somewhat lower colour temperatures are preferred for accurate colour matching. In the home setting, an important factor in the success of the local colour schemes has less to do with the lighting, but depends more on the presence of high contrast between the background decor and the colour of furniture objects in the room (IES lighting handbook, 1981).

2. Stimulus characteristics of light

Synopsis: Natural light is broad-spectrum radiation, as is most artificial light, but none provides illumination that is completely even across the spectrum ("spectrally flat") as ideal white light would be. Nevertheless, these sources are referred to loosely as "white". Light can be filtered in various ways to generate narrow bandwidth illumination that is described as "monochromatic" and has the appearance of a particular, relatively pure colour. Comparisons across experiments require detailed descriptions of light sources and their filtering.

Except for isolated cases arising from the optical interference of light or self-luminous objects (e.g., reflections from blue butterfly wings, firefly luminescence), light from naturally illuminated objects hardly ever arrives at the human eye as a narrow band of wavelengths, but rather as broad-spectrum radiation. When illumination from a source with a full range of wavelengths is presented at the same intensity across the visible spectrum, it is described as *white*, but this term is seldom used in a precise way (it is impossible to make a practical source that is exactly spectrally "flat"; i.e., contains the same number of photons at each wavelength). Illumination from several common sources is called "white" and these will have different stimulating powers for the human according to the particular spectral distribution. Such "white" illumination is readily produced from tungsten bulbs and various brands of fluorescent strip light, and from certain arc lamps (xenon).

"White" light is often used in visual experiments where colour mechanisms are not being studied, but it is more difficult to quantify and calibrate: the variability makes it usually almost impossible for other investigators to replicate the stimulus exactly with confidence. Narrow-band *monochromatic light*, on the other hand, is more useful for analysing most visual mechanisms and optical systems because of the ease of interpreting the calibrations, and the easy replicability. "Monochromatic" light can be produced by a variety of optical elements such as diffraction gratings, interference filters, and lasers. In practical sources it is not a single wavelength but has a bell-shaped distribution, and is usually characterized by its *half-bandwidth*, the width of the spectral band in nanometres at 50% of the maximum transmission at the dominant wavelength. When one of these sources is incorporated into commercial optical equipment such as the spectrophotometer, the monochromatic output is often the most useful form of radiation for studying both visual systems and the transmission properties of light filters, like window glass. The results are then essentially device-independent. Conversely, in tests involving comparisons of the visual appearance of surfaces, a white source may be needed where the meaning of "white" is device-dependent, so the setup, calibration, brand-names and numbers of lamps should be described in as much detail as possible, to allow replication by others.

III. THE HUMAN VISUAL SYSTEM

A. Introduction to the Analysis of Vision

Synopsis: The classical human visual system is now usually thought of as comprising a number of analytic modules that work in parallel to evaluate different aspects of the visual world. Their output in the brain's cortex generates an internal, cognitive model of the world that is the basis of our visually guided behaviour and our visual perceptions. Among the many visual features analysed by this system, light intensity and colour are most relevant to the question of home and office illumination, and these are the focus of the following sections. The primary sources of our information on these topics are animal experimentation, clinical neuropsychology, human psychophysics and modern brain imaging methods.

Good introductory accounts of the human visual system are books by Rodieck (1973) and more recently, Dowling (1987).

Many scientists have come to view the visual system as a type of modelling computer. Marr (1982) and others have argued that the amount of incoming information encountered in processing tasks like analysing a surrounding scene in real time while walking through it, is so overwhelming in its volume and demands that it cannot possibly all be handled even by the 10^{11} neurons of the brain and their estimated 10^{17} connections. The visual system must work instead by using small subsets of incoming sensory information in many small, parallel computing modules to generate an optimized, object-based working model of the local surroundings. This model gets updated periodically as the local scene around us alters. The resulting best-running-estimate, computed model in the cortex of the brain comprises the world that we are actually aware of and use operationally to get around. The hardware involved is not at all like the digital computer environment with a single ultrafast central processor, but comprises billions of neural units and synapses operating in parallel with multiple redundancies, on a quite slow time scale of milliseconds or more per operation. While there is no rigorous demonstration that the brain actually is analogous to an autonomous computing device, most modern scientific work on the cortex assumes this.

It then makes sense to think of visual analysis as a computational task that can be parcelled out to different *parallel-processing subunits* (DeYeo and van Essen, 1988). These can deal "in parallel" with extraction of specific useful information about the scene, concerning motion, depth, colour, pattern, brightness and so on. This picture actually emerged originally largely from the opposite end, from analysis of the neuroanatomy and physiology of the cells that make up each supposed subsystem. Currently, as many as 72 distinct parallel-processing areas are believed to exist in the cortex (Mountcastle, 1994) and at least 34 of these are visual (van Essen *et al,* 1992). "Parallel" in this context is seldom used rigorously. It is often difficult to articulate at all because there is some hierarchical layering of modules; neural cross-connections between both equivalent and non-equivalent levels are common, including recurrent connections back to "lower" centres which are often as dense as the ascending connections (Zeki, 1993). There is burgeoning interest in parallel-processor systems as the wave of the future for semiconductor-based computers.

Only some of these parallel-processed tasks are likely to be important in considering changes in interior lighting. Changes in the spectral composition of room light will be signalled as changes in hue through the colour processing networks, and this is the most important area to consider. In addition, changes in the absolute level of lighting affects the systems that code for the relative brightness and darkness of objects in a room (the grey scale, usually called the scale of object contrast), so such changes need to be considered. Accordingly, colour and brightness are emphasized in what follows.

It is unethical to conduct invasive experiments on humans, to try to discover the details of how our nervous system codes wavelength into colour, for instance. Some animals can be trained to make comparisons between stimuli, and to respond if they detect a difference. Repeated failure to choose reliably between two different colours (balanced for brightness), for instance, then leads to the important conclusion that the two coloured stimuli may actually be indistinguishable to the trained animal. The minimum wavelength interval needed to allow colour discrimination across the spectrum has been revealed in this way, for instance. Species of Old-World monkey like the macaque perform very similarly to ourselves in such tests of colour discrimination, so it is concluded that they must have three-component colour vision almost identical to ours (trichromacy). Such monkeys therefore can be used as substitutes for humans, in studies where invasive methods are required to analyse visual mechanisms (Bowmaker, 1984), and such results are included below.

Useful information has been obtained from work directly on humans in certain areas. Fundamental research on colour-coding in the retina has been done on surplus human retinae obtained postmortem from eye banks, following prior pioneering work on other species (see III.C.3). Studies on deficits of function in surviving human patients who have sustained local brain damage (through head injury, stroke, etc.) have long been recognized as an important source of information that can illuminate normal brain function in the areas damaged (Kolb and Whishaw, 1985; Zeki, 1993). This is especially true of attempts to assess the functions of the many subdivisions now recognized in the "highest" levels of the brain (the cerebral cortex). Most recently, computer-assisted imaging techniques have given relatively non-invasive (but expensive) views of local brain malfunction (particularly MRI and PET scan). These methods can give dynamic information about normal brain function and disability (Zeki, 1993), although the spatial resolution is quite limited (about 2 mm). Finally, objective study of people in the type of visual test situation mentioned above for monkeys comprises a large research area (visual psychophysics) with a long history of success in uncovering the properties of the eye and brain. For example, the realization that a single photon caught by a rod is sufficient to start the whole process that leads to a conscious visual sensation in humans can now be examined in electrical and biochemical detail directly in single rods (see III.B.2), but was predicted explicitly much earlier by Hecht *et al* (1942), based on purely psychophysical measurements that they performed upon themselves.

B. Structure and Function of the Eye

1. Filtering by eye structures

Synopsis: As light enters the human eye, it is filtered by the eye's optical components. The lens at the front of the eye filters out shorter wavelengths, reducing blue light transmission and especially UV light. This filtering grows much stronger as the lens ages. Additionally, the central part of the retina (fovea), which is most important for human vision, is pigmented so that it further reduces transmission of blue light to the receptors. Light scatter in the eye reduces contrast between brightly and dimly illuminated regions of a visual scene, but the process of lateral inhibition in the retina can also sharpen weak local contrasts.

The normal optical path in the eye is shown schematically in Figure 2A. Most of the visible light that reaches the surface of the eye enters it; unwanted reflections from the exterior and interior optical surfaces, most notably at the cornea, account at most for 4 percent of the flux (Wyszecki and Stiles, 1982). The ocular media, the so-called aqueous and vitreous humors, are normally neutral to visible light, but two significant spectral filters are interposed in the light path. The *lens* has little effect on wavelengths in the yellow-red part of the spectrum, but below this, absorption by it rises monotonically towards short wavelengths. Transmission gets progressively but variably worse with age (Figure 3A), and by middle age lens density can exceed 0.6 at 400 nm and 2.0 at 380 nm (i.e., transmission is reduced by 75% and 99%, respectively). The lens thus accounts for most of the eye's UV losses, and this affects the whole retina. The second significant filter affects only the fovea and is formed by the *yellow spot* (macula lutea). This consists of a layer of tissue overlying the fovea where the retina is thinnest. It contains a blue-absorbing carotenoid, the density of which is quite variable but averages about 0.5 at 460 nm (Figure 3B); this seems not to vary with age. The selectivities of the two filters combine to reduce further the effectiveness of the blue cones, which are present at only about 10 percent of the number of the green and red cones (Williams, 1986). These effects account in part for the small blue representation in the photopic spectral sensitivity curve (Figure 4A).

The ocular media and internal eye surfaces scatter light so some of this now falls outside the intended image formed on the retina (entoptic scatter). Scatter is spectrally neutral, but is optically serious nonetheless. The intensity of the scatter falls away roughly as the inverse square of distance so it is more concentrated near the image, and amounts to about 10 percent overall even for a small source (Wyszecki and Stiles, 1982). A bright spot on a black background, for instance, can easily be made with a 1000:1 brightness ratio, but this high contrast cannot be preserved in image contrast inside the eye, where it is severely degraded by entoptic scatter. The resulting range of contrast on the retina for an extended image is never locally better than about 10:1 (Barlow, 1965). Retinal neural mechanisms can amplify small local contrasts, sharpening the degraded image (lateral inhibition). In most natural scenes, high object contrasts are not encountered anyway because the sky forms an extended source of illumination and the differences in reflectivity between most objects are modest. The contrast range available in any one visual scene is surprisingly low, <10:1 between the darkest and lightest extremes, and mostly much less (Laughlin, 1989; Srinivasan *et al,* 1982).

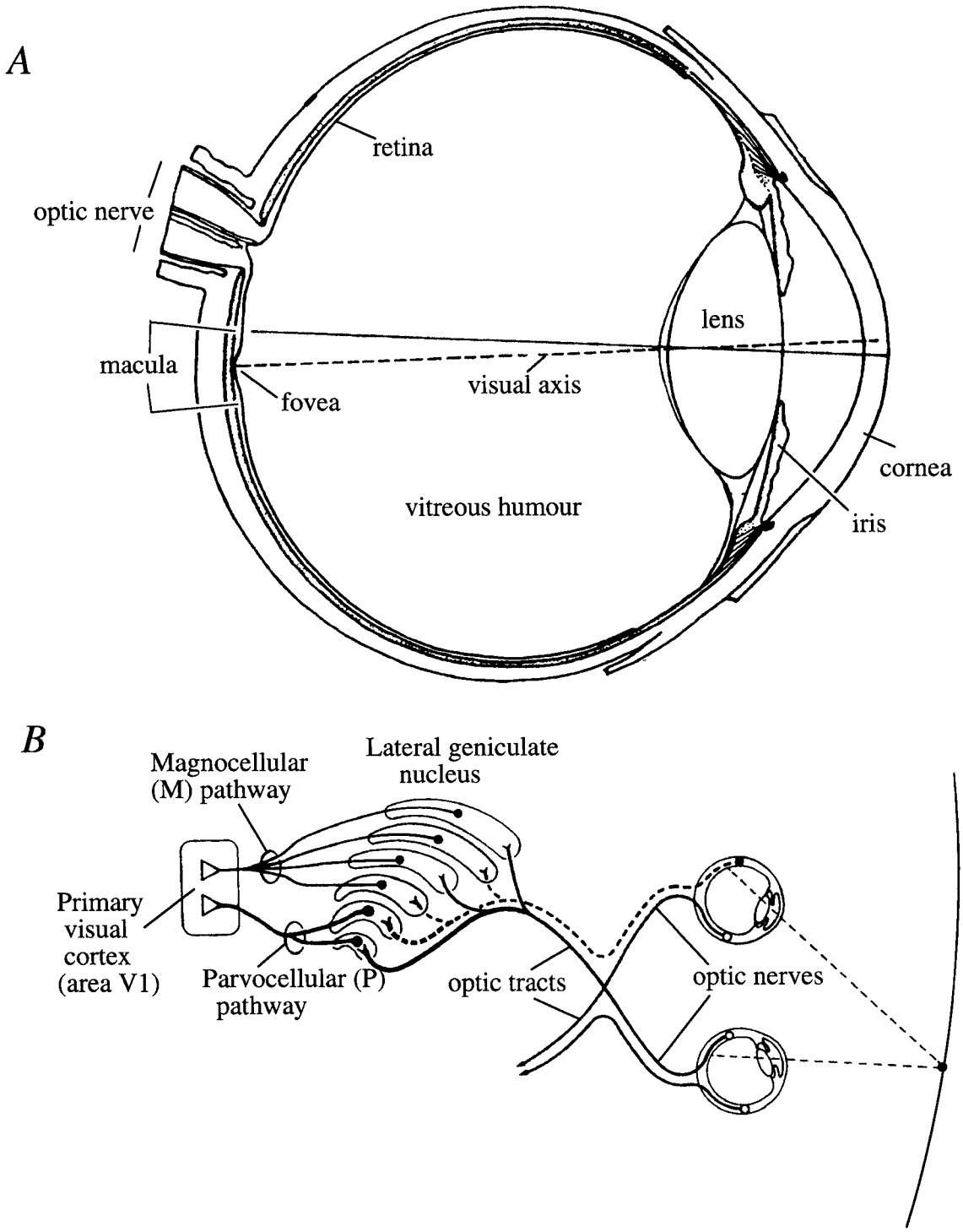

Fig. 2. Outline of the the main components of, *A*, the human eye, and, *B*, one half of the pattern-sensing part of the human visual pathway. *A* after Rodieck (1973), *B* after Mason and Kandel (1991).

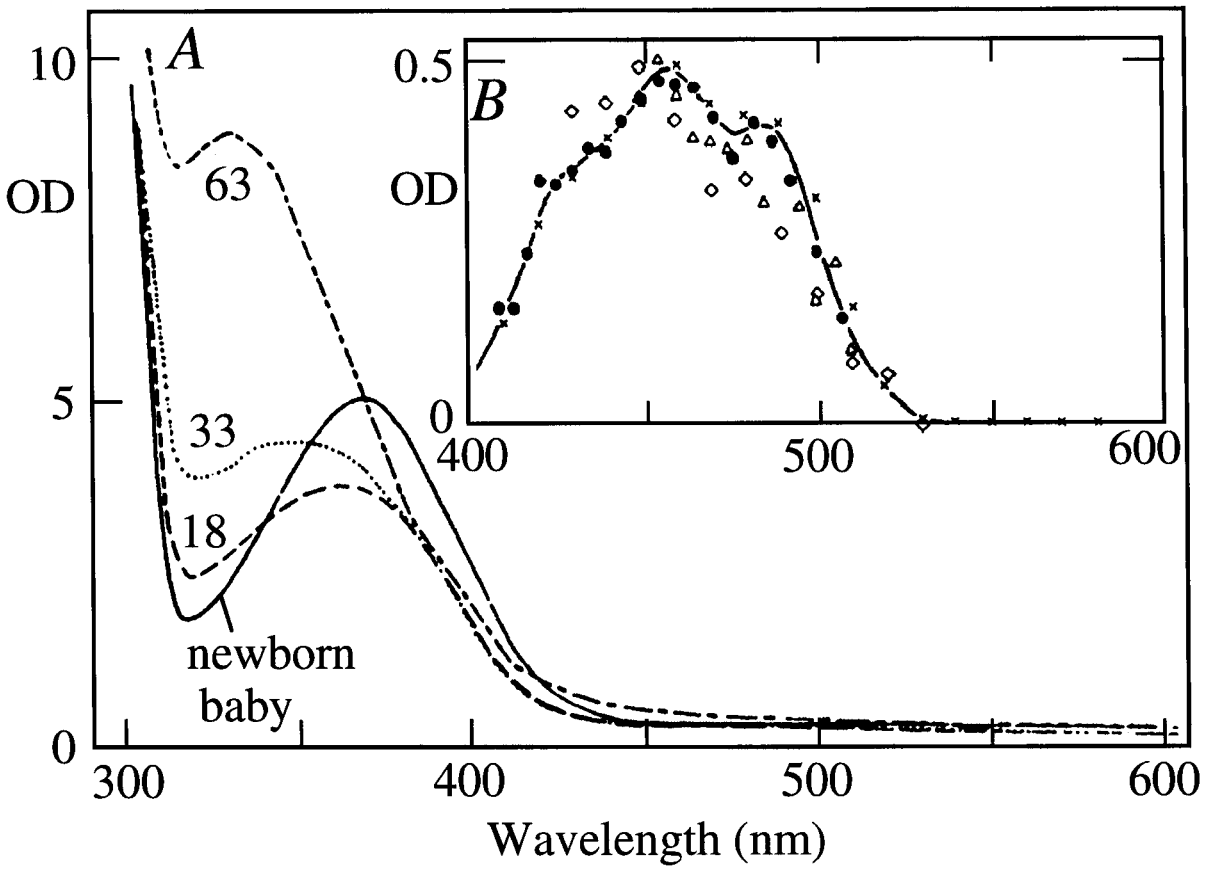

Fig. 3. *A*: spectral attenuation of light by intact human eye lenses, showing increasingly high attenuations of ultraviolet and violet wavelengths that increase with age, shown in years. Attenuation is expressed in units of optical density (OD). After Rodieck (1973) . *B* (inset): Attenuation of light by the macular pigment that covers the foveal region of the human retina. The yellow pigment blocks out light selectively in the blue-violet region of the spectrum (note the expanded OD scale). From data assembled from several sources by Wyszecki and Stiles (1982).

2. Retinal photoreception

Synopsis: Some features of the visual system's performance are set by the properties of the photoreceptors themselves, which function to capture incoming light photons. Rhodopsin is the light-sensitive molecule which is affected by the energy of a captured photon. The alteration induced in the rhodopsin molecule initiates a biochemical cascade within the photoreceptor which alters its signal to the cells with which it communicates. This altered signal is the first step in the visual process. The mechanism is so sensitive and the amplification within the photoreceptor so great that a single photon capture can give rise to a conscious perception of light.

The human eye capsule is roughly spherical (Figure 2A). Vision starts in the *retina*, the tiered collection of cells about 0.2 mm thick that adheres to the curved inner surface of the tougher eye capsule. Light from objects is focussed at the retina through the transparent optical media by the curved cornea, the focus is trimmed by the internal lens, the aperture of which can be adjusted by the variable pupil, as is well described in most textbooks (Figure 2A). Because the eye evolved from and now develops as an infolding of the brain, with the layer of photoreceptor cells closest to its inner surface, light passes first through the thin transparent layers of the retina before reaching the sites at which it is absorbed, in the parts of the rods and cones nearest the back of the eye, their *outer segments*. Some scattering can be measured there, but this is minimized by local absorption of stray light by black pigment granules in the surrounding non-neural pigment epithelium. The outer segments consist of tightly packed stacks of membrane disks or infoldings of the surface, in which the light sensitive photopigment generically called *rhodopsin* is concentrated, as about 85 percent of the total protein there, at a density of about 10^8 molecules per rod (Dratz and Hargrave, 1983). The specialized structure serves to massively increase the membrane surface area, and as a consequence, to increase the fraction of light absorbed that is incident upon the cells, estimated at 30 percent or more at the best wavelength. Of the photons entering the cross-section of the rhodopsin molecule in a variety of species, two thirds are capable of initiating an effective visual signal, one third apparently degrading to heat; i.e., the quantum efficiency is about 0.66 (Dartnall, 1968; Brindley, 1970).

The process of photoactivation by rhodopsin is complex and is the subject of much current research. It is basically similar in rods and cones, is now understood quite well and runs briefly as follows. Light is absorbed because each large opsin molecule, an apoprotein, holds bonded near its centre a small kinked carbon side-chain, retinal$_1$. Retinal$_1$ is related to vitamin A$_1$ and originally derived from dietary carotenoids (other forms of retinal occur in the visual pigments of other species, but not in human photopigments).

When light radiation resonates appropriately with the conjugated chain carbon backbone of this form (the isomer 11-cis retinal$_1$), the energy of a photon photoisomerizes it into its straighter form, all-trans retinal$_1$. Photoisomerization triggers complex conformational changes in the larger opsin part of the molecule, which rapidly results in formation of an activated derivative, metarhodopsin II; this activates the first loop of an enzyme cascade within the membrane. It catalyses the nearby molecular complex transducin (a G-protein) to undergo many cyclic changes, in each of which each transducin spins off then regains a subunit. The released subunit in turn activates many cycles of a local enzyme, phosphodiesterase, which breaks down small internal messenger molecules, cGMP, present in the cytoplasm of the photoreceptor. In

the dark, these cGMP molecules are partly bound to large protein channel molecules in the surface membrane of the cell, holding these channels in the open state. The channels are permeable to cations, particularly the sodium (Na^+) and calcium (Ca^{2+}) ions present in the tissue fluids. In the dark, a current of sodium ions flows steadily into the outer segment down the inwardly directed concentration gradient for Na^+ that all body cells establish by actively pumping out this ion. When activated by light, the cascade causes cGMP levels to fall, the channels close, the Na^+ currents decline and this reduction makes the inside of the photoreceptor become more negatively charged than it was previously, in the dark.

No nerve impulses arise in these photoreceptors. The negative voltage change during illumination simply spreads along the photoreceptor's short axon to its output at the synaptic terminal, where it forms the visual signal by which the axon communicates to the next type of neuron, one of several types of bipolar cell. The change in voltage alters the release of a small external messenger, the *neurotransmitter* L-glutamate, from the terminal (Massey and Redburn, 1987). Glutamate bridges the gap by diffusing to the processes of the bipolar cell, where it binds transiently to special receptor sites on the cell membrane (another form of channel molecule), altering the bipolar cell's electrical activity.

The scheme outlined above accounts for the huge amplification in early vision that allows the small amount of energy in a single photon to be translated into the much larger energy needed to drive the cell's output: one successful photon capture is sufficient to stop exchange of several million Na^+ ions across the photoreceptor cell membrane. This generates an electrical change big enough for a one-photon event to be recorded with a good signal/noise ratio (Baylor and Schnapf, 1987; Tessier-Lavigne, 1991), which provides a stimulus adequate to trigger the next neuron, through the change in neurotransmitter release. In extreme dark-adaptation, a single photon thus can have a large neural effect that is experienced in the cerebral cortex as part of a sensation.

C. Colour Vision

1. Colour discrimination and colour naming

Synopsis: The perceived colour of an object is determined largely by the wavelengths of light reflected from it, but the perception is also affected by light from adjacent surfaces. Despite this local contrast effect, colour constancy mechanisms ensure that perceptions of colour are relatively stable even under very spectrally different lighting situations. In test situations, people can discriminate millions of colours based on combinations of hue, saturation and intensity of the light reflected from a surface, but most cultures use only a few distinctive names to describe broad classes of colours.

There is a strong correspondence between the wavelength delivered as a stimulus and the colour reported back, for monochromatic lights projected into the eye or on to neutral backgrounds, seen by people with normal colour vision. In the middle of the visible spectrum, two wavelengths that differ by only about a nanometre can be distinguished when compared together (side-by-side, or in fairly slow alternation). From this perspective we can be thought to possess a quite subtle spectral palette of about 150 separable pure colours (Wyszecki and Stiles, 1982; Goldstein, 1984). However, the colour seen can also be affected quite markedly by what is expected for the object, and this is helped by colour constancy mechanisms mentioned later (in most parts of the world we expect grass to appear green even in the red-shifted light of a sunset, and the constancy mechanisms assist in ensuring this outcome). Adjacent surfaces with contrasting colours also exert an influence on the colour perceived (the colour contrast or Mondrian effect).

When a beam of light consists of a wide range of wavelengths delivered at similar intensities across the visible spectrum, it stimulates each of the different cone receptors about equally, and an observer will report the mixture as white. If white light is added to monochromatic light at, say, 600 nm, the original red colour becomes pink, and the pink becomes progressively paler or washed-out in appearance as more white is added. The appearance of the primary colour (red) is said to be *saturated* if presented alone, and becomes increasingly *unsaturated* as more white is added. The degree of saturation at each point of the spectrum adds another dimension to the range of colour that we can effectively distinguish. The number of these saturation levels varies from about 6-20 in different parts of the spectrum. The number of colours possible climbs at least into the thousands. Finally, there is the *intensity* or *brightness* domain, discussed in a later section. Each colour can still be perceived at different levels of brightness, but only a few brightness levels are distinguishable within one scene of a certain average ambient intensity. As the average ambient level rises a new set of intensity steps emerges. Overall, the number of possible colour steps distinguishable by humans thus rises into the millions.

Different cultures vary in the number of colours recognized by distinct names, but most have developed only a half-dozen or so primary names, plus a few modifiers (pale green, off-white). Obviously, each name is used to describe a much coarser subdivision of the visible spectrum than we can actually discriminate either in special visual tests, or in real-life situations where we can make careful simultaneous comparisons, such as when viewing paintings, observing the changing shades of natural lighting penetrating into a room, or even selecting wallpaper.

2. Rods and cones

Synopsis: Rod photoreceptors mediate scotopic vision in low light intensities (only shades of grey are discernible), using a single light-sensitive pigment molecule. Cones mediate photopic vision (including colour discrimination) at higher intensities using three classes of pigments that differ in their spectral sensitivities. By pooling signals over both space and time, rods achieve much greater sensitivity to weak signals than cones, but at the cost of poor discrimination in both space and time. Rods thus provide grainy, black-and-white images at low light levels, while cones provide sharp, coloured images, but only at high light levels.

The visual system requires 30-40 min to completely dark-adapt while the ambient light intensity sinks below a certain level, as happens naturally at twilight. Objects then lose the appearance of colour regardless of the illuminating wavelength, and appear along a black-white continuum as shades of grey. Illumination has slipped below the threshold for the less sensitive retinal cones but still lies within the range of the dark-adapted rods. Colour discrimination in animals always depends on comparisons between the electrical outputs of photoreceptor subsystems, made by a deeper layer of nerve cells, but the comparisons obviously must be made between subsystems having *different* spectral sensitivities to allow this. Rods in humans all share an *identical* spectral sensitivity function (Figure 4) and only the rods are signalling at twilight. Neither the rod itself nor the subsequent neural machinery in the brain has any way to distinguish a modest stimulus at, say, 400 nm from one of equal photon count and effectiveness at 550 nm where sensitivity is similar, or from a dimmer one at the most effective wavelength, 500 nm. No colour dimension at all is therefore available, so surfaces are seen only as possessing variable *brightnesses*, appearing to us as different shades of grey.

Three explanations have emerged for how the cone colour system is outperformed by the rods at low light level, but not at high brightnesses. (1) Rods have been found to be intrinsically more photosensitive than cones, where direct comparisons have been made, so their electrical signals at low intensity are much larger. Only about 200 of the 10^8 rhodopsin molecules need to catch a photon for that rod to be fully activated (Baylor *et al,* 1984). This is partly because the rods are longer and so absorb somewhat more of the incident light, but also because the biochemical machinery of the rod appears to have a higher amplification factor. The trade-off is that a rod reaches the ceiling of its electrical output at a much lower light intensity than does a cone, so it is effectively disabled at even moderate intensities, where cones start to dominate. (2) Large numbers of rods (up to about 500) pool their signals together in making connections with the next layer of nerve cells in the retina (the rod bipolar neurons), while only small numbers of cones contact single cone bipolars, reducing to only one per bipolar cell, in the fovea (Gouras, 1991). Summation of the inputs from many photon catches generates a more reliable, less noisy signal in a rod bipolar, which appears correspondingly more photosensitive than a cone bipolar. There is again a trade-off for the increased performance, of which we are all aware. Because spatial pooling necessarily removes the local information on the origin of individual photons reaching the retina, the "grain" of the visual image (our spatial resolution) is much worse for rod vision than it is for the cones of the fovea. Objects appear much fuzzier to us in low ambient lighting at night. (3) Less obviously, absolute sensitivity for rods is increased not only by *spatial* integration of photons across the retina, but also by *temporal* integration within each rod channel, so more photons are summed over a longer time interval to generate

a more reliable response: the rod system is slow. Here, the sacrifice lies in the poorer temporal resolution of the rod system, that is, the origin of visual signals in time becomes less certain (Barlow, 1965).

This might suggest that the early stages of vision simply involve switching between two subsystems, rods and cones, each optimized to its own different light intensity range, but this is an oversimplification. Early vision is best seen as a challenging signal/noise discrimination problem, involving the extraction of the desired visual signal from various forms of undesirable contaminating noise. This problem is serious at all light levels (Sterling, 1990), and has to be solved at the outset, or its effects contaminate the rest of the visual processing (Srinivasan *et al,* 1982). Prominent among these sources is the "photon noise" that arises inevitably from the irregular (random) fluctuation of photons arriving from objects, and which gets proportionally worse in low light. The solution to maximizing signal over noise is thus not a two-state problem but varies continuously with ambient light level. Optimization requires a continual dynamic adjustment of spatial and temporal pooling as ambient intensity changes in the retina, that will even differ between adjacent patches of retina when these are exposed to different local illumination. Dynamic changes in pooling with intensity close to the form predicted have been detected in both invertebrates (Laughlin and Hardie, 1978) and vertebrates (Werblin, 1971; Sterling, 1990), indicating the fundamental importance of this optimization process. The optimization concept has been extended to incorporate spectral processing as well (van Hateren, 1993).

Since the rod system is already an excellent quantum detector with an efficiency as good or better than the best current products of human technology, it might seem that performance cannot be improved further. Considerable improvement is possible, for instance in low-light cameras, by use of a much larger lens than the human eye's (the total photon catch is proportional to the cross-sectional area of the lens). Also, the electronic integration time can be extended beyond that used in the rods, and adjacent video frames can be averaged, further sacrificing time resolution, however.

3. Cone mechanisms for colour vision

Synopsis: Colour vision depends in humans on the presence of three classes of cone photoreceptors in the retina. These differ in the pigment molecules they contain, although all three pigments are members of a single protein family. These three pigment molecules absorb across the entire spectrum of visible light, but their sensitivities peak at different wavelengths. The photopic sensitivity curve is a product of their joint sensitivities. Colour vision is based on neurons which receive input from these cones and compare the outputs of these differentially sensitive receptors. Different wavelengths of light can be discriminated because each affects the three pigment molecules to different degrees.

Colour mechanisms were initially characterized indirectly as abstract processes defined by colour matching. They can now be identified explicitly with processes at the cellular, genetic and molecular levels from work in the last three decades. For instance, although the trichromatic theory was first suggested in the last century by Young and Helmholtz, a direct demonstration that there are three cone types finally came 30 years ago, when three visual pigments were measured by microspectrophotometry to reside in different cones, in excised human retinae.

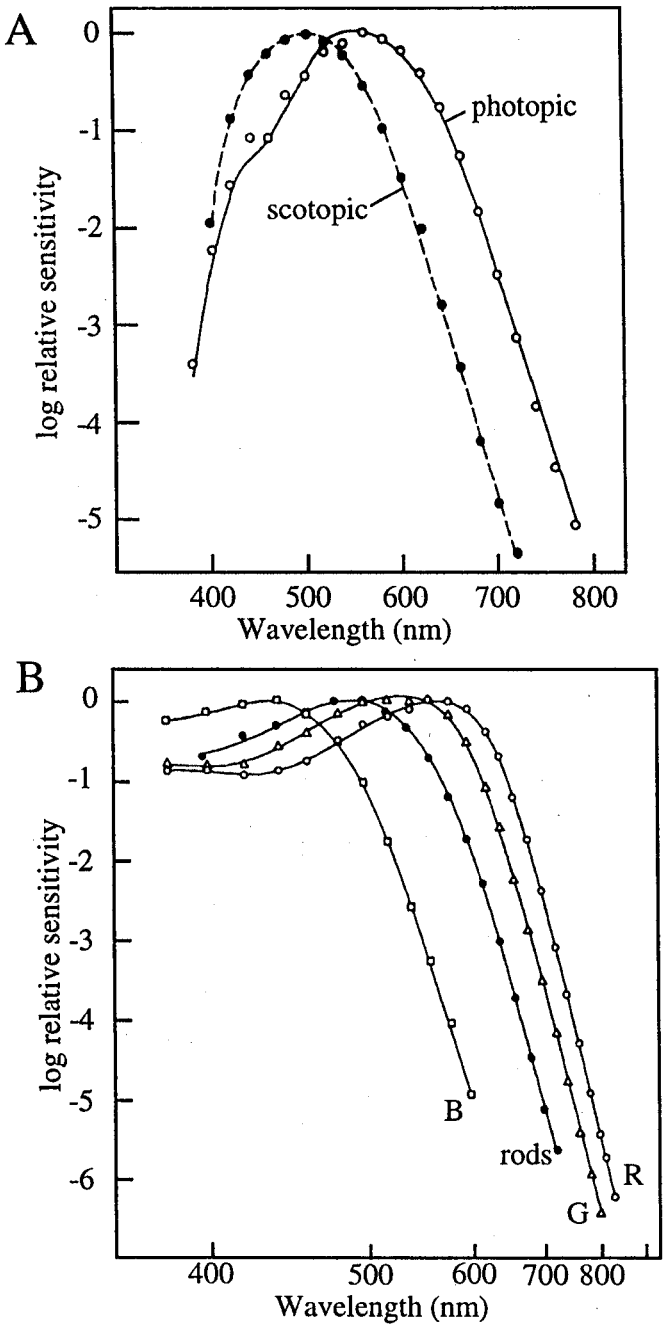

Fig. 4. *A*. The continuous curves are the scotopic (V'λ) and photopic (Vλ) spectral "luminosity" or sensitivity functions for humans operating in dim light and bright daylight, respectively. The superimposed filled circles show the average spectral sensitivity determined from electrical recordings from single macaque monkey rods, which are almost identical to those of humans; the data fit V'λ very well. The open circles are from monkey cones, and show the function expected if the red cones are about twice as effective as the green cones in contributing to Vλ, while the blue cones are assumed to be ineffective. Again this prediction from monkey photoreceptors fits the human photopic sensitivity function very well. *B*. Spectra taken from single rods and from single blue (B), green (G) and red (R) cones, then averaged by class, and used for the predictions in *A*. The data are plotted on a scale proportional to frequency, giving a non-linear scale of wavelength. After Schnapf et al (1988).

These and subsequent results suggest that each cone or rod normally expresses only a single gene coding for one pigment, not mixtures of pigments. In humans, some current estimates of the average wavelength of maximum absorption (l_{max}) for each of the three cone classes are 419, 531 and 558 nm, and 496 nm for the rods (Dartnall *et al,* 1983; Brown and Wald, 1964; Marks *et al,* 1964), while measurements on monkeys have yielded similar values (Bowmaker, 1984; Baylor *et al,* 1984) (Figure 4B). The values for the blue cones are probably too low, displaced to shorter wavelengths by scatter (MacNichol, 1986). Some variability in the individual results is expected from the noise in the recordings, but there are strong indications that real biological variations exist beyond this: the l_{max} values from samples of both the green and red cones in humans are bimodal, and appear to be drawn from two subpopulations with maxima 10-20 nm apart (Dartnall *et al,* 1983). This is unresolved (Bowmaker, 1984), but it is possible that populations and even single individuals may be polymorphic for the two longer wavelength cones. There is no clear parallel in the genetic results (see below).

The spectral absorption or sensitivity curves for all known visual pigments are quite broad and are similarly shaped, with half-bandwidths (HBW) around 100 nm. The curves are identical in rods of the same species, when determined in technically favourable cases in situ in the photoreceptor or from pigment extracts (MacNichol, 1986). If a logarithmic scale of absorbance or sensitivity is also used, the curves appear parabolic for as far as they can be followed towards the red end of the spectrum (Figure 4B), emphasizing that there is no wavelength at which absorption actually falls to zero, only practical measuring limits. At the short wavelength end, absorption levels off somewhat then rises in the far ultraviolet due to direct absorption by tryptophan residues on the opsin, but this is of no practical significance because far UV is removed by entoptic absorption, particularly by the lens. For all visual pigments including those that are retinal$_1$-based, the HBW broadens progressively as the l_{max} shifts towards longer wavelengths. It was originally thought that the shape would be invariant when plotted on a frequency scale, and so could be described by a single empirical nomogram that would predict the shape of any retinal$_1$-based pigment, given the l_{max} (Dartnall, 1953). Better measurements have shown that this is incorrect, but a simply transformed relative frequency scale has since been found that does yield shape-invariance to within the accuracy of the best measurements (Mansfield, 1985; MacNichol, 1986). This gives a unified way of specifying all the absorption curves.

The breadth of the spectral absorption curves ensures that each visual pigment in humans is sensitive at all points across the visible spectrum, though unequally so (and for the blue cones in red light the relative sensitivity is so low as to be insignificant). This breadth at first seems antithetic to possession of fine wavelength discrimination, until we realize that colour vision is possible by virtue of *neural comparisons* made by later neurons of the differing relative outputs of the three cone types. Colour discrimination is best where the curves actually overlap most closely with the steepest gradient, as occurs at long wavelengths because of the strong overlap of the green and red cones (Figure 4B) (Wyszecki and Stiles, 1982). Blue light is reduced at the fovea of the retina because of the yellow macula (Figure 3B), while blue cones are actually largely excluded from the central fovea where form and colour discrimination are excellent, but which is therefore practically dichromatic (Williams, 1986; Winkler and Rakic, 1982). The area is so small that this is not appreciated perceptually unless images are artificially stabilized there, because of saccadic (fast, involuntary) eye tremor movements.

Genetic characterization of the photopigments has come only recently. The opsin genes of normal and colour-deficient human males have been identified, their opsin proteins cloned and the complete amino-acid sequence of each opsin has been determined (Nathans *et al,* 1986a; Nathans *et al,* 1986b). In humans, the sequence similarities show that the three cone and one rod visual pigment all belong to a single protein family; i.e., they diverged during evolution from a single common ancestral protein (Hargrave and McDowell, 1992).

What causes the differences in spectral absorption between cones, if the photon-catching chromophore retinal$_1$ is exactly the same for each? Retinal$_1$ in its free chemical form absorbs in the near UV (Rodieck, 1973; Dowling, 1987), so it has long been realised that its absorption of visible wavelengths and the differential sensitivity of cones must depend on differences that arise when retinal$_1$ is incorporated into a larger pigment molecule. It appears that electronic interaction between retinal$_1$ and its local environment inside the folded protein are responsible for these features. This idea has been tested experimentally by site-directed mutagenesis of individual amino-acids in opsin (Savarese and Fraser, 1992; Rao *et al,* 1994). It appears that just 2-3 of these amino-acids are particularly important, through interaction with the retinal binding site on lysine-296, and that mutagenesis of either of two of these is enough to cause a large shift in the position of the spectral peak (Robinson *et al,* 1993).

4. Colour vision anomalies in humans

Synopsis: Genetic abnormalities can lead to different patterns of colour blindness based on loss of one of the three cone pigments. These defects are much more common in men than in women because the relevant genes are on the X chromosome. Less serious gene abnormalities lead to more modest changes in colour perception. Within the normal population, there is also variation in colour matching, based on differences in ocular mechanisms and other, unknown factors.

Do we need to worry about whether all people share a common colour sense? Where does the known variation come from? The most extreme differences occur in the small proportion of the population that is *colour blind*, almost all male. Such people are able to sense wavelength as shades of blue at short wavelengths and as yellow at long wavelength, but in the middle of the spectrum, perceived colour appears to wash out and at one wavelength becomes grey. The wavelength of this grey or neutral point varies according to the type of defect. Strict colour blindness of this sort affects only about 2% of the population, but if *colour anomalies* are included this rises to about 8%. In colour matching tests, individuals are asked to match a test wavelength or colour mixture by varying the proportions of a comparison stimulus: this is made up by mixing just three "primary" colours, usually blue, yellow and red (two such primaries are insufficient, four are unnecessarily many). Normal people choose similar proportions (see below), implying that the general population is rather uniform in its colour sensitivity. Persons with colour anomalies, however, need quite abnormal ratios of primaries to make successful matches, but many such people are unaware of their deficiencies until so tested and informed.

Defects of colour vision at retinal level are hereditary and have a common origin: the absence of either a red photopigment gene (in *protanopia*) or a green gene (in *deuteranopia*) that would normally be expressed in one of the three classes of retinal cone photoreceptor (Nathans *et al,* 1986a; Nathans *et al,* 1986b; Nathans, 1989). The green and red genes lie in a row on each

X chromosome. In men, the paired sex chromosomes are dissimilar (XY), and having only one X chromosome, an abnormality in one or other X gene always manifests itself as a colour deficiency in men. Women possess two X chromosomes (XX) and therefore have two copies of each gene. A normal copy can compensate for and mask the presence of a deficient partner present in a single dose (most gene mutations are recessive, including those for colour blindness). A defect in both copies is therefore required before the effect is expressed in women, so overt colour blindness is much rarer in them (0.03%, or 0.5% of the population when anomalies are included). Women act mainly as carriers for expression in male offspring in the following generation. There is usually one copy of the red gene per X chromosome, but commonly 1-3 copies of the green gene. It is believed that defects arise during genetic recombination to produce the sex gametes, when aberrant unequal exchange occasionally occurs between chromosomes. One gains an extra, duplicate gene at the expense of the other, which loses it and so generates the defect.

Anomalies of colour vision are also correlated with aberrant, unequal fractures of the X chromosome during genetic recombination, according to Nathans *et al* (Nathans *et al,* 1986a; Nathans *et al,* 1986b). They found that part of a green gene occasionally becomes spliced to the complementary part of the red gene. The hybrid gene successfully codes for a visual pigment, but one with altered (intermediate) spectral properties. Trichromatic vision is still present but one cone type has its absorption spectrum displaced, causing subjects to make the anomalous colour-matches. The remaining chromosomes are present as matching pairs which therefore carry two copies of each of their genes. Those for the blue cone photopigment lie on one of these (chromosome 7) and those for the rod pigment lie on another. Two missing or aberrant genes are therefore required before a visual defect is observed involving these pigments (Nathans, 1989). While such defects exist, as expected, they are very rare, with 0.001-0.002% of the population having *tritanopia*, colour blindness associated with blue cone defects.

When colour-deficient individuals are ignored, are the remaining members of the population with "normal" colour vision uniform in their appreciation of colour? This is difficult to specify simply. Variation about a mean across the spectrum is given in Fig. 5.17 of Wyszecki and Stiles (Wyszecki and Stiles, 1982), for the magnitudes of the three colour functions that can be extracted from colour matches. The easiest way to present the variation is to consider the ratio of red-to-green sensitivity obtained from individual colour matches. This variation is surprisingly large: about 30% on either side of the average R/G ratio is needed to encompass most of the population. If the normal population is genetically uniform for the three cone pigments, where could this range arise? This is unclear, but the two types of inert (non-photosensitive) pigments are implicated. The lens cuts off short wavelengths, and at least some of the variation in normal colour matching is attributable to differences in its pigmentation between individuals, augmented by changes with age (see above, Figure 3A). Another source is the variability in the density of macula pigmentation that covers the fovea (Figure 3B). Variability in the neural connections within the subsequent colour coding neural circuits is not known, but is difficult to study in humans; it is found in simpler animals. Finally, the distribution could probably conceal two normally distributed overlapping human subpopulations with peaks perhaps 10 nm apart as suggested by microspectrophotometry (see above), but there is no further evidence for this.

In summary, colour balance varies somewhat within the normal population, by perhaps ±30% on one metric, so modest variation in the sensory percepts of different people viewing the same scene is to be expected. This range is several-fold smaller than the distortion conferred by having a cone colour anomaly, however. Since even these cone anomalies are often not apparent to the individuals concerned until it is pointed out to them, it is likely that the lesser variations between "normal" people are subjectively neglected by them. This implies that they can also be ignored by us, in considering the effects of subtle changes in lighting inside dwellings, which depend on such subjective estimates.

5. Opponent processing and colour vision

Synopsis: Colour vision depends on comparisons of the outputs of the different cones to form red-green and blue-yellow colour channels. These comparison processes give rise to coloured aftereffects based on adaptation of a particular class of cone. Receptive field structures of central neurons are designed with paired opponent regions, so that the effects of a uniform coloured field of illumination are largely ignored by the system. On the other hand, contrasts between differently coloured adjacent regions are emphasized by this arrangement of receptive fields.

The evidence that human photopic vision at the photoreceptor level is made up of three independent mechanisms (trichromacy) is now overwhelming. *Comparisons* need to be made of signals from the three cone types, to generate a sense of colour (pooling signals without comparisons would have the same effect as mixing up different pigments in the same cone). Accordingly, there are human colour experiences which indicate that parts of the spectrum are analysed perceptually based on comparisons in opposition: one such colour pair is red-green. When a normal observer stares at a brightly illuminated red object then looks away at a neutral white or grey surface, a pale green *afterimage* of the object appears there, then fades. If the object is bright green, the afterimage is pale red. In the late 19th century, Hering explained these and related effects in terms of red and green interacting in opposition, forming two halves of an *opponent colour* process (Hering, 1964). This can be recast in modern terms if it is supposed that red and green cones connect synaptically into a local neural network in which they have opposing effects, where green excites and red inhibits the network (or vice versa). When one input, say red, is driven strongly, its own sensitivity reduces rapidly (*adapts*) to this and to subsequent stimuli (Boynton and Whitten, 1970; Green, 1986): the lowered photosensitivity persists after the original object is no longer viewed, then gradually recovers. In the case just considered, when the observer switches to view a white surface, the little-adapted green inputs signal nearly at full strength, exciting the network. The strongly adapted red system remains depressed for a while, so the opponent inhibition that it originally exerted is reduced, and the balance swings in favour of excitation, associated in this neural channel with the green mechanism. The colour balance signalled by it is therefore temporarily stronger in its green component than it would be normally, giving a green tint to the part of the visual field where the original object lay, explaining the afterimage effects above.

Afterimages are experienced also for the pairs blue-yellow, and black-white, so there appear to be three basic opponent processes, two concerned with signalling *colour*, while the black-white system signals *brightness* in the photopic range. The interactions in one opponent system can include all three classes of cone (Goldstein, 1984). In general, the later stages of the visual system show spectral sensitivities that reflect the opponent colour processes, not the underlying absorption curves of the three types of cone. Note that a consequence of the perceptual organization is that the perceived colour of a surface does not depend just on the spectrum of light reflected from it, but is influenced by the colour viewed recently. This is an example of *successive colour contrast* influencing our colour perception at each instant.

Do these deductions correspond to actual neural processes, and what is the nature of the interactions? Most evidence about neural processing comes from electrical recordings made one-by-one from individual neurons in adult animals, at different stages in the visual pathway concerned with colour. Since the colour-coding neurons tend to be small and difficult to record from, the information available is spotty and species-specific. Other useful information comes from the anatomical connections made between identified neurons, also difficult to unravel.

Spatial opponent processing occurs already in the bipolar neurons in the retina in goldfish. Light falling on the small centre of the cell's *receptive field* (RF) induces a positive electrical response, while that falling in the surrounding part of its field induces an opposing, negative response (Kaneko, 1970). These are ON-centre, rod bipolars; there is a complementary set of rod bipolars wired in exactly the opposite fashion, forming an OFF-centre channel (see III.C.2). *Colour* opponent processes are known in some of the horizontal cells that also connect to the axons of cones, in fish (Svaetichin, 1956); these cells form lateral networks in the retina. Recent work suggests that these may not form part of the colour-processing system of the brain (Reid and Shapley, 1992). The best studied retinal neurons are the ganglion cells that form the output to the brain. Here, true spectral-opponent cells have been described by Daw (1984) in goldfish. These cells are *double-opponent*, consisting of a *RF centre* excited by green light and inhibited by red, encircled by a zone (the *RF surround*) where green inhibits and red excites (Figure 5). What is the rationale for such a complex arrangement? Which inputs are effective, and which are ignored? When uniform red illumination floods the whole RF, its opposing effects in the centre and surround cancel, and the cell fails to notice this stimulus; similarly for uniform green illumination, so these cells largely ignore uniform illumination, even when this is changing over time. The strongest effect on the cells is obtained with a green object on a contrasting red background (or vice versa), so these cells are wired up to code best for *simultaneous colour contrast* . A consequence, even more important than for sequential colour contrast, is that the colour signalled by the cell is affected not just by the spectral distribution falling in the centre of its RF. Its RF surround (the local wavelength distribution *around* an object) also affects the perceived colour. There are striking parallels perceptually in humans (Goldstein, 1984) that start with this mechanism but are augmented by longer-range interactions among colour processes that are added in the cortex.

Since the centre of the ganglion cell receptive field is thought to arise from superimposition of a few smaller fields of the preceding cone bipolars that connect with it, these bipolars are thought to have opponent colour-coded receptive fields. Spectral opponency therefore is set up right at the output from the cones to some subset of the bipolars. A working hypothesis from Daw (1984) is given in Figure 5.

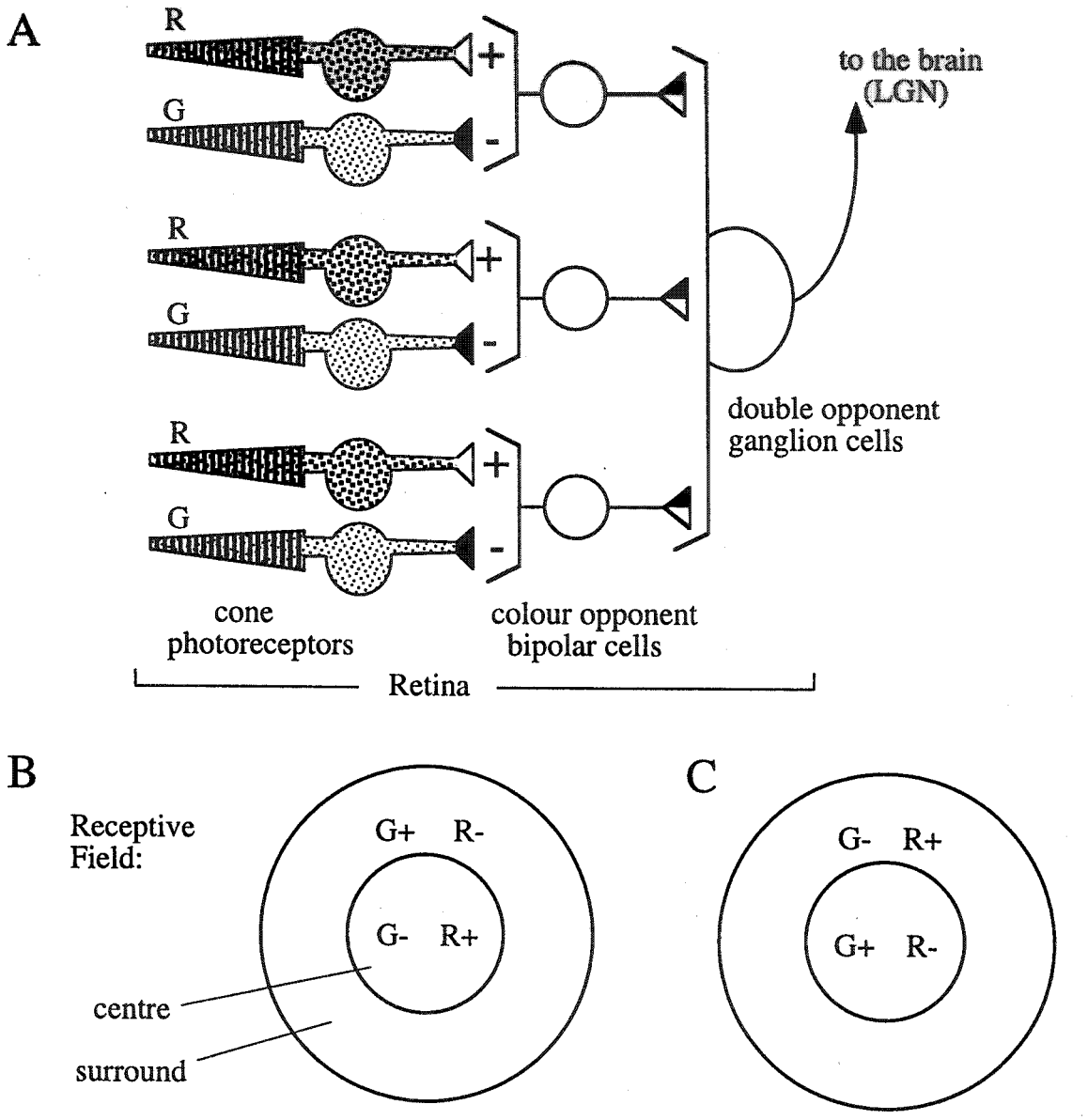

Fig. 5. *A.* Neural circuitry in the visual system that can generate colour sensitive double opponent neurons. The "circuit" starts with signals sent from green- (G) and red-sensitive (R) cones in this example from a lower vertebrate, modified from Daw (1984). The symbols + and - signify excitatory and inhibitory connections, respectively. The cells in the last stage that do the same operation in monkeys and humans are believed to lie in the cortex, not the retina. *B.* Through their connections, such neurons have "receptive fields" (RFs) that represent a portion of visual space, projected through the eye's lens on to the retina. A double opponent neuron has a two-part, concentric RF in which the response to colour is opposite in the centre and surround. The type here would respond best to a small red object on a greener background, and signal these local image features to the brain. Its complementary type (*C*) signals the reverse colour contrast. Other ganglion cells are connected on similar principles and involve the blue cones as well. They signal blue-yellow and black-white contrasts, in addition to the red-green mechanism shown here.

Similar double opponent cells are also found in monkeys and presumably occur in humans, but are not found until the primary visual cortex (area V1). Thus the neural design is the same but has been reached by a different route. Colour vision is even richer in fish (some are tetrachromatic and respond both to UV and deep red). These exceptional abilities are thought to have been later lost in primates; the ancestral primates are thought to have been nocturnal tree-dwellers with habits like modern lemurs, with rod-dominated retinae. Colour vision was secondarily re-invented somewhere on the line of evolution to the more recent primate ancestors of humans.

D. Central Mechanisms

1. Lateral geniculate nucleus of the thalamus

Synopsis: Retinal ganglion cells from the two eyes project to the two lateral geniculate nuclei (LGN) such that the left half of each visual field is represented in the right LGN and vice versa. Visual spatial information is mapped onto LGN neurons, and some also code for colour information. Activity in the LGN and its output to the visual cortex are shaped by input from other structures, especially from the cortex, which may be part of the mechanism underlying selective visual attention.

The axons of the ganglion cells run over the retinal surface and gather into the optic nerve that leaves each eye. The two nerves then run posteriorly and combine at the optic chiasm, underneath the brain. From here the pathways divide again into the two optic tracts, within which most of the retinal axons run to terminate in one or other of the two lateral geniculate nuclei (LGN) of the thalamus, near the centre of the brain. They re-group to do this, so that the axons that process signals from the *left* half of the visual field in each eye run to the *right* LGN, and vice versa (Figure 2B). The spatial maps (called *retinotopic* or *visuotopic*) from the retinae are still preserved in register in the LGN but now represent the contralateral visual hemifield. The maps are distorted by a variable *magnification factor*, such that about half of the LGN is devoted to representing the tiny fovea of the retina, while the retina towards the eye's periphery is progressively under represented. Although each LGN receives concordant information from the two eyes, the axons of each remain functionally separate, segregating in semi-alternation into six layers in each LGN. What is served by having so many layers is unclear, but the upper four contain smaller neurons (the *P*, or parvocellular, layers) concerned with colour vision, fine spatial pattern, shape and texture, and stereoscopic vision. The lower two layers have larger, faster conducting cells (the *M*, or magnocellular, layers), concerned with the first processing of motion and flicker information while not preserving colour.

Some of the earliest evidence that primates use spectral *opponent processing* came from work on monkey LGN (DeValois, 1960; DeValois and Jacobs, 1968), but the RFs of the cells were not explored. Although controversial, recent work (Schiller and Logothetis, 1990) shows that P cells that serve the fovea region not only have wavelength-specific RF centres (e.g. a single red cone) but also a specific opponent surround mechanism (always from green cones). This arrangement must stem from a similar organization at retinal ganglion cell level, and therefore at the preceding level of the midget bipolar neurons too, that service a single cone (Reid and Shapley, 1992). The cells are mostly the *single opponent* type, with, for example, a red

exciting input at the centre of the RF (R+), and green inhibition in the surround (G-).

No radically new neural transform appears to be introduced at the LGN, however. In the cat, many of the RFs of the LGN neurons resemble those found in the retina, but many appear to be made up of the superimposed RFs of only 2 or so closely overlapping retinal ganglion cells. These are wired together so that the background "noise" discharge of the retina is usefully suppressed in the LGN neuron (Cleland *et al,* 1971; Kaplan *et al,* 1987). Only a small percentage of the synapses in the LGN actually come from retinal axons, and accordingly there have been persistent suggestions that other sensory modalities can control ("gate") the visual message going through the LGN, perhaps as part of an attentional system (Sherman and Koch, 1990). The contrast signal issuing from the retina is also strongly regulated in monkey LGN neurons (Kaplan *et al,* 1987). Recently, Sillito *et al* (1994) have specified how the huge descending projections from the cortex to the LGN might amplify particular lines of information coming from the LGN at the expense of others, so selectively controlling what gets into the cortex. This is done by promoting the synchronization of nerve impulses in the selected block. This mechanism is now widely discussed for the cortex itself as a solution to the general "binding problem": in a complex parallel-processing system where some cells can belong to more than one network, it is unclear how cells doing the same task could be distinguished from others that are not, by the network itself, i.e. how their activities become "glued" together.

2. Visual cortex

Synopsis: The visual cortex receives input from the LGN and develops maps of visual space based on a variety of features, including colour. The receptive field structures in cortex also involve comparisons which ensure that changing the general colour of the environment (e.g., in reddish sunset illumination) does not prevent us from still seeing the same colours in objects as seen under spectrally different sources. This form of colour constancy is important for ensuring that the human visual system adapts to various lighting situations without losing the ability to recognize and discriminate colours.

Neurons that originate in the LGN fan out to make up the optic radiation on the same side of the brain, which distributes the neural messages to the visual cortex situated in the occipital lobe at the back of the brain. Most axons run to the largest subdivision of it, the primary visual area or striate cortex, now usually called V1. They terminate in one of its layers, often in part of layer 4C for the colour fibres, establishing another distorted map of visual space there in which the fovea again dominates. The initial pioneering work of Hubel and Wiesel in the 1960s failed to find colour-sensitive cells in V1, but now discrete cylindrical islands of such cells have been found (Livingstone and Hubel, 1984). They are called "blobs", after their appearance when stained for the cytochrome oxidase enzyme system that is densely expressed there. Receptive fields of cells in the blobs are thought to be made up by superposition of fields of the geniculate cells, for they have, in primates, *double opponent* properties (e.g. R+ G- in the RF centre, R-G+ in the surround). This arrangement confers greater sensitivity to chromatic stimuli, and effectively factors out white light (see above). The best stimulus again is *simultaneous colour contrast*, e.g. of a red spot on an opponent green background. Double-opponent yellow-blue cells are also found, and are thought to underlie the *perceptual colour-opponency* described above for humans, originally by Hering.

An important group of phenomena mentioned only in passing so far are the "perceptual constancies". These are a set of mechanisms that collectively help simplify the process of generating the perceptual model of the world that we actually inspect: if an object is reconstructed internally in the brain so that it always appears the same under a variety of taxing visual conditions, it is easier to "compute" neurally. In one of this set, *colour constancy*, the perceived colour that we attach to an object is relatively insensitive to real changes in the composition of the overall illuminating light, as might occur with a shift to the red at sunset in- or out-doors. The double-opponent cells are thought to play a major role in this. For instance, while the increased red cast at sunset will enhance the response of one type of cell to an object situated in its RF centre (R+G-), it will provoke an opposite, offsetting effect in the same cell's surround (R-G+) to which it is being compared, tending to keep the colour perceived in balance. This compensating effect also should be an important antidote to modest changes in hue inside a room, downplaying the effects due to altered window transmission.

The main parallel processing centre for colour in the cerebral cortex is believed to be the area further forward now called V4, which receives a projection from V1. This is based on diverse evidence from PET scans, and local cerebral damage as well as recordings from cells (Zeki, 1993). Most of the cells in V4 are strongly affected by object colour, unlike many of those in surrounding areas (although these are not simply achromatic). However, V4 is not exclusively colour selective but also processes object pattern, having cells interested in the orientation of an object as well as its colour (Schiller and Logothetis, 1990; Zeki, 1993). In other cortical areas, visual scientists have long been excited by the fact that fairly orderly cellular "maps" can be found of the parameter supposedly being coded there. For instance in V1, Hubel and Wiesel originally found that there is an orderly progression over the brain surface of the *orientation* of an object that best stimulates each neuron: clusters (stripes) of cells with the same orientation are found together. Intersecting with this array is another superimposed map of *eye dominance*, according to which eye best drives each neuron. It is concluded that part of the neural systems that the brain uses to identify the line-orientation of parts of objects, and of which eye is sending signals, originate in V1 (Hubel, 1982). Conceptually, this is because of the orderly progression of each coded property across the surface, which is deemed fundamental to how orientation, etc, is extracted: by local comparisons among adjacent cells with slightly different but related properties (as we saw for cone comparisons, in the retina). How each map operates as a unified neural machine (or if it does) is simply not understood.

By comparison, the manner in which colour is coded in V4 is so far not understood even at the level of whether there is some kind of coherent map. A map probably exists, however. A technique using activity-sensitive dyes infused into the neurons at the cortical surface, allows optical imaging of activity with high resolution. This has revealed that colour is mapped in area V1 in pin-wheel-like island configurations, again superimposed on the other maps for orientation, eye dominance and retinal position (Blasdel, 1989). What emerges is that V1 is the primary generalist area in which all functions are first mapped in some kind of local relation to each other. Parcels of this local information are then exported to other processing areas "in parallel", which deal specifically with either colour or with motion, and so on (Zeki, 1993). A major unresolved question in cortical function is how the different attributes of an object (colour, motion, form, etc) become re-associated together once they have been processed separately in parallel, to give a unified sense of the object that we are aware of. For specific

familiar human faces, this may be the province of single neurons, 1-5 for each face (Perrett and Mistlin, 1987). It seems likely that most other objects are represented by the distributed activity of many neurons that also code for other objects as well, raising again the binding problem (Crick, 1994).

E. Coding for Contrast and Brightness in the Visual System

Since the brightness domain also interacts with colour and all other aspects of vision, discussion of it has crept into earlier sections. It is extended here. The brightness signal in the photopic range originates from the summated output of all three cone types, which constitutes the third of Hering's opponent pathways in the visual system (the black-white, or intensity channel). As only about 10 percent of cones are blue, these play only a minor role in brightness detection.

1. Amplification and noise in the visual system

Synopsis: The visual system is required to make very fine discriminations of light intensities in a scene, but to do so over an extremely broad range of background illumination levels. Part of the solution to this problem is the process of adaptation which effectively "subtracts out" background illumination levels. As a result, the visual system can make very fine comparative judgements of intensity within a scene, but is extremely poor at identifying the absolute level of environmental illumination. Adaptation ensures that we can perform visually based tasks effectively at a number of different ambient illumination levels.

A fundamental physical conflict exists in trying to design a visual system for the natural range of light available. One half of this problem is that the extremes of contrast in a scene at one time cover a quite small intensity range (grey scale) stretching to no more than about 10:1, while within this range, differences in contrast of only 1-2% need to be detected reliably (Srinivasan *et al,* 1982). The eye viewing a scene therefore needs to code efficiently in small, subtle steps of brightness. The other half of the problem is that the total range of natural light intensity over which the human eye can operate is enormous, at the limit 10^{14}:1 (Pirenne, 1967), but more realistically in the range of about 10^{7}:1, because of the changing position of the sun, extending down through to moonlight. The range is covered in part by splitting intensity coverage in two, with cones for bright light and the rods covering the photon-poor lower reaches (see above), but this arrangement still leaves a very large range to be covered by each subsystem.

The solution to the conflicting demands is that employed in commercial hand-held test meters. The system is designed with initial high amplification to effectively cover the current scene (10:1) with the best resolution, while autoranging (*adaptation*) mechanisms are added to switch scales as the mean ambient light intensity rises or falls. Both functions are partially implemented early in the visual pathway in the photoreceptors, so as to avoid amplifying sources of noise that arise later. High amplification is conferred by the high gain synapse from photoreceptors to the following cells, which has been studied in vertebrates (Werblin, 1971; Sterling, 1990), but best in invertebrates (Laughlin, 1989). The system's output increases rapidly from no response to a maximum over about a factor of ten increase in intensity, and maintains this ability over a very wide range of mean background intensity (Laughlin and Hardie, 1978).

As the light level rises, adaptation mechanisms start to shift the sensitivity range to progressively higher levels in the photoreceptors, including those of primates (Boynton and Whitten, 1970), but the major effect occurs at the first synapse. Here, the surrounding photoreceptors average the local scene and generate an average signal that gets subtracted from that of the particular photoreceptor, so that only *differences* from mean background are amplified and sent on as signals. In effect, very large background signals can be almost completely removed, so this information is lost, while slight deviations above and below mean background that distinguish an object, are amplified to form the transmitted signal. This is why human and animal visual systems are very good at detecting contrast variations within a scene, which are the basis for object detection and of great biological value, but are notoriously poor at estimating the absolute illumination level (which usually has no value in form perception, and which is signalled weakly by a different set of ganglion cells). Because of this built-in intensity gain control, we can comfortably perform tasks like reading or cooking in a room over an intensity range that is very broad, but which is not perceived as such because of our poor sense of absolute intensity.

These selective early amplification mechanisms are augmented by the centre-surround organization of the subsequent receptive fields of bipolar and ganglion cells and those of the LGN. This is an opponent-processing, differential amplifier arrangement where the larger, less sensitive surround opposes the action of a smaller but more sensitive centre. The result is that an overall increase in background light level affects both centre and surround almost equally but in opposite directions, cancelling the effect of a change in background. The cell continues to signal differences between centre and surround, which represent the actual contrast of the object, but is immune to large overall changes in background lighting (Sterling, 1990).

2. Light and dark channels for intensity

Synopsis: The mechanism involved in detecting light intensity changes includes separate "ON" and "OFF" channels of information coding. The ON channel codes for increasing illumination with increased firing rates of neurons, while the OFF channel codes for decreasing illumination levels, but also with increased firing rates. These anatomically and neurochemically distinct information routes provide excellent sensitivity for detecting small differences in illumination level.

For the photopic cone system, a universal feature of the centre-surround organization at each level is that these RFs comes in two types, found in adjacent cells: some cells have excitatory centres and inhibitory surrounds, while others are organized in exactly the opposite manner (Figure 5B,C). A good stimulus for the first type is a small object brighter than the local background (the so-called ON channel), while an object darker than the background is best at causing the inhibitory-centre cells to fire nerve impulses (OFF channel). The two pathways originate in ON- and OFF-centre bipolar cells, where the direct cone connection produces the centre response and an indirect pathway from horizontal cells, the surround. The significance of this complementary arrangement was unclear until the elegant experiments of Schiller's group (Schiller, 1992). The ON pathway may be selectively desensitized at the inputs to ON bipolars (their glutamate receptor subtype can be selectively blocked by a chemical injected into the retina, APB). APB has little effect on the OFF bipolars. Recordings at subsequent levels

revealed that the ON and OFF pathways remain largely separate up to the cortex, where they combine on cortical cells. Monkeys trained to do brightness discrimination tasks and then given retinal injections of APB were seriously impaired for tasks involving increases in brightness, but unaffected for those incorporating intensity decrements.

This points to primates having two separate subsystems for coding brightness, one signalling for objects brighter than the mean local background with an increase in nerve impulse frequency, the other for objects that are darker, also signalling with an increased impulse frequency. Why have two subsystems evolved when brightness discrimination could be done by just one? If the neurons had a significant background discharge, both increments and decrements could be signalled directly as modulations of this background level. As discussed above, the centre-surround antagonistic design suppresses background impulse firing to a level quite close to zero, so that decrements cannot in fact be signalled effectively. The solution is to invert the OFF signal so that decrements are coded as *increases* in firing frequency, using a separate OFF channel for this purpose (Schiller, 1992).

IV. LIGHT EFFECTS ON PHYSIOLOGY

A. Extraretinal Effects of Light

By contrast with most other vertebrates, mammals lack organized photoreceptors outside the retinae linked directly to behavioural regulation (Nelson and Zucker, 1981). In birds, for example, extraretinal photoreceptors (in the brain) are involved in synchronization of daily rhythms and measurement of daylength in relation to seasonal reproductive phenomena (Menaker and Underwood, 1995). The lack of similar photoreceptors in mammals does not, however, imply a lack of effects of somatic light exposure on human physiology. In fact, such effects are well-documented and have important implications for human health, relating to problems arising from both under- and over-exposure of the body to light. These extraretinal effects are summarized in this section.

1. Phototoxic and photoallergic effects

Synopsis: UV light can have burning and tanning effects on exposed skin surfaces, and can promote the development of skin cancers, depending on levels of screening skin pigmentation. Light (especially UV light) can also induce toxic or allergic responses in sensitive individuals even at low intensities. These responses can be severe in people exposed to environmental or medicinal chemicals which act to sensitize them to light. Among these are commonly used antibiotics and tranquilizers, as well as chemicals in common household products.

Two physiological effects of extraretinal light that are familiar to most people are the burning and tanning responses of the skin, which are phototoxic responses (damage not mediated by changes in immune system function). These processes depend on direct exposure of skin to ultraviolet light primarily in the UVB range, but also including less pronounced effects in the UVA range. The intensity of the erythemal (reddening) response of the skin to UVB radiation depends on several factors: the amount of UVB light reaching the skin surface, the length of exposure, and the degree of pigmentation in the skin. Skin pigmentation reduces the erythemal response and the "tanning" of the skin, both of which occur in two stages. Tanning is a complex protective response which includes darkening of skin pigmentation and thickening of the external skin layers. The combined effect is to reduce subsequent sensitivity of the skin to further UVB radiation exposure.

Prolonged exposure, high UVB content or fair skin promote more extreme responses, including burning of skin tissues to varying degrees, skin thickening and premature aging of skin. In addition to the immediate discomforts or dangers of the burn (depending on the severity), sunburns early in life appear to predispose skin to develop cancerous growths (melanomas or non-melanoma cancers) later in life. These effects appear to result in part from the action of UV light in damaging cellular DNA and inhibiting DNA repair responses (Urbach *et al,* 1974).

There is a strong correlation between degree of skin exposure to sun and the development of skin cancers within a population that is relatively homogeneous in terms of skin colour and genetic makeup (Moan and Dahlback, 1993). Across more diverse populations, the correlations are more complex (Weinstock, 1993). There is a strong negative correlation between the occurrence of most forms of skin cancer and degree of skin pigmentation. The highest risk

groups are fair-skinned individuals, originally from northern regions, who have little capacity for gradual tanning. In other, so-called "sun-resistant" people, the potential for tanning appears to afford some protection against subsequent UV exposure. A recent epidemiologic study in Washington state indicated that melanoma risk was unrelated to occupational or other exposure in adulthood, but early childhood sun exposure was an important risk factor in those with little ability to tan. By contrast, substantial early sun exposure appeared to afford protection against melanoma occurrence in adulthood among those reporting moderate or dark tanning response to chronic sun exposure (White *et al,* 1994).

Although regular occupational exposure to UV light is an important risk factor for some skin cancers, brief, intense exposures may be more potent melanoma-inducing events. Exposure during vacations or summer recreational activities appears to be particularly risky for those fair-skinned people not normally exposed to much sunlight (Setlow and Woodhead, 1994). In addition, there is a tendency for early, intense exposures to produce higher frequencies of cancer in otherwise similar populations (Weinstock, 1993).

Although UVA light is less effective than UVB light (by 1000-fold on average) as a tanning agent, UVA does contribute to erythema and tanning in natural sunlight. In addition, UVA wavelengths are those used most often in tanning equipment, and they are typically found in light from commonly used (especially unshielded) fluorescent sources (Thorington, 1985). In addition, ordinary window glass, which effectively screens out most UVB wavelengths, allows for the passage of some UVA light into homes and workplaces (Diffey, 1990). However, the levels of UVA light penetrating through windows and the very limited access this light has to the skin of home occupants ensures that it is improbable that it could add to the risk of skin cancer, nor provide any significant tanning opportunity.

There is growing evidence that the incidence of melanomas, which are the most dangerous forms of skin cancer, is increasing in the population because of the use of sunscreens which are selective for UVB radiation (Setlow and Woodhead, 1994). Because UVB sunscreens eliminate most erythemal activity, people are encouraged to lengthen sun exposure and therefore exposure to UVA light. There is evidence, however, that UVA light (320-400 nm) is actually very effective in inducing melanoma formation, especially in response to episodic exposure of untanned skin to sunlight (Setlow and Woodhead, 1994). By contrast, carcinoma incidence appears to reflect the total duration of longterm exposure to UVB wavelengths.

Photoallergic responses are pathological responses of the skin which are considered to result from acquisition of altered photoreactivity as a result of antigen-antibody or cell-mediated hypersensitivity (Epstein, 1974). A variety of pathological skin changes may be induced by sunlight exposure, but these are strongly potentiated by the presence of photosensitizers. These may be endogenously produced, such as porphyrins, or may be of exogenous origin. The exogenous sensitizers may act topically, such as some cosmetics and coal tar derivatives, or they may act systemically following ingestion. Among known ingested and topical photosensitizers are antibacterial agents (sulfonamides, tetracycline), phenothiazene tranquilizers such as chlorpromazine, whiteners used in many household products, sunscreens (PABA), and some fragrances, among others (Epstein, 1974; Harber *et al,* 1985). Psoralens are particularly potent photosensitizers which are commonly administered when increased responses to UV light are required as part of UV phototherapy.

Most of the responses to light induced by photosensitizers have the character of phototoxic responses, but some immune system-mediated (photoallergic) responses may also occur. Most of these effects are the result of UVA exposure, but some effects may also result from short wavelength visible light in particularly sensitive individuals. Because indoor fluorescent light sources typically include some UVA wavelengths, and these wavelengths penetrate ordinary window glass, susceptible individuals may be affected by ordinary interior lighting, as well as being strongly affected by sunlight outdoors (Harber *et al,* 1985).

2. Vitamin D_3

Synopsis: Vitamin D_3 is synthesized by the body in response to UVB light exposure and is ingested in some fortified foods, typically dairy products. It plays a crucial role in allowing adequate absorption of dietary calcium and therefore normal formation of bones and teeth during development, and maintenance of healthy bones in adulthood. Lack of adequate sunlight exposure may put some populations at risk of developing vitamin D_3 deficiencies. In particular, individuals with the following characteristics are likely to be at highest risk: elderly, housebound, having dark skin pigmentation, eating poor diets, and living in inner cities with high pollution levels.

Rickets is a serious crippling disease of childhood associated with poverty and malnutrition, and characterized by bone deformities, dental abnormalities and tooth loss. It results in severe retardation and stunting of development, and in women with childhood rickets, results in pelvic abnormalities that lead to increased incidence of maternal and infant deaths. It was a major scourge in cities like London during the Industrial Revolution, but its cause was unknown. In the early 19th century, it was noted that children spending time outdoors and in sunlight were less prone to develop rickets, but it was not until late in that century and into the 20th century before anyone took serious note of the correlation. In the first part of the 20th century, the evidence accumulated that sunshine or exposure to light from some types of lamps could prevent or cure rickets. It was also found that some foods could prevent the development of rickets; in particular, a fat-soluble vitamin in cod-liver oil was identified as a cure, and was named vitamin D (Holick, 1985; Holick *et al,* 1982).

Subsequent research demonstrated that the curative substances ingested in cod-liver oil or produced in humans in response to light were the same. Cod-liver oil was found to be rich in vitamin D, and it was also established as a physiological product of the conversion of 7-dehydrocholesterol by sunlight in the epidermis of mammalian skin. Because it is produced physiologically, some authors refer to vitamin D as a hormone (cholecalciferol). The conversion depends on exposure of skin to light wavelengths in a restricted portion of the UVB range (290-315 nm, with greatest sensitivity around 295 nm). UVB light causes the formation of previtamin D_3; it is isomerized in a temperature-dependent manner to vitamin D_3 (in about three days), which is then transported into the circulation. The critical physiological action of vitamin D_3 is in facilitating the absorption of dietary calcium, thereby allowing for normal formation of bones and teeth during development, as well as their maintenance in adulthood.

Although vitamin D is now added to milk in North America, this does not necessarily make environmental exposure to sunlight irrelevant. Dietary intake of vitamin D may be quite inadequate in some populations, therefore causing reliance on physiological production to maintain normal levels. Because of the characteristic absorption of UV light in the range of 290-320 nm by melanin in skin, heavily pigmented individuals require longer exposures to produce equal amounts of D_3 (Holick, 1985). Populations of people at high risk for rickets in North America are therefore those with heaviest skin pigmentation; namely, populations with African, Hispanic, Asian and First Nations ancestry. The risk appears to be increased in the inner-city cores of metropolitan areas. In these regions, there may be limited access to outdoor illumination (especially during winter months in the northern U.S.A. and throughout Canada), and high levels of air pollution (which blocks UV light transmission) which together would reduce access to the critical UV wavelengths. In addition, poor diets (lacking sufficient calcium and vitamin D content), and skin pigmentation may combine to further prevent adequate production or consumption of this critical vitamin in both children and adults. The apparent re-emergence of rickets in inner-city ghettos in the US has been associated with these factors (Rudolf *et al,* 1980).

Other populations at risk for malabsorption of calcium as a result of insufficient vitamin D levels include the elderly, especially in northern regions. One study reported that 10% of the general population of ambulatory elderly in Boston may be vitamin D-deficient, while 21% of the elderly patients admitted to Massachusetts General Hospital with hip fractures were found to be deficient (Neer, 1985), implicating demineralization from vitamin D deficiency as contributing to the fragility of bones in the elderly. Another study found that elderly, institutionalized women with Alzheimer's disease in Britain have lower vitamin D levels than controls, who were also institutionalized with various longterm mental disorders (Martyn *et al,* 1989). The authors attributed the difference to likely differences in time spent outdoors in the two groups.

The observation that long-term institutionalized elderly patients have abnormally low vitamin D levels has been replicated (Lamberg-Allardt, 1984). Seasonal variations in vitamin D levels were documented in England and Finland among both young and elderly people that were outdoors more in the summer months (Lester *et al,* 1977; Lamberg-Allardt, 1984), but long-term institutionalized elderly who got little time outdoors at any season showed very little seasonal variation in their low vitamin D levels (Lamberg-Allardt, 1984). A lack of sufficient dietary intake of vitamin D is also characteristic of elderly institutionalized patients (Lamberg-Allardt, 1984). Factors that could contribute to insufficient vitamin D levels in the elderly, and therefore to the potential for loss of calcium from bones, include institutionalization, lack of time outdoors (especially in winter), living in an inner-city neighbourhood, poor nutrition and skin pigmentation.

In one detailed metabolic study, healthy, young, Caucasian men were screened from all UV light below 380 nm. Only a few weeks of living under such deprivation were sufficient to cause depletion of vitamin D stores, and, consequently, insufficient intestinal absorption of calcium (Davies, 1985). It seems probable, therefore, that other, less-healthy populations with inadequate diets would be even more sensitive to reduced UVB exposure in terms of their ability to maintain adequate calcium balance. There is, therefore, reason for concern that the especially susceptible populations identified above be exposed to adequate sunlight to maintain appropriate vitamin D levels.

3. Neonatal jaundice

Synopsis: One of the few documented physiological effects on humans of light in the visible part of the spectrum that is not mediated by the retina is the breakdown of excess circulating bilirubin (a poisonous product of haemoglobin metabolism). Neonates often have immature livers that are incapable of fully detoxifying accumulating bilirubin, and sustained exposure of the skin to bright light, especially in the blue range, can accelerate the process and rid the body of excess bilirubin.

A large number of full-term infants have insufficient liver enzyme function to break down and allow the excretion of bilirubin, a toxic product of haemoglobin metabolism, leading to neonatal jaundice (hyperbilirubinemia). Prematurity of birth, low birth weight and other factors can further increase the incidence and severity of neonatal jaundice. High bilirubin levels in the circulation lead to a jaundiced appearance and have the potential for producing permanent brain damage and even death. Exchange transfusion of blood and other invasive methods were once used to treat hyperbilirubinemia, but the accidental observation that infants in a nursery that were exposed to sunlight improved rapidly suggested a role for environmental light in treating neonatal jaundice (McDonagh, 1985). This led to the idea of using artificial light to treat hyperbilirubinemia. Light, especially blue light, was found to be absorbed by the bilirubin molecule and to have several effects on it, including altering its molecular structure to a less toxic form and accelerating its excretion from the body (McDonagh, 1985).

It is now a routine treatment for jaundiced neonates to be exposed to visible light over as large a skin surface as possible, while protecting the eyes. This approach alone allows for rapid and effective treatment of neonatal jaundice in most infants, with no significant side effects. There are two concerns with this approach relative to the natural light exposure patterns experienced by untreated infants. One is that gauze eye-coverings may only somewhat reduce light exposure and still leave premature infants' eyes continuously exposed to much higher light intensities than they could experience in utero even intermittently (Daniels, 1974). A complementary concern is that when effective light-screening masks are used on full-term infants with jaundice, this essential protection for the eyes also ensures that infants are exposed to reduced retinal illumination during a developmental period when light exposure is normal and visual experience may be developmentally important.

Excess light exposure, independently of treatment for jaundice, is frequently encountered in neonatal care units, especially intensive care units, where infants are often exposed to continuous bright illumination. Such illumination facilitates care at all hours of the day and night and allows for closer observation of infants, including monitoring colour changes that might signal jaundice or respiratory difficulties. On the other hand, such illumination patterns may have other negative effects on the developing visual and endocrine systems of neonates, that have not been well studied. There is some evidence to support concern for the effects on visual system development of continuous high levels of illumination in neonatal facilities (Abramov, 1985).

The portion of the light spectrum that is effective for treatment of neonatal jaundice is centred on the blue region, around 450 nm, so UV light is routinely screened out, since lengthy exposure of a neonate to UV light would be harmful and it would play no role in treatment. The fact that bilirubin absorbs visible light and is detoxified by it, makes treatment of neonatal jaundice relatively simple, and it represents one of the few known extraretinal effects of visible light on human physiology (McDonagh, 1985).

4. Infectious diseases and immune function

Synopsis: Ultraviolet light exposure can kill bacteria and other microorganisms. At high doses, its antimicrobial actions are used in sterilizing equipment;low doses from natural or artificial illumination might also affect microbes in dwellings and offices. UV light can also affect the immune system. UV-induced immune suppression may be useful in treating autoimmune diseases, but may also be harmful in immunosuppressed individuals or in combination with the DNA-damaging effects of UV light.

Before the development of antibiotics, phototherapy was in common use as a treatment for infectious diseases. Niels Finsen won the Nobel Prize in Medicine in 1903 for his development of an effective method for applying UV light to cure skin tuberculosis (lupus vulgaris). During the 1920's and 1930's UV phototherapy was used commonly for the treatment of several forms of tuberculosis and of streptococcal skin infections (Daniels, 1974). These cumbersome, but effective methods, were rapidly supplanted by the development of broadly effective antibiotic and other antimicrobial agents, which dominate medical treatment of infection today. The recently growing awareness of and concern about the development of antibiotic-resistant strains of many bacteria, however, demonstrates that the pharmacological approach is also not without problems (Travis, 1994). The therapeutic use of UV light, often in combination with photosensitizers such as psoralens, remains an active clinical approach to the treatment of acne, psoriasis, vitiligo (a depigmenting disorder) and some fungal infections (Parrish *et al,* 1985). Low doses of UV from natural or artificial lighting might have some useful antimicrobial effects in buildings.

There is a relatively inaccessible older Russian literature on the general health-promoting and tonic effects of UV light therapy on humans, summarized, for example, by Daniels (1974). The general theme is that features of human physiology such as exercise tolerance, resistance to infectious diseases, and "general fitness" can be improved by UV light therapy. The wavelengths responsible and mechanisms involved in these effects were not defined, nor is it clear whether these claims are reliable or valid. Although specific mechanisms were not described, the assumption was that UV light might act by improving immune system function. Whether any of these claimed effects might be mediated instead by correction of undetected vitamin D deficiencies (see IV.A.2) was not assessed, and should be considered as an alternative explanation. More recent reviews have found little evidence to support a role for winter UV supplementation in reducing infectious diseases (Morison, 1983).

There is, however, considerable evidence that UV light can alter immune system responsiveness in both rodent models and in humans (Morison, 1985; Noonan and De Fabo, 1994). The effects of UV exposure include suppression of some delayed immune responses to antigens in animals (Bergstresser *et al,* 1983; Noonan and De Fabo, 1994) and reduced skin

immune responses in humans with known sensitivities to an antigen (Morison, 1983). The immune-suppressing properties of UV light were first identified with high-intensity UV light using artificial sources, but the evidence now indicates that the spectral sensitivity for these responses is in the UVB range and that the intensities available in ordinary sunlight are sufficient to demonstrate immune suppression (Noonan and De Fabo, 1994).

Although the evidence that UV radiation can affect immune system function is substantial, the underlying mechanisms are not well understood. One proposed mechanism involves a small molecule (urocanic acid) which is chemically related to histamine, and responds to UVB light with isomerization from the *trans* to *cis* conformation. The *cis* form appears to regulate the production by skin fibroblasts of immune-suppressing agents (Noonan and De Fabo, 1994). This response is independent of pigmentation, probably because the urocanic acid is located in the stratum corneum, above the layer in which skin pigment molecules are found. Surprisingly, in animal models the immune-suppressing response to UVB is not attenuated by most common sunblocks, such as PABA, which effectively prevent erythemal responses. The reason for this is unknown but it may be because sunblocks do not screen UV light completely, and laboratory UV sources can generate very high intensities. The available data are still limited, but the evidence suggests that prolonged UV exposure of humans under the protection of sunblock may not be entirely innocuous with respect to the immune system (Noonan and De Fabo, 1994).

The physiological consequences of UV-induced immune system changes are not well understood. Suppression of immune system activity may promote development of some skin tumours, and may therefore interact with other effects of UVB light on DNA and on pigment cells in causing serious human skin cancers (see IV.A.1). Another important consideration is that individuals may be more or less susceptible to immune system suppression by UV light, and this sensitivity may depend on genetic characteristics, as has been demonstrated in some inbred strains of mice (Noonan and De Fabo, 1994). Some populations may therefore be at higher risk, and already immuno-suppressed people may also suffer more harmful effects of even partial additional suppression. It remains to be established how UVB-induced suppression interacts with immune systems that have been rendered hypo- or hyper-responsive by other agents.

In addition to the potential risks of tumour development posed by immune system suppression in some people, this effect of UVB light may also prove therapeutically useful in other circumstances. It may, for example, find application in suppressing unwanted immune responses, such as those occurring in autoimmune diseases (e.g., multiple sclerosis), and may, therefore, have therapeutic potential for some patients (Morison, 1985).

B. Retinally Mediated Effects of Light

1. Circadian rhythm entrainment

Synopsis: One of the most important non-visual effects of daily light exposure mediated by the eyes is the synchronization of the body's daily rhythms to local time cues. Disturbance in this process can give rise to symptoms of jet lag or to sleep and eating disorders, such as those associated with rotating shift work schedules. Light information conveyed from the retina to the suprachiasmatic nucleus (SCN) in the hypothalamus is responsible for this process. This system appears to require quite bright light intensities to operate, although it remains unclear whether humans are also somewhat responsive to ordinary room lighting, or whether very bright light similar to that available outdoors is required in order to remain normally synchronized.

Daily (circadian) rhythms pervade human physiology and behaviour (Aschoff and Wever, 1981; Aschoff, 1984). Behaviourally, they are evident in our cycles of sleep and wakefulness, our daily meal patterns and our work habits. Physiologically, they are found in every aspect of endocrine, neural and immune system function. These daily rhythms are generated by internal timing devices or pacemakers, the principal one of which has been localized to the suprachiasmatic nuclei (SCN) of the hypothalamus (Hastings *et al,* 1991; Harrington *et al,* 1994). The rhythms generated in the SCN may be influenced by a variety of social and behavioural cues (Mistlberger and Rusak, 1994). But in humans, as in other species, the daily cycle of light exposure is the critical synchronizing (entraining) cue that aligns our rhythms with the changing cycles of day and night (Wever, 1989), a process accomplished by *phase shifts* of the pacemaker system. The effects of light on the circadian system can, therefore, be felt in virtually every aspect of human physiology, including regulation of body temperature, control of endocrine activity, and immunological responses to antigens. Similarly, through its effects on the circadian system, light influences many behavioural functions, from the timing of sleep and waking, to cognitive performance and mood (Monk, 1994).

The neural mechanisms by which light information is conveyed to the SCN have been studied for over 20 years and are well characterized (Hastings *et al,* 1991; Harrington *et al,* 1994). Retinal ganglion cells project directly to the SCN and convey information related to the intensity of environmental light via the retinohypothalamic tract (RHT) (Rusak and Boulos, 1981; Rusak, 1989). In addition, the SCN receive photic information related to entrainment from cells in the lateral geniculate nuclei which themselves receive direct retinal input (Harrington and Rusak, 1986). The spectrum of wavelengths of light responsible for entrainment in rodents closely matches the rhodopsin sensitivity curve (Nelson and Takahashi, 1991a), but it is unlikely that classical rod receptors are responsible for mediating light effects on rhythms. Some mouse strains which undergo programmed retinal degeneration during development, resulting in the loss of all rods and many cones from their retinas, still show normal circadian rhythm entrainment (Foster *et al,* 1991); see IV.B.3. Similarly, neurotoxic treatments that damage the visual system still preserve retinal projections to the SCN intact and permit normal entrainment (Pickard *et al,* 1982). These results indicate that a novel class of retinal photoreceptor may be responsible for daily rhythm entrainment. The receptor remains to be identified and characterized, but it may be a modified cone structure using a photopigment molecule with spectral characteristics similar to those of rhodopsin.

The mammalian circadian rhythm entrainment mechanism is remarkably insensitive to light. Photic responses of SCN cells and phase shifting effects on circadian rhythms are both elicited at only quite bright light intensities in nocturnal rodents, as compared to the absolute thresholds for light detection by the retina and for visual responses by parts of the classical visual system (Nelson and Takahashi, 1991b; Harrington *et al,* 1994). Functionally, this high threshold ensures that very dim light which is clearly visible (to our visual system), such as starlight and moonlight, cannot affect the circadian pacemaker system. The sensitivity of the pacemaker in nocturnal rodents is tuned to a narrow range of light intensities typically encountered around dawn and dusk (Groos and Meijer, 1985; Nelson and Takahashi, 1991a). These daily phases are the ones at which such organisms receive their entraining information from lighting cycles in the natural environment (DeCoursey and Menon, 1991).

For diurnal species, the story is much less clear. Neurophysiological studies of one diurnal rodent species suggested that the thresholds for SCN cells to respond to light in that species were about 100-fold higher than for nocturnal rodents (Meijer *et al,* 1989). Other data also suggest that diurnal and nocturnal mammals handle entrainment information from the environment differently (Abe *et al,* 1995), but considerably more work is required to characterize these apparent differences.

For humans, the story is still less clear, since the study of human entrainment to light stimuli is still in its infancy. The short history of this area has been characterized by conflict about the interpretation of the few data available (Beersma and Daan, 1993; Kronauer *et al,* 1993). Although early studies of human circadian rhythms indicated that light did affect circadian rhythms in humans, as in other species (Wever, 1989), the relative importance of photic relative to non-photic cues (social factors in particular) was less clear. Light appeared to act as a relatively weak cue for the circadian system, compared to the potent effects of non-photic cues on humans and of light on other species (Aschoff *et al,* 1971). More recent studies have shown potent phase-shifting effects of light on human circadian rhythms, predicated on the use of brighter, longer or repeated light pulses (Czeisler *et al,* 1986; Wever, 1989; Honma *et al,* 1987; Minors *et al,* 1991). It is now established that light by itself can strongly influence the circadian systems of humans when other factors are held constant, but it is also clear that non-photic factors are important. It is not obvious which factors will dominate if photic and non-photic cues are placed in competition (Eastman *et al,* 1995).

There appears to be general agreement that humans require much brighter light intensities than nocturnal rodents to show phase-shifting responses to light. It is still unclear, however, whether humans are responsive only to very bright light intensities, characteristic of normal sunlight (2×10^3-10^4 lux), or whether some responses are also obtained with dimmer illumination intensities typical of most indoor work and home environments (e.g., 5×10^2 lux). While brighter light is assumed to be more effective, it remains unclear whether there is a high threshold for phase-shifting effects or whether dimmer light simply has modest effects that are difficult to detect.

One difficulty is that much of our knowledge of human light sensitivity is not derived from direct studies of human entrainment responses to light (which are technically very difficult to do). Rather, it stems largely from related studies of melatonin suppression by light (see IV.B.2) and from studies of sensitivity to the therapeutic effects of light on seasonally depressed people (see V.C). One reason for making the assumption that these effects are related to

circadian rhythm entrainment is that melatonin suppression in mammals is known to be mediated by light information reaching the SCN pacemaker in the hypothalamus via its retinal innervation (see IV.B.2), the same site at which circadian entrainment information is received and processed. Nevertheless, neither of these approaches necessarily addresses the issue of the threshold for circadian rhythm entrainment. In hamsters, for example, the threshold for melatonin suppression is 25 times higher than the already high threshold for circadian rhythm phase shifting (Nelson and Takahashi, 1991b). It is, therefore, not legitimate to assume that the characteristics of light that affect melatonin secretion are identical to those that affect circadian rhythms.

2. Pineal gland regulation

Synopsis: The pineal is an important gland found in the brain that secretes the hormone melatonin. Melatonin has been implicated in helping to regulate daily rhythms but may also influence diverse processes such as sleep, body temperature, and tumour development. Its secretion is regulated strictly by the circadian clock in the SCN and by retinally perceived light, so that it occurs only during the night. Although very bright light is required to suppress melatonin secretion completely, ordinary room lighting may have partial effects.

The pineal gland is a unique neuroendocrine transducer in the vertebrate brain, the function of which has been the subject of speculation for centuries (Arendt, 1995; Rusak, 1982). During the last thirty years, it has become clear that the pineal gland is closely tied to the vertebrate circadian system, as well as serving a variety of other functions (Underwood and Goldman, 1987; Arendt, 1995; Rusak, 1982). The pineal gland secretes the indoleamine hormone melatonin and does so only during the night. The secretion of melatonin can be seen as a signal related to the duration of the night phase which is transmitted to a variety of central neural structures as well as to numerous peripheral organs (Arendt, 1995; Reiter, 1991). The photic information used by the pineal gland to synchronize its secretory activity to the external day/night cycle is derived in mammals exclusively from the retina, and is conveyed first to the SCN (Klein and Moore, 1979; Moore and Klein, 1974; Rusak and Morin, 1976). From the SCN a complex pathway arises by which the information is conveyed via the superior cervical ganglion of the sympathetic nervous system to trigger the pineal gland's secretory activity during the night time. This mechanism is not shared by other vertebrates. In pigeons, and probably other birds, the eyes appear to contribute little to the photic regulation of pineal gland function, and extraretinal photoreception (in the brain or the pineal gland itself) is the major route for entrainment of melatonin secretion (Hasegawa *et al,* 1994).

Since melatonin secretion by the mammalian pineal gland is regulated by the central circadian pacemaker in the SCN, light that shifts the circadian pacemaker also shifts the pineal melatonin rhythm (Illnerová and Vanecek, 1987). In addition to this indirect effect on melatonin levels, light acting via retinal projections to the SCN also has an immediate effect in suppressing melatonin levels. Bright light exposure (>2.5 x 10^3 lux) at any time during the night when melatonin levels are elevated causes a rapid decline in melatonin levels by inhibiting the enzymatic machinery that underlies melatonin production (Lewy *et al,* 1986; Petterborg *et al,* 1991). Recent studies have also observed that bright nocturnal illumination increases body temperature in humans (Badia *et al,* 1991; Myers and Badia, 1993). Since melatonin adminstration can depress temperature (Cagnacci *et al,* 1992; Cagnacci *et al,* 1994), it is possible

that the reduction of melatonin levels by light is the mechanism by which light elevates temperature. A recent study has supported this interpretation by showing that exogenous melatonin treatment can prevent the light-induced elevation of body temperature (Cagnacci *et al,* 1993).

Ordinary room lighting was once considered to be ineffective in inhibiting melatonin secretion; indeed, it was the use of such light intensities in early studies that originally led to the erroneous conclusion that human melatonin secretion is not light sensitive (Lewy *et al,* 1980). However, more recent detailed analyses of how light affects human melatonin secretion have indicated that much dimmer lights than previously supposed can depress melatonin levels. Even illumination as dim as ordinary room light (3.5×10^2 lux) can cause a partial reduction in melatonin levels. The response appears to be continuously graded, with no apparent intensity threshold (McIntyre *et al,* 1989). These recent results suggest that exposure to ordinary indoor lighting at night might alter the levels and patterns of melatonin secretion. Artificial lighting, which allows humans to extend their activities well into the period of natural darkness, may, therefore, have effects on melatonin secretion. In addition, nocturnal shift workers must be exposed to similar or even higher light intensities in the workplace, thereby triggering changes in their melatonin secretion patterns, with unknown physiological consequences for workers.

The physiological significance of lighting effects on human melatonin secretion remain uncertain in part because the normal physiological roles of melatonin are still poorly understood. It is clear that melatonin has dramatic effects on the reproductive status of rodents (Underwood and Goldman, 1987) and plays a role in the circadian systems of mammals as well as other vertebrates (Armstrong, 1989; Cassone and Menaker, 1984; Rusak, 1982; Rusak and Yu, 1993). But there are a number of other potentially important effects that have been much less thoroughly studied. For example, melatonin is reported to be an effective scavenger of free radicals, and might therefore have anti-cancer effects (Reiter *et al,* 1994; Tan *et al,* 1993). Low frequency electromagnetic fields have been linked in some studies to cancer development (Wilson *et al,* 1992), and have been reported in other studies to inhibit pineal function (Reiter, 1992), providing a second theoretical justification for suggesting a linkage between melatonin and cancer. Pineal gland function has also been linked to aging: transplanting pineal glands from young to old mice is reported to increase longevity and slow development of some signs of aging (Armstrong and Redman, 1991; Pierpaoli and Regelson, 1994; Pierpaoli and Lesnikov, 1994; Reiter *et al,* 1994; Tan *et al,* 1993). While some of these linkages are preliminary or based on indirect evidence, they suggest that the physiological consequences of altering melatonin secretion patterns might be quite profound and should receive further experimental attention.

3. Spectral sensitivity of light effects on physiology

Synopsis: The sensitivity curves for light effects on daily rhythms and on pineal function both resemble the sensitivity curve for rhodopsin, peaking in the blue-green part of the visual spectrum. In rodents, whose smaller eyes allow for UV transmission, UV light can affect both processes, but this is unlikely in humans. The photoreceptors responsible for these processes remain unidentified, although classical rod photoreceptors do not appear to be relevant in rodents.

Light effects on circadian and pineal gland function appear to be mediated by a retinal photoreceptor with sensitivity peaking in the blue-green part of the spectrum, and with an action spectrum similar to that of the rod pigment rhodopsin (Takahashi *et al,* 1984) Despite this similarity, the classical rod photoreceptors do not appear to mediate light effects on circadian rhythms in some rodents. There are mouse strains which bear a mutation (*rd*) which causes animals to undergo progressive retinal degeneration during early development; eventually there is a complete loss of rod photoreceptors. Despite this loss, in one mouse strain neither the threshold nor the spectrum of sensitivity to the circadian phase shifting effects of light are altered by this progressive loss (Foster *et al,* 1991). On the other hand, a second mouse strain that bears the same gene mutation (against a different genetic background) shows a very considerable loss of retinal phase-shifting sensitivity to light (Yoshimura *et al,* 1994).

These results imply that the genetic background against which the *rd* gene is expressed is important to its consequences. It may be that in one strain the specialized photoreceptors involved in rhythm entrainment are also affected by the gene's expression, while they are not susceptible to its effects in the other strain. The suggestion that there are separate photoreceptive systems for light effects on rhythms and for classical visual functions is reinforced by studies of prenatal damage to the visual system by treatment with large doses of monosodium glutamate. This treatment severely damages the classical visual system and disrupts vision, but it does not disrupt projections to the SCN and has no effect on circadian rhythm entrainment by light (Pickard *et al,* 1982; Edelstein *et al,* 1995). The relevant photoreceptors have yet to be identified, but they may be a distinctive class of cone-type photoreceptors with an unidentified pigment molecule.

A few studies have examined the role of UV light in affecting circadian rhythms and pineal gland function in rodents. There is considerable evidence that exposure to UV light can suppress melatonin secretion in rats, hamsters and other rodents, and can phase shift their circadian rhythms (Brainard *et al,* 1994; Podolin *et al,* 1987). UV light has a broad array of extraretinal physiological effects in humans and other mammals (see IV.A), but the effects on rhythms and the pineal gland appear to be mediated via the retina and not by extraretinal receptors. Retinal pigments are sensitive to UV light and the lenses of rodent eyes transmit light down to 300 nm, thereby allowing for these physiological effects of UV. In larger eyes, such wavelengths are strongly filtered, and increasing lens pigmentation during aging further attenuates transmission of wavelengths <400 nm to the retina in humans. It appears that lenses in children and even young adults can transmit significant amounts of light in the UVA range (Brainard *et al,* 1994), but the physiological significance of such transmission in humans remains to be established.

4. Role of pineal melatonin

Synopsis: Melatonin has been shown to play a role in the circadian organization of several vertebrate species, and can also affect sleep and circadian rhythms in humans. Pharmacological doses of melatonin can help entrain or phase shift human rhythms, and may be clinically useful in a number of situations. A high-affinity receptor for melatonin has been identified and cloned, opening the way for development of even more effective drugs for these purposes.

The full range of melatonin effects on vertebrate physiology has not yet been established, but some functions have been studied extensively. In seasonally breeding species, the pattern of melatonin secretion is a critical signal conveying information about night length, and therefore time of year, to the rest of the neuroendocrine system (Underwood and Goldman, 1987). The changing pattern of melatonin secretion across the year is therefore responsible for initiating the neuroendocrine changes that result in triggering or blocking seasonal reproductive activity and a host of related metabolic, endocrine and behavioural changes (Arendt, 1995). In addition, in a number of species, including humans, melatonin has been implicated in the regulation of puberty onset (Cavallo, 1993; Rivest *et al,* 1986).

Melatonin has also been shown to have a profound effect on the circadian system. In birds, pineal and hypothalamic mechanisms act together to regulate circadian rhythms (Cassone and Menaker, 1984; Rusak, 1982) The role of melatonin rhythmicity in birds appears to be to act on a complex of pacemaker components, especially in the eyes and the SCN, to help synchronize their oscillatory processes. In mammals, melatonin appears also to act on the SCN pacemaker to affect circadian rhythms (Rusak and Yu, 1993; Cassone *et al,* 1986), probably through a high-affinity melatonin receptor which has been identified in several brain structures, including the SCN of humans and other species (Weaver *et al,* 1993; Weaver *et al,* 1989; Reppert *et al,* 1988).

The apparent regulatory role played by melatonin in the circadian system has prompted the recent development of melatonin and related analogues as potential phase-shifting agents for humans. These have been proposed for use by shift workers, jet-lagged travellers, people with sleep difficulties and others suffering from inadequate or abnormal synchronization of their circadian systems. Some studies have demonstrated at least partial effectiveness in the treatment of rhythm-related disorders (Arendt, 1995). Melatonin has, for example, been used with some success in phase-shifting or entraining some blind subjects who normally have difficulty maintaining synchronization of their circadian rhythms to the external day-night cycle (Sack *et al,* 1991), and in helping travellers adjust to rapid time-zone transitions (Arendt *et al,* 1987). In large doses, melatonin appears to have modest sleep-facilitating properties (Tzischinsky and Lavie, 1994), and while this action may also contribute to its phase-shifting efficacy, it is likely that its effects are mediated in large part by action on the SCN-based circadian pacemaker.

5. Light effects on autonomic nervous system function

Synopsis: Some studies report that brief increases in light exposure can activate parts of the sympathetic nervous system in both rodent models and humans. These effects can be manifest as increased body temperature or blood pressure. Whether they are mediated by neural effects on the autonomic nervous system or by the intermediate steps, such as hormone release, has not been determined.

As mentioned earlier, the mechanism by which light regulates pineal gland function involves effects on the sympathetic innervation of the gland, originating in the superior cervical ganglion, but involving the retinal innervation of the SCN. Recent results suggest that this role of the retinal innervation of the hypothalamus in regulating the pineal gland may be only a part of a more general mechanism by which this pathway regulates a number of autonomic nervous system functions. One report described very broad effects on autonomic system function of exposing rats to light: there was increased sympathetic activation of several visceral organs, along with suppression of activity of the vagal innervation of several organs (Niijima *et al,* 1993). Similarly, light can accelerate heart rate in rats, another marker of sympathetic nervous system activation (Amir, 1992).

Related results in humans also hint at effects of light on autonomic system activity. Studies of humans exposed to lights with different colour temperatures indicate that some wavelengths can affect blood pressure and other indicators of autonomic system stability. One study using light sources ranging in colour temperatures from 3000°K to 7500°K observed increased diastolic blood pressure only in response to exposure to the 7500°K source (Mukae and Sato, 1992). Similarly, a 6700°K source caused a change in stability of heart rate, which was also interpreted as reflecting an increased general level of autonomic system activation. These results imply that exposure of people to short wavelength light (high colour temperature; blue light) may arouse the autonomic nervous system. In particular, the elevation in diastolic blood pressure is suggestive of specific effects on vasomotor function (Mukae and Sato, 1992). Extrapolating from the results on rats, one might speculate that some light exposures may increase sympathetic activation, including adrenal activity, but it remains to be explained by what mechanisms specific wavelengths would have such selective effects (see also V.E).

These observations are quite recent and they need to be confirmed in other empirical studies. In addition, interpretation of such findings will depend on the development of a plausible mechanistic explanation for them. These preliminary results do, however, provide some justification for consideration of the role of room colour and illumination type on human mood and function (see VI.C). They are also consistent with evidence that light can raise human body temperature (Myers and Badia, 1993) (see IV.B.2), and that light has a general arousing effect on humans (see V.E)

6. Effects of blindness

Synopsis: For some people, reduced photic input as a result of visual system damage causes severe difficulties in entraining to environmental cycles. These people appear to lack a surviving retinohypothalamic projection. For others, this projection remains intact, despite the lack of conscious awareness of light, and they may have fewer difficulties with rhythm synchronization. For a third group of blind people, lacking the retinohypothalamic projection, non-photic cues may be able to compensate for the lack of light input, and sleep and rhythm difficulties are minimal.

Blindness in humans is defined with respect to visual contrast sensitivity in various ways in different legal jurisdictions, but about 10% of those who are legally blind also have no conscious light perception (Czeisler *et al,* 1995). Because of the crucial role of light in regulation of circadian rhythms, it might be expected that such blind people would fail to entrain their circadian rhythms to their environmental cycles. Some blind people do, in fact, have serious difficulties with rhythm synchronization (Miles *et al,* 1977; Sack *et al,* 1992). These difficulties give rise to periodic sleep disturbances as their circadian rhythms express non-24 h, free-running periods that cause them to drift in and out of synchrony with external time cues, as if they were undergoing repeated experiences of jet-lag. This is not, however, a universal problem in blind people; some appear to entrain their rhythms normally and with no evidence of regular sleep disturbances.

A recent study (Czeisler *et al,* 1995) investigated whether people who were completely blind as a result of severe ocular abnormalities might still retain normal innervation of the SCN via the RHT, despite a lack of functional input to other visual centres, and even a lack in most cases of detectable electroretinographic (ERG) responses to light. In this study, photic suppression of melatonin during its nocturnal secretory phase (which is known to be mediated via the SCN; see IV.B.2) was assessed in a group of blind subjects. Three blind people with no history of complaints of sleep disorders, and apparently entrained rhythms, showed normal melatonin suppression in response to very bright light pulses (of which they had no conscious perception). By contrast, subjects with a history of sleep disturbances did not show normal melatonin suppression in response to light, indicating a loss of normal retinal innervation of the SCN. Among these people, some had daily rhythms that appeared to be currently entrained, while others did not. The apparently entrained subgroup (but with a history of sleep disturbances) included two who were bilaterally enucleated. Entrainment in these people might be mediated by social and other factors that are less reliable and consistent than light cues as entraining agents.

The findings demonstrate that a RHT/SCN/pineal projection can survive despite severe ocular damage, and they suggest that non-photic cues may be at least partially adequate synchronizing cues for some blind people lacking this pathway, but not for others (Czeisler *et al,* 1995). These results reinforce the conclusion that separate retinal projection routes mediate different photic effects on human physiology and behaviour. They also emphasize the idea that a lack of adequate photic entrainment cues can be very deleterious for some people, but may be less important for others.

C. Summary

This review of the physiological effects of light acting via both extraretinal and retinal routes indicates the great variety and complexity of its physiological effects beyond the phenomena of vision. Light acting through the skin can have beneficial effects on our physiology, as in the production of vitamin D or the breakdown of bilirubin. Its antibiotic activity, especially in the UV range, can be used to treat skin and other infections, and its ability to cause altered immune system function may be useful in treating diseases marked by immune system hyperactivity.

Light can, however, also have harmful effects in triggering erythemal responses, burns, DNA damage and immune system suppression, leading sometimes to the development of malignancies in the skin. Although these actions are most readily caused by high-energy UVB wavelengths, light in the UVA range can have similar, but less potent, effects. In individuals sensitized by environmental agents, disease, ingested drugs or other unknown factors, photoallergic and phototoxic effects of UVA and even visible light can be quite potent and destructive.

By acting as the major synchronizer of daily and seasonal biological rhythms, light can affect virtually all physiological regulatory systems of the body. Altered light exposure can cause specific ecologically relevant changes in the timing of daily rhythms and seasonal cycles. Light can also have rather general effects by influencing neural mechanisms that can alter sympathetic nervous system function. This is most clearly exemplified by the effects of light on pineal gland function, mediated by the sympathetic superior cervical ganglion. Light might, however, also affect other aspects of autonomic nervous system function that have not yet been studied very thoroughly. Light acting probably via retinal innervation of the hypothalamus can affect sympathetically regulated heart rate in rats, and body temperature regulation in humans. Light-regulated alterations in body temperature and autonomic system function might have quite broad consequences for human physiology and behaviour.

Evidence from studies of blind people reinforces the idea that separate retinal projections regulate classical visual effects of light and photic effects mediated via the hypothalamic suprachiasmatic nuclei. Studies of blind people also demonstrate that loss of adequate light input to the photic system can seriously disrupt rhythm entrainment and sleep-wake cyclicity in some people, while others can apparently respond to non-photic cues and achieve rhythm entrainment, although perhaps not as reliably as with photic cues.

Melatonin is an exquisitely light-sensitive hormone, and the spectrum of light effects on physiology is extended by the broad range of physiological and pathological processes in which melatonin has been implicated. Besides its well-documented role in the regulation of seasonal reproductive cycles and reproductive development in some species, melatonin also affects circadian organization and probably a variety of endocrine systems. It has been used with some initial success in the treatment of some circadian-based sleep and rhythm disorders. It has also been proposed to have significant anti-cancer and anti-aging properties that are under current investigation.

The role of light of different wavelengths in each of these processes has been studied to some degree. Visible light acts to cause circadian rhythm entrainment, with peak sensitivity in the blue-green part of the spectrum. Visible light also affects pineal gland function, but in rodents at least, UV light can also affect pineal function. Different portions of the UV spectrum

are most effective for stimulating vitamin D synthesis and producing erythemal and other toxic effects.

The evidence related to phototoxicity implies, however, that "normal" sensitivities to different wavelengths should not be accepted as necessarily universal. Depending on drug status, immune system function and environmental variables, sensitivities may arise to wavelengths that are not normally of any physiological significance. Subpopulations might therefore show photic sensitivities that are not characteristic of the population as a whole. By the same token, different subpopulations might be especially sensitive to the deleterious effects of relative light deprivation. Thus, inner-city populations with pigmented skins in northern regions may suffer from reduced vitamin D synthesis because of low levels of light exposure, particularly in the winter months, while other members of the general population may be unaffected. Disorders of calcium metabolism might arise and be exacerbated by poor diets, old age, lack of time outdoors, and UV-screening pollution levels. The epidemiology of light-related disorders in subpopulations therefore deserves careful analysis in parallel with assessment of overall effects on the general population.

V. Light and Mental Health

A. Seasonal Affective Disorder: Phenomenology and Prevalence

Synopsis: Seasonal affective disorder is a regularly recurring depression associated with the short days of autumn and winter. It includes both typical depressive symptoms and atypical symptoms such as carbohydrate craving and overeating. It appears to be more prevalent as one moves northward away from the equator, and is more common in women than in men. This form of depression is effectively treated in many patients by increasing artificial bright light exposure during the winter months. The etiology of the disorder and the mechanisms of light's therapeutic effects remain to be established.

Many aspects of human behaviour and psychopathology have been reported to show seasonal variation, such as human conception, suicide and psychiatric hospital admissions (Aschoff, 1981; Maes *et al,* 1993; Roenneberg and Aschoff, 1990a; Roenneberg and Aschoff, 1990b; Randall, 1993; Schreiber *et al,* 1993; Souêtre *et al,* 1990; Temte, 1989). Seasonal variation in mood has also been reported, as measured by retrospective and prospective self-reports and questionnaires in large samples (Kasper *et al,* 1989b; Haggag *et al,* 1990; Harris and Dawson-Hughes, 1993), as well as in parental reports of mood in children (Carskadon and Acebo, 1993). These studies typically report that most respondents felt worse in fall/winter, with improvement in spring/summer. Kasper *et al* (1989b) found that changes in social activity, weight, and energy were also associated with the mood changes. Many of these seasonal fluctuations appear to be correlated with daylength; however, since changes in photoperiod and weather variables such as temperature and humidity occur simultaneously, it is difficult to evaluate the independent contributions of light and/or daylength to these patterns.

The role of light in seasonal cycles of clinical depression has been more thoroughly investigated. Seasonal affective disorder (SAD) is a major depressive disorder distinguished by an annual cycle of recurrence, usually in the fall/winter, with remission in the spring. It was first described carefully by Rosenthal *et al* (1984) and accepted in the Diagnostic and Statistical Manual-III-Revised of the American Psychiatric Association (American Psychiatric Association, 1987) as a "seasonal pattern" to characterize recurrent major depression and bipolar disorder. Since its initial description, SAD has been characterized by a number of researchers from many parts of the world, including: Australia (Boyce and Parker, 1988); the U.K. (Thompson and Isaacs, 1988); Switzerland (Wirz-Justice *et al,* 1986); and Iceland (Magnússon and Stefánsson, 1993). The syndrome consists of a major depression usually in winter (ranging in severity from mild to severe), associated with fatigue, hypersomnia, overeating, carbohydrate cravings, as well as many typical symptoms of depression (e.g., feelings of guilt, agitation, depressed mood). Remission in summer may be accompanied by hypomania, although there is disagreement about the frequency of occurrence of hypomania (Blehar and Rosenthal, 1989).

Studies of the prevalence of SAD in the normal population through random mail or telephone surveys in the United States report a rate of 1.4% in Florida to 9.7% in New Hampshire (Kasper *et al,* 1989b; Rosen *et al,* 1990). While a correlation with latitude, and therefore with seasonal change in daylength, is suggested, a recent study of the prevalence rate for Iceland (a country at extreme northern latitudes) did not support the latitude hypothesis (Magnússon and Axelsson, 1993). They found SAD was no more prevalent in Iceland (3.8%)

than in areas in the northern U.S. Differences in diagnostic criteria or population sampling may account for this inconsistency. In terms of prevalence among patients with depression, one study of a consecutive group of patients admitted with major depressive disorder found 15.6% of depressed patients fulfilled the criteria for SAD (Thase, 1986). Most studies find it occurs more frequently in women than men, with women making up 68-75% of cases (Rosen *et al,* 1990; Blehar and Rosenthal, 1989). The age of onset is typically during young adulthood (20s-30s), although children and adolescents have been identified with the disorder (Blehar and Rosenthal, 1989; Thompson and Isaacs, 1988; Lucas, 1991; Rosenthal *et al,* 1986).

A "sub-syndromal" pattern of seasonal variation in mood and related behaviour (termed S-SAD) has also been identified in epidemiologic studies of SAD (Kasper *et al,* 1989a). These individuals experience mild dysfunction and vegetative changes similar to those found in SAD, but do not meet clinical criteria for SAD, nor do they tend to seek treatment for their difficulties. The prevalence of this milder form of seasonal mood disorder was estimated as 13.5%. The identification of this group suggests a continuum of severity in seasonal changes in mood and related behaviours, with normal individuals and SAD patients on each end, and subsyndromal SAD individuals falling in the middle.

A less frequent variant of the typical seasonal pattern of SAD in which patients become depressed in the summer and recover in the fall was also reported (Wehr *et al,* 1987a). The existence of this variant has been confirmed by more recent studies (Magnússon and Stefánsson, 1993; Rosen *et al,* 1990; Boyce and Parker, 1988). Because of this summer pattern, the terms "winter depression" and "summer depression" have been adopted to maintain a distinction between SAD patients with depression in the winter versus summer. Little information about summer depression is available, although there is some speculation that the underlying mechanism may be related to temperature, rather than light (Wehr *et al,* 1987a). A third form of SAD that has been described is a combined type of winter and summer depression (Wehr *et al,* 1987a). SAD in this review refers to winter depression; summer or combined type depression will not be discussed further in this report, given the lack of evidence that these forms are related to light exposure.

The association of SAD with the short daylengths of winter, and the anecdotal remission of SAD symptoms when sufferers travelled south to areas of increased daylength led researchers to investigate the efficacy of light treatment in SAD patients. Lewy *et al* (1982) successfully treated a patient with light in an open trial, and Rosenthal *et al* (1984) also reported the efficacy of light in a more controlled study with a series of patients. These studies established that a short daily exposure to bright light could rapidly reverse many of the debilitating symptoms of seasonal depression. Light therapy has proven to be a very popular treatment for SAD symptoms, which has also been extended to a number of other clinical conditions. Its attractiveness stems from the lack of invasiveness of the treatment, the few side effects compared to pharmacological treatments, the rapidity of its effects, and its intuitive appeal for both patients and physicians.

While there is general agreement that light therapy is an effective treatment for SAD (but see the discussion concerning placebo effects, V.C.3), there is less consensus on the timing, duration and quality of light needed to produce a response, nor on the potential interactions among these variables. A number of studies have examined the effects of varying one or more parameters of light treatment. However, no single study has systematically manipulated the

relevant parameters of light exposure (e.g., daily timing of light, duration of daily light exposure, duration of light treatment) in a way that would allow one to evaluate more than one or two parameters of treatment at any one time.

B. Light Therapy for SAD

1.Timing of light treatment

Synopsis: Some models of the etiology of SAD predict that the daily timing of light therapy should be critical. The evidence suggests, however, that effective treatments can be given at a variety of times of day, although morning treatments may be more effective for some patient groups.

A meta-analysis of all controlled light treatment studies prior to 1988 examined the rates of remission under different light treatment protocols using a combination of two criteria to determine that treatment had produced a significant clinical change. One is an absolute criterion, requiring that scores on a rating scale for degree of depression (the Hamilton Depression Rating Scale [HDRS]) fell to <8, and the other is a relative criterion, namely, a 50% or greater reduction in HDRS score from baseline (Terman *et al,* 1989). Time-of-day effects of bright light treatments given in the morning, midday, evening, and morning-plus-evening were analysed and compared to two control treatments (dim light and brief bright light exposure) in studies involving a total of 332 patients. These analyses indicated that bright light exposure at all three times of day was significantly more effective than the dim-light control procedure (11% control remission rate). Overall, however, bright light exposure (2500 lux) for at least 2 h daily for 1 week resulted in more clinically significant remissions when administered in the morning (53% of patients in remission) than when given in the evening (38%) or at midday (32%). It was also noted, however, that morning treatments were superior to evening treatments only for patients with mild levels of depression (67% remissions for morning versus 43% for evening treatments). For patients with moderate to severe baseline depression ratings, no significant differences were observed in the effectiveness of morning versus evening light in achieving clinical remission (66% *vs* 57%).

More recent studies have suggested that light at any of several times of day can produce equivalent antidepressant responses (Isaacs *et al,* 1988; Magnússon and Kristbjarnarson, 1991; Wirz-Justice *et al,* 1993). Wirz-Justice and Anderson (1990) have outlined some of the reasons for the lack of consistency among studies, such as differences in baseline severity of depression, the use of cross-over trials with resultant order effects, and the use of small sample sizes reducing statistical power. Despite these considerations, they concluded that the bulk of the evidence suggests that SAD patients can be effectively treated using any one of a number of different schedules of timed light exposure.

2. Temporal aspects of light therapy

Synopsis: Light therapy usually involves 1-2 h of light treatment daily. It appears that the total dose of light (duration x intensity) is important. Therapeutic effects are typically seen after 1-2 weeks of treatment using bright light daily for 1-2 h.

The initial studies of daily light treatment by Rosenthal *et al* (1984)used one week of daily treatment consisting of 3 h of 2500 lux light in the morning followed by 3 h of light in the evening. For practical reasons, subsequent research went on to investigate the minimal effective dose of light needed daily to achieve a response. Using "full-spectrum" bright light of 2500 lux, Wirz-Justice *et al* (1987) found that 1 or 2 h of light daily for one week was significantly more effective (77% and 69% improvement) than 0.5 h (31% improvement) in producing an anti-depressant response, as defined by the criteria of Terman *et al* (1989). While few studies have gone on to replicate the effects of 1 h of light daily, the effectiveness of 2 h of 2500 lux light has been confirmed in later studies (Doghramji *et al,* 1990); and the overall lack of response to 0.5 h of morning light for one week was subsequently confirmed by the meta-analysis conducted by Terman *et al* (1989).

Recent research has indicated that the effectiveness of a particular light regime depends on total dose; i.e., on the product of exposure time and light intensity. When light intensity was increased to 10,000 lux, 0.5 h of morning treatment produced a 75% remission rate, which is equivalent to that found in previous studies using 2 h of 2500 lux light (Terman *et al,* 1990). Furthermore, using this duration, 10,000 lux was more effective than 3,000 lux (19% remission). Forty minutes of 10,000 lux light was also confirmed as more effective than a dim red light control by a group in Iceland (Magnússon and Kristbjarnarson, 1991).

Since antidepressant responses to light can be seen after one week of treatment, most studies have adopted a 1-2 week exposure protocol to evaluate different lighting regimes. Few studies have manipulated the length of treatment directly in order to determine whether less effective doses of light can become effective if given for longer periods of time. An interaction of duration of treatment (in days) with light dose (intensity x length of light pulse) was hinted at in a study in which 15 min and 1 h daily light treatments using 3,300 lux were compared after 1 and 2 weeks of treatment (Partonen, 1994). A significant response to the 1 h light treatment was found at one week, while a response to the 15 min light treatment was seen only after 2 weeks of treatment.

3. Characteristics of effective light sources

Synopsis: Both fluorescent and incandescent lights are effective in light therapy; there is no evidence that any particular wavelengths of light are essential. Adding UV light in so-called full-spectrum sources has no therapeutic advantage, and the risks of additional UV exposure militate against the use of such sources for daily light therapy.

Most studies have used treatments involving so-called "full-spectrum" fluorescent tubes (see VI.C.) behind a plastic diffusing screen facing the patient. A few treatment studies using different light sources have also found that cool-white fluorescent tubes and incandescent bulbs are effective in light therapy (Lewy, 1987; Yerevanian *et al,* 1986). More direct study of the efficacy of different light sources has found that cool white light (broad band fluorescent light

with more power in the green and yellow wave bands) did not differ in its effectiveness as compared to full-spectrum light (broad band light with a larger UV component) using the strict Terman *et al* (1989) criteria (Bielski *et al,* 1992).

The response to treatment may depend on using broad band light since no narrow-band light stimulus has yet been clearly demonstrated to be as effective as white light in treating SAD (Stewart *et al,* 1991; Brainard *et al,* 1990). Most of these studies used lights of different wavelengths equated for photon densities (i.e., equal numbers of photons reaching the eye), although the perceptual intensities of the lights necessarily differed, given that the retina is differentially sensitive to different wavelengths (see Figure 4). Thus, Stewart *et al* (1991) reported that broad-spectrum white light was more effective than green light in reducing the absolute HDRS score, although there was no difference when the stricter Terman *et al* (1989) criteria were applied. Broad-spectrum white light was also more effective than red or blue light (which were equal in effectiveness) in terms of relative change (50% fall in HDRS), but there were no differences among groups in absolute change in HDRS. Oren *et al* (1991) compared the efficacy of green and red light and reported that green wavelengths resulted in a significant reduction in HDRS, while red wavelengths did not. Green effects were equivalent to red effects, however, using the stricter Terman *et al* (1989) criteria. Interpretation of all of these studies is difficult, however, since treatments did not differ in effectiveness for all measures or criteria. Secondly, it is not clear which light characteristic is the important one for therapeutic effects. Resolution of this issue will require comparison of the effects of stimuli equated for either the physical intensity of illumination (photon density) or its perceived brightness.

There is more general consensus relating to the inclusion of ultraviolet light in therapy; the ultraviolet (UVA) component present in full-spectrum sources does not appear to be necessary for the therapeutic effect (Lam *et al,* 1992), and many studies use filters to screen out these potentially harmful wavelengths.

4. Side effects of light therapy

Synopsis: Few side effects of light therapy have been reported, and retinal and ocular physiology do not appear to be adversely affected by it, at least in patients starting with normal retinal and ocular function.

No light-induced pathology has yet been identified in standard ophthalmological examinations of patients with normal ocular/retinal function undergoing light treatment over the short duration, although longer duration studies have yet to be done (Terman *et al,* 1989; Terman, 1994). Patients with glaucoma or cataract have routinely been excluded from treatment studies; however, there are no specific data to support such exclusion and open trials including patients with these conditions have used light therapy successfully in conjunction with ophthalmological monitoring (Terman, 1994). Patients with retinopathies or those using photosensitizing drugs are advised to avoid light treatment. Complaints of side effects are occasionally reported; these include eye irritation/strain, headache, feeling "wired", insomnia and irritability (Oren *et al,* 1991; Levitt *et al,* 1993).

5. Novel light delivery systems

Synopsis: New light delivery systems, including a light visor and a dawn simulation device have yielded therapeutic effects in some studies at lower light intensities than are typically used in standard light therapy. These approaches are quite new and have not yet been tested sufficiently.

New procedures and mechanisms have recently been assessed as treatments for SAD.

Dawn simulation. Patients have been treated by exposure to lighting conditions which attempt to mimic the intensity changes at dawn and dusk by gradually increasing or decreasing light intensity in a pattern similar to what might be experienced during natural dawn or dusk in the summer. In the initial uncontrolled study by Terman *et al* (1989), either light exposure simulating dawn (i.e., gradual increase in light beginning at 3 AM, progressing to a maximum of 1000 lux at about 5 AM before the patients awoke), or a combination of dawn and dusk simulation was effective in treating 2/3 SAD patients. Further studies with more patients confirmed that dawn simulation comprising an increase in light over a 2 h period peaking at 100-500 lux was effective in inducing full remission of SAD in 6/8 patients (Terman and Schlager, 1990). Follow-up studies have reported that dawn stimulation with white light is more effective in reducing SAD symptoms using various criteria than a presumed control dim red light (Avery *et al,* 1993; Avery *et al,* 1994). None of these studies, however, compared the dawn treatment directly to a traditional bright-light treatment. One study that did compare dawn simulation to bright light found that bright light was more effective than a dawn simulation (Avery *et al,* 1992). More research is clearly needed to define the effective parameters of light delivery using this protocol.

Light visor. A portable head-mounted light-delivery system has been developed (Stewart *et al,* 1990b) to make daily light therapy more convenient for patients. Surprisingly, multi-center studies using large numbers of patients have shown that visor light is effective in treating SAD over a broad range of intensities, from presumed control levels which were found to be ineffective in a standard, "light-box" treatment paradigm (60 lux) to bright light levels (6000 lux) (Stewart *et al,* 1990b; Rosenthal *et al,* 1993; Joffe *et al,* 1993). This lack of differential effectiveness suggests either that all the effects are placebo effects, or that the light-visor is considerably more effective than standard light boxes. One study comparing the effects of bright (4000 lux) light delivered by visor or by light box found no difference between treatments in relieving depression (Stewart *et al,* 1990b). Future studies will need to compare the effectiveness of a variety of light intensities and delivery mechanisms, as well as to use inactive control procedures in order to assess the usefulness of these novel light delivery systems.

C. Mechanisms of Light Therapy

1. Hypotheses concerning light effects

Synopsis: Several hypotheses have been advanced to explain the therapeutic effects of light treatments. These include a photon-counting hypothesis, a phase-shift hypothesis, a circadian rhythm amplitude hypothesis, and a melatonin hypothesis. None of these is convincingly supported by the available data, and some observations are inconsistent with those hypotheses that have been most thoroughly investigated.

The underlying etiology of SAD is still unknown. Studies of changes in physiological phenomena in response to light treatment have led to various hypotheses about the underlying etiology of SAD and the antidepressant mechanism of light treatment. Early proposals concerning the mechanism of light effects included the photon-counting hypothesis, which suggested that sufficient quanta of light were not available in short winter days to sustain a physiological process that is essential to preserving a euthymic (normal mood) state. This hypothesis is supported by data indicating that there is a dose-response relation underlying light effects, as well as an inverse relation between duration and intensity of effective light treatment. Although these data support the idea that the amount of light exposure is critical, the photon-counting hypothesis is severely limited by the failure to specify which internal physiological processes are affected by the degree of light exposure, and which processes mediate photic effects on mood.

Some proposals focus on the idea that exposure to short days initiates changes in the circadian system which underlie the development of SAD. There are two main hypotheses based on changes in the circadian system: the *phase-shift hypothesis* and the *amplitude hypothesis*. The phase-shift hypothesis states that SAD patients have a circadian pacemaker that is abnormally entrained relative to external cycles and perhaps to other behavioural cycles, such as that of sleep and waking (as indexed, for example, by the timing of the melatonin secretion rhythm relative to clock time or sleep phase). By analogy with the phase-shifting effects of timed light exposures on other mammals, extra light exposure at certain daily phases would shift the circadian pacemaker, restore the appropriate phase relations, and normalize the rhythmic pattern. Lewy and colleagues (Lewy *et al,* 1985; Lewy *et al,* 1987; Sack *et al,* 1990) have claimed that bright light in the morning not only produces greater remission of symptoms compared to evening light, but that the anti-depressant effect appears to be correlated with the phase-shifting effects of light on the rhythmic secretion of the pineal hormone melatonin, which is tightly regulated by the circadian clock in the hypothalamus (see IV.B.2).

In the phase-shift model, the greater efficacy of morning light is attributed to the hypothesis that most patients have phase-delayed circadian systems. Extra morning light should act to compensate by phase-advancing these rhythms to their normal position and thereby restore normal physiological function (Lewy *et al,* 1987). This hypothesis has received considerable attention and has been revised to also include phase-advanced patients who are hypothesized to require delaying (evening) light. The model remains controversial since in other studies, light treatments at various times of day, when they would presumably not have the appropriate phase-shifting effect, are capable of alleviating SAD symptoms; see the meta-analysis by Terman *et al* (1989) In addition, SAD patients do not differ consistently from control subjects in the timing of rhythmic variables such as melatonin secretion or body temperature, nor is the antidepressant

response consistently related to changes in such variables (Eastman *et al,* 1993; Rosenthal *et al,* 1987). Thus, there is little consistent evidence across a variety of studies to support either baseline rhythm phase differences between depressed and control subjects or systematic phase alterations in response to light treatments.

The amplitude hypothesis, by contrast, suggests that the amplitude (rather than phase) of critical circadian variables is reduced in SAD patients, and it is rhythm amplitude that is enhanced by extra light exposure. One study did find that light treatment significantly enhanced the amplitude of body temperature rhythms in SAD patients (Czeisler *et al,* 1987), but the amplitude hypothesis requires further investigation.

A third hypothesis also relates to melatonin, but not to its role as a marker of the circadian system. The *melatonin hypothesis* has attracted a great deal of interest, since melatonin has been linked causally to seasonal changes controlled by daylength in other mammals. According to this hypothesis (Rosenthal *et al,* 1986), changes in the pattern of melatonin secretion as daylengths shorten may be responsible for the seasonal mood changes. Treatment with light is suggested to have an antidepressant effect by suppressing the release of melatonin, which light is known to do (see IV.B.2). Several studies have, however, failed to find evidence to support this suggestion. First, administration of melatonin did not induce a relapse in recovered SAD patients, and depressed SAD patients treated with atenolol, a drug which, like light exposure, suppresses melatonin secretion, failed to improve (Rosenthal *et al,* 1988). Finally, light treatment can improve mood even when given during the middle of the day, at a time when it should have no effect on melatonin secretion, which is strictly nocturnal (Wehr *et al,* 1986).

Other hypotheses have focused on changes in the levels of neurotransmitters (e.g., dopamine and serotonin) or hormones as the mechanism responsible for SAD and for its reversal by light treatment (Oren and Rosenthal, 1992; Skwerer *et al,* 1988). In general, while changes in the regulation of the serotonergic or noradrenergic systems have been suggested by comparisons between SAD patients and controls, it has been difficult to determine which biological changes and responses to light are primary and/or which are relevant to the clinical symptoms observed in patients and their alleviation.

2. The role of the eyes in light therapy

Synopsis: The eyes mediate therapeutic effects of light on SAD patients. The possibility that light-induced changes in retinal function may be critical to treatment of SAD has been investigated but the results are inconsistent.

The question of how light exerts its antidepressant effects on the central nervous system was addressed in a study which compared the effects of skin versus eye exposure in SAD patients (Wehr *et al,* 1987b). This study found that the eye plays the primary role in delivery of light's antidepressant effects. The retina also contains melatonin and one role of light may be to stimulate dopamine production in the eye, which would suppress retinal melatonin and improve mood by consequent effects on retinal sensitivity to other effects of light or on dopaminergic system activity (Oren and Rosenthal, 1992). Others have also suggested that SAD could be linked to changes in retinal photoreceptor renewal mechanisms (Lewy *et al,* 1982). Reports of changes in retinal function in SAD have, however, been inconsistent. Both increased and decreased sensitivity of retinal responses to light have been reported in studies using

electrooculography, electroretinography or dark adaptation methods (Oren, 1991; Lam *et al,* 1991; Lam *et al,* 1992; Murphy *et al,* 1993). The variability of the findings within and between studies precludes drawing any firm conclusions about retinal mechanisms at this point.

3. Light and placebo effects

Synopsis: It is extremely difficult to administer a placebo treatment to which therapeutic light effects can be compared without the patients' awareness of the difference. There is some evidence of moderate improvements induced by placebo treatments, suggesting that light effects may be mediated in part by patients' beliefs and expectations, but these results remain controversial. Other evidence is inconsistent with light acting primarily as a placebo treatment.

To establish rigorously that light is an active treatment, its effects should be compared to placebo treatments, similar in all respects to the light treatment, but without the presumed active ingredient. This requirement for outward similarity between the two conditions is relatively easy to meet in drug studies by using identical capsules, that differ only in their contents (even then, perceptible side effects can still confound the analysis of some drug studies). While studies of light effects have typically used lights differing from "active treatment" in intensity, timing, wavelength or duration as control conditions, these conditions clearly cannot function as true placebo controls. Subjects are necessarily aware of the reduced intensity, duration or altered spectral properties of the presumptive placebo treatment. These apparent differences could lead to effects that are a secondary result of subjects' beliefs or expectations with respect to the two treatments, rather than a result of differences in their direct physiological effects.

The fact that the experimental and control treatments cannot be made indistinguishable, and the small to moderate (but consistent) antidepressant effects of a variety of "placebo" treatments that have been used, have led Eastman to suggest that any conclusion about efficacy beyond placebo effects is problematic (Eastman, 1990a). In fact, Eastman *et al* (1992) found similar antidepressant responses in SAD subjects receiving light therapy and in those exposed as a control group to a covertly deactivated "negative ion generator". Since no stimulus was delivered by the deactivated machine, yet it was as effective as the light treatments, Eastman *et al* (1992) suggested that light treatment may be working via a simple placebo response. However, the response of the patients to light therapy was poor relative to many previous studies, which may have resulted in the control and experimental treatments having similar effects. This provocative study deserves replication in other centres with more SAD patients in order to evaluate the usefulness of this novel control procedure.

In defense of the hypothesis that bright light does have active therapeutic effects beyond those of placebo treatments, Wirz-Justice and Anderson (1990) noted that SAD patients are not particularly placebo-responsive in studies using pharmacological placebo controls. In addition, many studies measured patient expectations before any treatments, and did not report any consistent bias among patients in favour of bright light. Thus, differences in expectations about the treatments do not appear to account for the superiority of bright light over control treatments. Finally, light treatment effects can be sustained over many years, while placebo effects tend to be more transitory (but see Eastman, 1990a). It is important to note that the claim

that brief daily exposures to bright light can produce consistent anti-depressant responses in SAD patients is not in dispute: the unresolved issue under discussion is the mechanism by which light therapy achieves these often dramatic clinical improvements.

4. Natural light exposure in SAD patients

Synopsis: Our very limited knowledge of typical patterns of light exposure indicates that both normal controls and SAD patients have only very brief exposures to bright outdoor illumination on a daily basis. The role these exposure patterns play in the etiology of SAD or subsyndromal symptomatology is unknown. Single case reports hint that some people may be quite sensitive to natural variations in environmental light availability.

Given the observed occurrence of SAD during seasons with short daylengths and the effectiveness of light therapy in altering mood in these patients, their levels of natural light exposure and the effect of decreasing light exposure might be expected to be critical variables in the etiology and treatment of this syndrome. Recent studies examining these issues have suggested that natural light exposure is fairly limited for most people, with extreme variability seen in individual patterns. Eastman (1990b) has recently reported estimates of the duration of daily sunlight exposure in 12 subjects with SAD during the winter and the summer. She found that daily sunlight exposure in the summer was twice as long as that in the winter (3 *vs* 1.2 h/day), but generally much shorter than the bright light period that is naturally available in the environment. Normal subjects have also been found to expose themselves to only these short durations of natural bright light (Okudaira *et al,* 1983; Savides *et al,* 1986). These patterns of self-exposure to bright light may be relevant for both SAD and sub-syndromal SAD sufferers, but a good deal more information is needed on natural patterns of light exposure in different populations.

The intensity, as well as duration, of natural light exposure may also play a role in some sensitive people who do not fit the classic pattern of relatively strict seasonality of symptoms. In one case study, a lengthy record of repeated depressive episodes indicated a positive correlation between onsets of depressive episodes and falling light intensities due to cloud cover, without regard to season (Summers and Shur, 1992). This relationship is consistent with anecdotal reports of the effects of inadequate light on SAD patients. Such single-case studies can only be suggestive, but the results may indicate that an otherwise uncharacterized subset of individuals may respond with strong mood changes to relatively minor changes in daily light exposures.

D. Light and Other Mood Disorders

1. Normal subjects and subsyndromal SAD

Synopsis: Light therapy does not have mood-enhancing effects for normal populations, but a subgroup of people with mild seasonal symptoms, but no clinical depression, do show improved mood in response to light treatments.

Several studies have concluded that bright light therapy delivered in a standard protocol which is effective for patients with SAD has little or no beneficial effect on individuals who do not suffer from SAD, as assessed by objective or subjective ratings of mood, fatigue, and/or sleep length (Genhart *et al,* 1993; Kasper *et al,* 1988; Kasper *et al,* 1989a; Kasper *et al,* 1990; Rosenthal *et al,* 1987). When the population of individuals without SAD were analysed more closely, however, it appeared that light was more efficacious in improving the mild symptoms of a subset of people with somewhat more pronounced seasonal difficulties (but without clinically defined SAD). The people with a significantly better response to light therapy than asymptomatic controls were described as having "subsyndromal SAD" (S-SAD) (Kasper *et al,* 1989a).

These findings of an effect of additional light exposure in a subset of the general population who are otherwise regarded as "normal" (i.e., not clinically depressed) may have important practical implications. The results suggest that there may be potential negative consequences of restricted access to bright light, and potential benefits of enhancing environmental light exposure in a larger portion of the population than those that meet the strict clinical criteria for SAD.

2. Non-seasonal depression and premenstrual syndrome

Synopsis: There is little evidence that light therapy is effective for treatment of non-seasonal, major depressions. A few preliminary results suggest that symptoms of late luteal phase dysphoric syndrome (premenstrual syndrome) can be improved with light therapy.

Less work has been done on the efficacy of light treatment for non-seasonal depression than for SAD. Early work by Kripke and colleagues (Kripke, 1985; Dietzel *et al,* 1986) reported small effects of light therapy on depression ratings, using brief treatment protocols lasting only 2-5 days for non-seasonally depressed patients. A follow-up study (Kripke *et al,* 1992) compared bright (2000-3000 lux) and dim (50 lux) light delivered for one week for 2-3 h in addition to psychotherapy. Although they reported a significant group difference in depression levels after bright versus dim light treatments, few patients would have met the joint clinical criteria of Terman *et al* (1989) for remission.

Other studies have found no effects of light treatment on non-seasonal depression. Yerevanian *et al* (1986) compared light treatment in SAD and non-SAD depression and reported that only SAD patients showed remission in response to light. This study was limited, however, by the lack of comparability of the patient populations, since the two groups differed in medication usage and in whether the treatment was delivered on an inpatient or outpatient basis. Stewart *et al* (1990a) also found that patients with nonseasonal depression, selected because they also displayed the "atypical symptoms" of hypersomnia or hyperphagia which characterize patients with SAD, also failed to respond to a light treatment protocol that was successful in

treating a matched group of SAD patients. Inconsistent findings were also reported in larger scale studies by one group which found significant effects of dim and bright light treatments in a group of 30 non-SAD patients in one study, but no effects of bright light treatment on 42 non-SAD patients in another study (Volz *et al,* 1990; Mackert *et al,* 1991). Taken together, the bulk of the evidence suggests that if light treatment has any effects on non-seasonal depressions, they are small and inconsistent; the clinical significance of these effects has yet to be determined.

The effectiveness of light therapy has been examined in preliminary studies of mood in patients with late luteal phase dysphoric disorder (premenstrual syndrome) by Parry and colleagues (Parry *et al,* 1989; Parry *et al,* 1990; Parry *et al,* 1993). These studies have generally found significant improvements in objective measures of mood and irritability following light treatment, although mood also improved in a dim light control group in one study (Parry *et al,* 1993), suggesting placebo effects may underlie some or all of these responses. These studies warrant extension and replication in larger groups with systematic manipulation of different light and control treatments.

E. Light Exposure and Sleep

Synopsis: Light therapy has been used with some evidence of effectiveness to treat disorders that are characterized by disrupted sleep, especially in the elderly, and demented elderly patients. Despite evidence for some arousing and alerting effects of light treatments for people, evening light therapy does not appear to disturb subsequent sleep.

Sleep patterns become more fragmented with age, leading to chronic sleep disturbance in a substantial proportion of the elderly population (Partonen, 1994). Aging also results in phase advances of circadian rhythms such as that of core body temperature. These observations have led to the suggestion that sleep disturbances in aging may be related to abnormalities of circadian rhythm entrainment, which has led to the idea of using light therapy to treat such sleep disturbances. Based on knowledge of light effects on the underlying circadian clock mechanism (see IV.B.1), a few studies have examined the effects of bright light treatment that has been timed to attempt to restore the appropriate underlying circadian phase (Campbell *et al,* 1993; Lack and Wright, 1993). The findings suggest that 2 h of bright light exposure in the evening results in a significant improvement in sleep quality compared to dim light control exposure, as measured by EEG recordings or self-reports. Given the potential difficulties with pharmacological treatment of sleep in the elderly (including addiction and drug-withdrawal induced insomnia), this non-pharmacological alternative warrants further study.

The effects of light have also been evaluated in elderly patients with dementia who are housed in nursing homes. These patients often suffer from behavioural disturbances such as wandering and sundowning (recurring confusion and agitation in the early evening), which are a major problem for their caregivers. Bright light exposure appears to improve sleep quality in these patients. For example, two open, uncontrolled studies using 2 h of bright light in the evening or sunlight exposure for one week found significant improvement in clinical ratings of sleep time, decreased waking during the night, and an increased amplitude of 24 h activity rhythms, as measured by wrist sensors (Satlin *et al,* 1992). Okawa *et al* (1991) also found that morning bright light exposure for 1-2 months improved sleep/wake patterns in 12/24 demented

patients with Alzheimer's disease more effectively than did a placebo, non-light treatment, as assessed by nurses' observations; similar results were reported by Mishima *et al* (1994). The timing of effective light treatments has not been consistent across studies. These differences in the reported effectiveness of light treatments may be attributable to the inclusion of different patient populations, to the subjective scoring of sleep by non-blind raters, or to different underlying etiologies of the sleep disturbances in the groups tested.

The use of light therapy for insomnia in some people has led to an examination of one potential drawback of using light treatment at night, that of a possible alerting or activating effect which could interfere with the immediately following sleep phase. Light treatment can result in subjective activation, as noted in reports of its side effects in SAD patients (see V.B.4), and autonomic arousal (see IV.B.5). Some patients have been reported to feel hypomanic irritability and hyperactivity during light therapy, which subsides when the light treatment is discontinued (Rosenthal *et al,* 1984). Bright light-induced alertness/activation has been reported in normal subjects in a number of studies using different light protocols, including all-night exposure or exposure during only parts of the night or early morning (Czeisler *et al,* 1990; Badia *et al,* 1991; Dawson and Campbell, 1991; Clodore *et al,* 1990; Hannon *et al,* 1992). Badia *et al* (1991) reported increased tonic skin conductance levels and increased EEG beta wave activity (indicators of physiological and cortical arousal) when subjects were exposed to bright light, suggesting a direct physiological arousing effect. This arousal is likely to be short-lived, however, since evening light exposure has not been reported to interfere with subsequent sleep periods (Drennan *et al,* 1989; Bunnell *et al,* 1992).

F. Summary

SAD is a clinical condition that has been linked to light by both its occurrence during the short days of fall and winter, and by the fact that it can be treated effectively with daily supplementary light exposure. Despite a great volume of research on SAD over the last decade, the physiological mechanisms that give rise to its symptoms and that mediate the therapeutic effects of light remain uncertain. While some results suggest a close linkage to neural mechanisms regulating circadian and seasonal rhythms, other findings are inconsistent with models based on these mechanisms.

The implications of the clinical research on SAD for issues related to light exposure in the home are uncertain. Single case studies document the existence of individuals who are susceptible to depression when deprived of adequate environmental light levels. In addition, individuals with symptoms of subsyndromal SAD may also be affected by light availability even though they do not develop the full-blown clinical symptoms of SAD. These results suggest that while reduced light exposure is not linked to depression in the general population, there may be a subset of individuals who could respond with clinical or subclinical symptoms when they are relatively light deprived, either seasonally or otherwise.

This conclusion suggests that manipulations of lighting in the home or workplace by coated windows which reduce light penetration could affect a subset of the population, depending on the degree of light attenuation, their exposure to other sources of illumination, and other unknown factors. This suggestion is, however, speculative in the absence of a sufficient database documenting such effects.

VI. ILLUMINATION IN HOME AND WORKPLACE

> *"Some buildings have windows which consist of tinted thermopane...not only is natural light reduced by 50%, but also--in keeping with the purpose of those windows--daylight's spectrum is considerably reduced, above all in its heat-producing, long-wave red portion. As a result of this decrease in brightness, artificial sources of light with their abbreviated spectrum are turned on much earlier and much more frequently during the day."* (Hollwich, 1979)

A. Attitudes to Windows

Synopsis: People generally favour having large windows in homes and offices because of the attractions of extra natural light and a view. This enthusiasm is tempered by concerns for heat loss in winter, heat gain in summer, the effects of sunlight on fabrics in the home, and increased glare in work environments.

By allowing transmission in both directions of both visible and infrared light, and by allowing a view, windows have a profound impact on the experience of people in a room. Most reports describe a variety of psychological, social and physical factors which make people favour substantial window areas in homes and offices. Among the issues raised by people in surveys related to lighting are the desirability of having a view to the external world; an expansive sense of contact with the outdoors; positive mood effects of seeing sunshine; and desire for the warmth and "atmosphere" provided by sunlight in certain rooms at certain times of day. A common attribution is that sunlight has a therapeutic effect ("sunlight makes you feel better") (Hopkinson, 1967; Bitter and van Ierland, 1967; Boyce, 1981; NEMA, 1989). In new buildings, most concern is expressed about the quality of lighting, ventilation and temperature control, but views of the outside are also considered desirable (Cooper *et al,* 1973).

The degree of enthusiasm expressed for large window areas is tempered somewhat by concerns with respect to negative features of sunlight. These depend on climate and season, and include the thermal burden imposed by large glass surfaces both in southerly regions and in temperate regions during the summer, and excessive heat loss (and therefore heating costs) in winter. In addition, fading of fabrics exposed to sunlight, glare from windows and the desire for privacy are also factors affecting preferences for large windows. People prefer to have sunlight available for work environments, and value external views highly, but issues related to the control of heat and glare are also most important in the workplace.

The level of enthusiasm for sunlight as an office versus home illumination source is reduced by the perception of a lack of adequate control of these negative factors in office environments (Boyce, 1981). In fact, by comparison with people in their homes, office workers may have little opportunity to regulate these features by opening windows, closing drapes or altering heating or air conditioning, especially in modern, "sealed" office buildings. Another factor that reduces control over sunlight exposure is the degree to which people share a large workspace in

common using space dividers, rather than having individual offices. Complex models have been developed to address the cascade of costs and benefits that can arise from allowing office workers control of window shades. In one such model (Newsham, 1994), allowing manual control of both window blinds and lighting resulted in increased occupant comfort and less overheating, but at the cost of increasing energy use for heating and, especially, for lighting. In other words, allowing workers to regulate window glare and thermal discomfort from sunlight reduces passive solar heating and lighting. The result is not only an increase in costs, but an increase in the use of artificial lighting sources. Allowing active control of window coverings therefore is predicted to have some of the same effects as the use of tinted glass (see VI.B).

B. Effects of Window Tinting

Synopsis: Window tinting that reduces the intensity and alters the spectrum of sunlight entering a room is not usually regarded as objectionable on aesthetic grounds. The processes of adaptation and colour constancy make these changes not very noticeable unless a contrasting light source is also available. Reduced light availability and altered perceptions of brightness as a consequence of such window tinting does, however, encourage use of additional artificial lighting sources, the consequences of which need to be considered as part of the cost or benefit of window tinting.

A critical, but often ignored, indirect effect of the character, size and distribution of windows is the impact these features have on decisions to use artificial lights to supplement or replace window lighting during the day. As a result of this indirect impact, any physiological effects that artificial room lighting may have can be magnified by window designs that encourage increased use of artificial lighting. Tinting glass alters its spectral characteristics, but also reduces the intensity of light passing through the window; the latter change may encourage additional use of artificial light (Robertson *et al,* 1989).

Sunlight and artificial light have been reported to have different effects on people in a work environment. In one study, illumination of a desk surface with >1000 lux was regarded as excessive, but the same intensity provided by daylight was not (Boyce, 1981). Similarly, in another study, exposure to bright, artificial light (3,500 lux) over a long period of time triggered increased levels of cortisol secretion, presumably reflecting a stress response. By contrast, natural sunlight of similar or brighter intensity was not reported to have similar effects (Hollwich, 1979). These results imply that either the spectral characteristics or the physical characteristics of the source (spotlight *vs* diffuse illumination; glare *vs* non-glare) might affect attitudes towards the light. Hollwich (1979) claimed (without presentation of data) that full-spectrum artificial light did not have effects similar to those of other artificial lighting sources, implying that wavelength characteristics were critical. These claims should be assessed rigorously, but that is obviously not possible in the absence of any presentation of relevant data.

Window glazing that distorts the spectrum of light is generally not viewed by room occupants as objectionable on the grounds of colour distortion. For particularly colour-sensitive tasks, the distortion may be relevant, but for most tasks and for general illumination the processes of adaptation and relative colour judgements (see III.C) make these distortions less obvious (Cooper *et al,* 1973; Illuminating Engineering Society, 1977; Boyce, 1981). However, the spectral changes induced may affect perceived brightness. Different types of window glazing were reported to affect judgements of brightness of room lighting, but these judgements did not correlate strongly with percentage light transmission through the glazing. Rather, the pattern of spectral distortion induced appeared to be most relevant to perceptions of insufficient or excess brightness (Cooper *et al,* 1973).

The presence of nearby unglazed windows or windows that can be opened to admit natural daylight may allow contrast effects which provoke unfavourable judgements about tinted windows and general room illumination (Cooper *et al,* 1973). Thus, recommendations for lighting engineers suggest that while tinted glass can affect colour rendering, this is not a serious problem. A major ancillary recommendation is that nearby lighting sources have to be harmonized to avoid comparative colour judgements which are negative for tinted glazing. Another concern is for glare affecting those outside the building, since solar control glass increases reflectance and glare outside (Illuminating Engineering Society, 1977).

C. Room Colour and Full-Spectrum Lighting

1. Room colour

Synopsis: Despite the fact that the popular literature often refers to effects of room colour on mood and performance, controlled studies demonstrating such effects are difficult to identify. There is no obvious physiological mechanism to account for such claimed effects and no empirical evidence to support most of the claims made.

Room colour is of some relevance to discussions of the effects of tinted glasses, in part because they distort the spectrum of sunlight differently than do untinted glasses. In addition, inferences about the normal effects of natural solar illumination on humans have often been based on comparisons of the effects of two artificial sources which differ in their approximation to the solar spectrum.

There are numerous claims in the popular literature and in publications related to interior design and architecture; e.g., Mahnke and Mahnke (1987), that lighting type and room colour have profound effects on human mood and performance. It is more difficult, however, to find convincing experimental evidence that this is the case. Claims, for example, of beneficial effects of full-spectrum versus other sources of artificial lighting, or of particular room colours, on various measures are often supported by reference to general reviews, opinion pieces, and uncontrolled studies (Mahnke and Mahnke, 1987). One report of an attempt to study wall colour effects on human performance and mood reviewed the relevant literature supporting the idea that red environments promote arousal and

aggressiveness while blue rooms are associated with low arousal levels. The authors concluded that the background literature on these issues was "scattered and varied" and they then failed to find any experimental support for these claims in their own study (Ainsworth *et al,* 1993). Note that these "widely believed" views are inconsistent with the claims that exposure to high colour temperature (blue) lights is associated with increased, not decreased, autonomic arousal in other studies (Mukae and Sato, 1992); see IV.B.5.

2. Full-spectrum lighting

Synopsis: "Full-spectrum lighting" is a vague concept, often used to describe UV-supplemented fluorescent sources. There are numerous published claims that UV-supplemented light can have beneficial behavioural and physiological effects on humans. The literature in this area could provide some insights into effects of exposure to sunlight, which also contains more UV wavelengths than most artificial lighting sources. The studies that led to the claims of benefits from such light were, however, poorly designed and flawed in both execution and analysis. No claims of beneficial effects of UV-supplemented or full-spectrum lights can be supported by the available data. These studies therefore provide no useful information with respect to the likely impact of windows and daylighting on human physiology and behaviour.

One view that has received considerable attention is that "full-spectrum" fluorescent lighting has numerous beneficial effects on human physiology as compared to other fluorescent sources. The definition of "full-spectrum" lighting is itself problematic, since the term is used loosely to describe sources that include more UV than most fluorescent sources, and that more closely approximate the spectrum of sunlight in the visible range (see Figure 1). However, no fluorescent lighting spectrum is actually equivalent to that of natural sunlight reaching the earth; indeed, it is not possible to define a single spectrum for sunlight. The spectrum of natural sunlight reaching any point on earth varies considerably with time of day and season of year. It is, therefore, not clear whether any "full-spectrum" source should be expected to be functionally equivalent to sunlight illumination. Nor is it clear, for the purposes of this review, that inferences about sunlight effects can legitimately be drawn from studies of full-spectrum lighting. Nevertheless, since studies of sunlight effects are themselves rare, the more extensive literature on full-spectrum artificial lighting should be examined for any hints of differential lighting effects, which might lead to reasonable inferences about differential effects of sunlight and other illumination sources. The reports reviewed here are primarily of field studies, which appear to have the most direct application to real-world lighting problems; they are summarized below and discussed in more detail in Appendix A. Laboratory studies of full-spectrum lighting have been reviewed recently by Veitch and McColl (1994).

An early report (Mayron *et al,* 1974) on children in school classrooms comparing full-spectrum and "cool white" fluorescents failed to find any significant effects on school performance. That report did describe a reduction in "hyperactivity" in the full-spectrum classrooms. This study was unfortunately seriously flawed in design and no conclusions about the effects of lighting spectrum on children's behaviour can be drawn from it (see Appendix A).

Other reports have made much stronger claims for the beneficial effects of full-spectrum lighting on school children. Claims have been made for improvements in children's scholastic performance, school attendance, and rates of physical development, and for reductions in dental caries associated with the substitution of full-spectrum lights for other forms of lighting in the classroom (Hathaway *et al,* 1992). This report is worth considering in some detail because it describes an extensive recent study involving five schools over a two-year period, was conducted by a provincial government department, and it appears to include a number of controls and design features that might be expected to allow for reliable assessment of any effects of full-spectrum lights. Its stated conclusions suggest that striking improvements in academic performance, physical growth and dental health can be achieved by the simple expedient of replacing older lighting systems with full-spectrum lights. These conclusions might be extended to apply to situations in which greater exposure to natural illumination could be achieved, or to situations in which window characteristics alter the "full-spectrum" character of daylight (e.g., when windows are tinted). This report is, therefore, worth considering in the context of trying to understand both the effects of access to unfiltered daylight and the effects of different artificial substitutes for daylight. Unfortunately, this study was seriously flawed in design, execution and analysis, and essentially no reliable conclusions can be reached based on it. While a detailed review of all its flaws would be beyond the scope of this report, a few critical failings are discussed in Appendix A.

Another study making inappropriate claims for effects of room colour and full-spectrum lighting on the physiology and behaviour of severely handicapped children exemplifies many of the problems in this research area (Wohlfarth and Sam, 1981). The authors concluded that certain colours and lighting conditions could improve behaviour and reduce unwanted arousal and aggression among these children. This study was riddled with serious flaws which make its results uninterpretable and its conclusions untenable. A few of these problems are summarized in Appendix A.

A general problem with such studies is the failure to consider what the *a priori* probability is that classroom lighting could have any significant effect, and a failure to consider the children's patterns of exposure to indoor and outdoor illumination as a serious confounding factor. Consider the information *not* provided about the Alberta schools in the Hathaway *et al* (1992) study. Did all schools have outdoor recesses? Were these available twice daily? Did children go out at lunchtime? What was the pattern of natural light exposure at other times of day? In the sunny (but cold) Alberta winter, what proportion of UV exposure came from the many possible opportunities for daylight exposure during recess, lunch and the

rest of the day versus during the hours of exposure to very low UV levels in the classroom from full-spectrum light sources? If the classroom exposure constituted only a very small proportion of total daily exposure, it is *a priori* improbable that it would have any effects such as those claimed. None of these issues were addressed in the studies reviewed, but they are absolutely crucial to their interpretation, and for the design of any future studies on this topic.

One review of the limited older literature on full-spectrum light effects on human physiology and behaviour concluded that the results were contradictory and that well-controlled studies showed no significant impact of such lighting (NEMA, 1989). Another, more recent review of this literature also concluded that the results of earlier studies on full-spectrum light are either inconclusive or have reached contradictory conclusions (Küller and Lindsten, 1992). Similarly, the extensive review by Veitch and McColl (1994) concluded that there is no support for claims that full-spectrum fluorescent lighting improves performance, mood or health in the general public. Our more limited review of this topic has also failed to find any credible evidence to support the claims made for beneficial effects of full-spectrum lighting.

In summary, the quality of research on this topic has been generally very low. Although field studies are necessarily somewhat constrained, the studies reviewed here, which have been cited as finding important effects of lighting on the behaviour and physiology of children, are clearly inadequate even by the relaxed standards of field studies. The defence that field studies are difficult to control should be countered firmly with the assertion that the strength of conclusions reached in any study must match the quality of the results obtained. If the results are ambiguous and uninterpretable, no conclusions should be drawn from them. Similarly, the assertion that confounded results and incorrect conclusions should be accepted, at least tentatively, because the research question is important, should be turned back on itself. It is precisely *because* the question is of considerable economic and public health significance that only well-controlled studies and well-founded conclusions should be accepted.

It was not possible using citations from the studies reviewed or other sources to identify similar studies that presented data that were any more believable than those reviewed. One must conclude that the effects of partially simulating daylight with "full-spectrum" fluorescent lights in classrooms have not been assessed appropriately. Claims for dramatic effects of artificial UV supplementation on children's growth, academic performance and health are not supported by any credible data and should not form the basis for public health decisions.

D. "Sick-Building Syndrome"

Synopsis: The causes of the allergy-like and other physiological and psychological symptoms called "sick-building syndrome" are uncertain, but problems with building ventilation are often cited as a critical issue. Indoor fluorescent lighting may contribute to air-quality problems when the UV light from such sources interacts with chemical pollutants to generate a form of indoor photochemical fog. Window tinting or reduced access to outdoor illumination may contribute to the problem by encouraging additional use of artificial lighting sources. Whether these factors also contribute directly to generating the symptoms remains unknown.

Occupants of modern sealed, artificially ventilated and illuminated office, educational and hospital buildings often complain of an array of physical and psychological symptoms that have been labelled collectively as "sick-building syndrome" (Morrow, 1992). Transfer to a building with these characteristics has been reported to lead to a steep increase in reported illnesses and worker absenteeism (Sterling and Sterling, 1983). The usual symptoms include eye, nose and throat irritation; headache, fatigue, confusion and dizziness; allergic types of reactions; and decreased concentration and task performance. The causes are difficult to pinpoint, but the symptoms appear to be associated with low fresh-air supply, chemicals from building, furniture and carpeting materials, and the presence of volatile organic compounds from worker activities (e.g., photocopiers, smoking).

Although most studies do not address lighting conditions as a factor, complaints about lighting are often associated with the other complaints. Workers in a modern sealed building with windows screened to reduce sunlight complained more about the quality of the artificial (fluorescent) lighting than did workers with better access to natural illumination (Sterling and Sterling, 1983; Robertson *et al,* 1989). The complaints centred on inadequate illumination levels (which promote more use of artificial illumination), unacceptable levels of glare, and eye irritation. These observations suggest that some attention should be paid to the patterns of daylighting and artificial lighting, as well as the spectra and intensities of lighting available, as contributors to symptoms in so-called "sick" buildings, but there has been little exploration of this issue in the literature.

Despite the frequent documentation of correlations among symptoms and physical features of the environment, few studies have attempted experimental manipulations of variables that could allow some inferences about causality. One unusual study (Sterling and Sterling, 1983) examined the hypothesis that a source of sick-building syndrome in a Vancouver office building was a form of photochemical smog similar to that created by the catalytic action of UV from sunlight on outdoor chemical pollutants. The underlying hypothesis was that a variety of pollutants (including formaldehyde, toluene and hydrocarbon vapours) in a new building, arising from off-gassing from building materials and workers' activities, interacted with UV light from fluorescent sources to generate a form of photochemical smog indoors. The new building used a large number of "full-spectrum" fluorescent tubes, with enhanced levels of UV light emissions, and the

air vents blew air over the light sources into the offices. A critical feature of the study was an attempt at a controlled manipulation of ventilation or lighting alone, and of the two combined, along with a restoration of original conditions to assess the reversibility of any observed effects. Ventilation was altered by adding a high proportion of fresh air to the mixture, while lighting was altered by replacing full-spectrum tubes with cool-white tubes which have much lower levels of UV emission.

The findings were that changes in either ventilation or lighting caused reductions of ~7-17% in some symptoms (e.g., eye irritation, sleepiness, concentration), but none of these changes achieved statistical significance. When the two changes were combined, however, most symptoms were reduced by more than 5%, and eye irritation declined by more than 31%, which was highly statistically significant. Reversal of these environmental changes caused symptoms to recur at previous levels. These experimental results provide interesting support for a model of generation of eye irritants in the workplace based on a buildup of environmental pollutants because of poor ventilation, and their conversion to toxic substances by exposure to UV radiation from full-spectrum light sources. One assumption was that the placement of the air inlets adjacent to light sources was a contributing factor, but this was not subjected to experimental assessment.

These results suggest that the role of lighting in the generation of indoor pollution should be explored further. In addition, the results support the idea that environmental manipulations that encourage artificial lighting (smaller windows or light-reducing tinting) can have unexpected and undesirable consequences for room occupants. They also raise concerns that wholesale installation of full-spectrum lights may not only fail to improve health (see VI.C), but may actually contribute to some health problems. Whether reduced or spectrally altered daylighting can also contribute directly to the development of sick-building syndrome remains unknown.

E. Windowless Environments

Synopsis: While most people would agree that windowless work environments and classrooms are unpleasant and undesirable, the experimental evidence does not support any strong conclusions as to negative effects of the lack of windows. There are even some benefits in terms of reduced heat gain and loss and reduced distractibility of room occupants. Most of the research in this area has been methodologically flawed, and more systematic research is needed to examine the impact of windows in a variety of situations.

Boyce (1988) reviewed a number of papers that examined the effects of windowless factories and schoolrooms. He reported that some studies found no ill effects of windowless environments, while others indicated that people were unhappy with the environment. One apparently well-controlled study reported an increase in post-surgical delirium in hospital patients kept in a windowless room during recovery, as compared to those with access to a window (Wilson, 1972). Another study described an initially negative response of teachers to a windowless

classroom, followed by a switch to a preference for it, because of reduced distraction of children, reduced problems with temperature control and glare, and increased wall space. Clearly such factors as the local climate and levels of insolation will have an important impact on these assessments by teachers and students, but little more can be said because the evaluation of the windowless conditions was quite superficial in most studies. The degree of control permitted over heating, air-conditioning and glare will undoubtedly have a large impact on attitudes toward windows in the classroom, as it does for home and office environments (see VI.A); see Boyce (1988) for a review of these issues.

Another review of similar studies (NEMA, 1989) concluded that there is not much published evidence supporting a consistent effect of daylighting via windows or its absence on task performance. That review also concluded that the measures used so far in studies have probably been insensitive, and in most studies the results have been confounded by other factors.

A more recent report summarized scattered references in the literature to beneficial medical effects of a view through a window, and attempted to assess whether school children are benefitted by the presence of windows in the classroom (Küller and Lindsten, 1992). This study attempted to correct some shortcomings of earlier studies by using long-term exposures to altered lighting environments (during a full school year), using schools that were close to each other, and comparing both natural to artificial lighting and full-spectrum to other fluorescent lights in a single study. Most features of children's health and behaviour were not systematically different among the four classrooms studied. However, urinary free cortisol was found to show a different pattern across four samples taken at different times of year in the one classroom lacking both windows and full-spectrum lighting, as compared to the other three classrooms. Since adrenal cortisol secretion can be affected by stress, differences in the patterns of secretion may be related to stressful environmental events or features.

However, there was no indication that the one change observed in adrenal activity was in any way a reflection of a harmful change in physiology. In addition, the relevance of the results observed to the role of daylight or full-spectrum illumination was not established. The one room with altered cortisol patterns not only lacked full-spectrum lighting, but also was much more dimly illuminated than any of the other rooms. This seems at least as likely a source of the difference observed as any differences in spectral properties of the illumination sources. The decision, based on these results, to reconstruct these classrooms to include windows was not justified by the findings. The shortcomings of this study are discussed further in Appendix B.

One must conclude that the case for windows in classrooms is similar to that for full-spectrum lighting. There is an obvious need for dispassionate, independent, carefully controlled studies by researchers with a sophisticated knowledge of experimental design, statistical analysis of data, lighting physics and human visual physiology. Conclusions about the effects of windows or full-spectrum lighting

sources on children in classrooms cannot be made in the absence of such studies. Previously published reports in these areas amount to little more than expressions of opinions on both sides of the issue.

F. Effects of Aging

Synopsis: Aging is accompanied by a variety of natural changes in the eyes and increased incidence of ocular diseases that reduce and alter transmission of light to the retina. In addition, older people are more likely to be housebound as a result of illness and to spend less time outdoors than younger people, especially during the winter and in inclement weather. These factors probably combine to reduce the exposure of older people to natural light, although there are few empirical results to confirm this speculation. The combination of reduced exposure to daylight and reduced transmission to the retina may make older people especially susceptible to any further reductions in light intensity as a result of reduced size of, or transmission through, windows in the home.

Because tinting windows reduces overall light transmission into homes, another consideration is what effect reduced light availability has on particular target groups. Older people are of particular interest for several reasons. First, they will usually tend to stay outdoors less than younger people, who are generally more mobile and engage more in outdoor sports and other outdoor activities. This difference is probably exacerbated by inclement weather, especially during the winter, when environmental light availability is already lower. Because of retirement from the workforce, chronic illnesses, and reduced mobility, older people may spend many more hours in their homes than do people during their working years. As a result, older people will be exposed to less of the beneficial effects of sunlight outdoors. Tinted windows, depending on their spectral transmission characteristics, may further reduce access to useful light wavelengths in this population.

Second, during aging, there is a reduction in pupil aperture size, increased lens opacity and increased light scatter in the eyes. The net result is that if a healthy 60 year-old and 20 year-old are both exposed to identical light intensities at the level of the cornea, the older person will receive only one third of the light at the level of the retina as the young person (Weale, 1963; Boyce, 1981). Human eyes can adapt over a large intensity range, but these profound changes in even healthy older people still imply a dramatic reduction in available light at the level of the retina. In addition, the non-visual or "energetic" effects of light may depend on absolute, not relative (adapted), light levels. Thus, the decrease in retinal illumination with aging may be of great significance for processes that depend on the absolute amount of light received by the photic system (see IV.B), rather than on the capacity of other parts of the visual system to adapt to different background intensity levels.

The lens becomes yellowed as well as more opaque with age and therefore may act as a spectral filter relative to the lens of younger eyes (see Figure 3). This implies decreased transmission of shorter wavelengths to the retina. As a result, light sources of equal nominal intensities but different wavelength distributions will be filtered differentially by young and old eyes. This observation may contribute to explaining why elderly subjects were described as preferring "full-spectrum" lights to "warm-white" lights of equal nominal intensity (300 lux) in one study (Kolanowski, 1990). It should be noted, however, that the main result of that study was a failure to observe any behavioural changes in older people as a result of exposure to these two light sources.

Since a yellowed lens will transmit less of the shorter wavelengths to the retina, lights with a stronger blue component than a relatively yellow "warm-white" source, might appear brighter to older eyes because they supply more light energy in a part of the visible spectrum that is more strongly filtered by the characteristic pigmentation of the aging lens. In addition, since window glass that filters out UV light will also reduce some short wavelengths, it will reduce light availability in the region of the spectrum that is already affected by increased lens pigmentation in older people.

A third issue relates to the increased incidence of ocular abnormalities with aging (Kapperud, 1983). The most common form of ocular abnormality is cataract formation, which increases dramatically after age 50. Virtually everyone over age 85 has some degree of senile cataract formation, which reduces acuity, affects colour perception and increases glare. Glaucoma is the leading cause of blindness in adults, and its incidence also increases dramatically over age 40. Macular degeneration, usually resulting from changes in the eye's vasculature, is another important cause of blindness in those over 55. Diabetic retinopathy is the leading cause of blindness for all ages, but its probability increases with the duration of the diabetic disease process, and therefore obviously increases with age (Kapperud, 1983). Together, these pathological processes, which are either characteristic of aged eyes or at least increase in frequency over the lifespan, result in a substantial loss of visual function during aging.

The consequences of both normal aging and disease processes for visual function are that large portions of the late middle-aged and older population have reduced light availability at the level of the retina, and will be especially susceptible to the effects of any additional attenuation of environmental lighting in the home. Since the proportion of the general population composed of these age groups is increasing in Canada, these facts may have significant public health implications. However, people with cloudy ocular media or cone deficiencies may actually be helped by reducing shorter wavelength components of light sources. Recent studies suggest that some people experiencing chromatic aberrations and other perceptual distortions when reading text can be helped by screening out short wavelengths (<450 nm) from their light sources (Zigman, 1992).

The effects of reduced visual function in aging may include degradation of some mechanisms that depend on light exposure, such as daily rhythm entrainment by environmental lighting cycles. There is evidence that several features of

circadian system function alter with age, including neural activity in suprachiasmatic nucleus cells, and the size of the daily peak-to-trough variation in several physiological rhythms, including that of pineal melatonin (Brock, 1991; Mirmiran *et al,* 1989; Copinschi and van Cauter, 1995). The integrity of the circadian rhythm entrainment mechanism may be compromised by such neuroendocrine changes, especially when combined with reduced effectiveness of light exposure, as outlined above.

Another predictable consequence will be the increased use of artificial light to compensate for reduced sensitivity, as well as for reduced exposure to natural light in more sedentary, house-bound individuals. Insofar as tinted windows reduce and alter spectral properties of home lighting, their installation will promote increased use of artificial lighting sources by these segments of the population. One implication, therefore, is that one has to consider any potentially harmful or beneficial results of increased use of artificial lighting sources as an indirect consequence of any manipulations that reduce illumination through windows in the home or office (Hollwich, 1979).

G. Summary

Windows have a profound impact on room occupants. While most people find windows, the sunlight they convey, and the view they allow intrinsically desirable, there are limitations imposed on this desirability by several features of window illumination. Among these are the thermal burden imposed by insolation during warm seasons, the loss of heat in winter, and the fabric-fading effects of UV light. Attitudes toward the costs and benefits of windows are strongly influenced by the occupants' opportunities to regulate light transmission with blinds as well as to control heating, air conditioning and lighting within the room. Tinted windows, like windows with blinds, reduce the amount of illumination provided and promote increased use of artificial illumination. As a result, one consequence of altering window illumination is to increase the impact on occupants of whatever potentially beneficial or harmful effects artificial lighting sources might have.

Because few studies have compared the effects of natural solar radiation with those of artificial illumination, there is little information available with respect to any differential effects of manipulating the balance of these sources of illumination. More research studies have addressed comparisons among different artificial sources, some of which are claimed to produce light that more closely matches the spectrum of solar radiation than do other artificial sources. This literature therefore has the potential to provide some indirect insights into the effects of solar illumination versus artificial illumination with a non-solar spectrum, as well as into the effects of distorting solar radiation with tinted glass windows. Unfortunately, most studies of this issue are seriously flawed and their often extravagant claims of benefits from so-called "full-spectrum" lighting are not credible. These studies suffer both from limitations inherent in field studies, as well as from major flaws in research design and data analysis that should have been avoidable. This area cries out for appropriately designed and conducted studies to address some of these issues.

More questions about the presumed benefits of full-spectrum lighting are raised by a study (Sterling and Sterling, 1983) that suggested that such lighting may under some circumstances contribute to the generation of toxins that can lead to some symptoms of sick-building syndrome. The interaction of lighting with other environmental factors in the generation of these symptoms deserves further analysis.

Studies of windowless environments have yielded contradictory results, and no clear consensus has emerged. The effects of windowless environments must be related to a variety of environmental features, including external temperature, adequacy of heating and cooling control, adequacy of ventilation and the types of tasks being performed in the environment. Studies published so far on this issue have either been too small in scale or limited in time to address these issues, or they have been marked by confounds that make interpretation difficult or impossible.

Among special populations that may be affected differentially by changes in environmental illumination conditions, the elderly are of particular concern. Aging has dramatic effects on ocular characteristics that reduce the intensity and alter the spectral characteristics of retinal light availability. Coupled with other changes in elderly populations these alterations have the capacity to cause significant physiological effects. For example, circadian rhythm organization is altered in old age, and ocular abnormalities may further contribute to changes in daily rhythm adaptation to environmental cues. In addition, the elderly may be exposed to less environmental illumination outside the home than younger people, so that the spectral and other properties of home illumination may be more salient physiologically for them than it is for other groups.

Appendix A

Analysis of Full-Spectrum Lighting Studies

The first study reviewed is that by Mayron *et al* (1974), who claimed that full-spectrum lighting reduced hyperactivity in children in classrooms. This claim should be regarded with caution since the numbers of students and classes studied were very small, and the report did not note whether the classrooms had windows, nor how much time students spent exposed to natural illumination during the day. Either of these factors could drastically alter one's assessment of the effectiveness of the lighting manipulation. In addition, the lighting fixtures holding the full-spectrum sources were also modified in two additional ways to presumably reduce "soft X-ray" emissions and "low-frequency electromagnetic radiation". The modifications that were supposed to produce these changes were not described, nor was any evidence presented that such emissions occurred initially or were subsequently altered by the undescribed modifications. In any case, given these additional confounded modifications, if any changes in behaviour had occurred, it would not be possible to attribute them to the different spectral characteristics of the two types of light sources.

The second full-spectrum study discussed was the Alberta school study (Hathaway *et al,* 1992). As mentioned briefly, a critical concern in this study is the likelihood that the results reflected pre-existing social or economic differences among schools, since each treatment was applied to a different schools. This leads to several concerns: First, there was no attempt to match the five treatment groups (schools) for initial scores on any of the measures taken. Second, there was no attempt to examine the records of the students in the different classes retrospectively to determine whether the observed trends were a continuation of their previous trends or a shift in response to the lighting manipulation. Third, there was no attempt to compare the target classes that were exposed to an altered lighting environment to unmanipulated classes in the same schools, which could have provided a reasonably comparable control group. Many of the results reported could more easily be explained as the result of pre-existing socioeconomic, cultural or public health differences among the five schools studied than as the result of the lighting manipulation.

The only attempt to address this possible confound was an analysis of the *self-reported* contents of lunches brought to the schools by a subset of children. This analysis of macronutrient and sugar content of the lunches was predicated on the idea that cultural or socioeconomic differences among schools would be reflected in these features of the lunches. This assumption, however, is entirely gratuitous and was not supported by reference to any published documentation. Nor did the authors even address the thorny issue of the reliability and validity of retrospective self-reports of meal contents by 12-year-olds, which one can guess are probably of doubtful reliability.

In addition, the numbers of students participating in this "control" experiment was inadequate and differed dramatically among the schools (and, therefore, among the treatment conditions). While different data tables in this report show measures for inexplicably changing numbers of students, one can guess that one school had a participation rate in the dietary "control" study of 19/67 students (~28%), while another had a participation rate of 37/44 (~84%). Interestingly, the school with the lowest participation rate in this "control" study had the highest self-reported caloric intake, highest sugar intake, most dental caries, worst attendance and lowest rates of physical development of the five schools (although some of these differences were not significant). This rather broad hint that there were pervasive pre-existing differences among the schools was ignored by the report's authors. Instead, the differences in caries, school attendance and physical development observed in this one school were arbitrarily attributed to its lighting environment. This obviously inadequate approach should have been replaced by a systematic analysis of socioeconomic levels, ethnicity, health care and social factors that might have differentiated the five schools studied.

Another serious problem was that, although the authors suggested that teachers and administrators were not likely to have any expectations of particular benefits from full-spectrum lighting (no documentation provided of their views), one of the schools that received these lights did so because its staff had made an "informal request" to the provincial education department for full-spectrum tubes to replace their cool-white lamps. Clearly, the staff had some strong expectations of benefits from this lighting, or they would not have requested it; this process of self-selection for participation in the study makes a mockery of any attempt at control procedures.

While the present report was in preparation, another report appeared that addressed the issue of full-spectrum lighting effects in detail (Veitch, 1994). Among the components of the Veitch (1994) report was a summary by Hathaway (1994) of the results of the Alberta school study, and a presentation of arguments in defence of that study's conclusions. A detailed response to these arguments is not possible, but a few key elements should be examined in order to better address both the essential problems inherent in the analysis of this study and the general difficulties with the design and execution of such field studies.

One critical problem is that it is apparent from an overview of all the data that one school site (Site 1) differed from all the others along most dimensions, and it also had non-full-spectrum lighting. The authors attempted to deal with these apparent pre-existing differences by the use of an analysis of covariance (ANCOVA) on achievement results. Unfortunately, the application of this method is inappropriate for the purpose intended, and the authors reveal their lack of understanding of the problem and of the application of this method in their description of the analysis (p. 22) (Hathaway, 1994). The authors believed that by application of an ANCOVA they could "control" for pre-existing differences in achievement scores among classes. The flaw in this logic is that the differences in baseline achievement scores are not what needs to be controlled for. In fact, the

very existence of such differences (which is strongly implied in the text, but unfortunately not documented), demonstrates one fundamental flaw in the study and in its analysis.

If students at Site 1 had low achievement scores before entering the study because of socioeconomic or other differences from students at other sites, an ANCOVA that takes their low baseline achievement scores into account still fails to control for the underlying causal factors that led to these score differences. If the rate of achievement gain at Site 1 during the two-year study was identical to the low achievement rate which earlier resulted in lower baseline scores, the ANCOVA would only factor out the role (if any) of starting at a lower baseline. It cannot and does not factor out the role of the socioeconomic and other factors that probably led to the lower baseline achievement scores in the first place. These factors continued to operate during the study, and undoubtedly led to continued low achievement in these students. Unless it can be demonstrated that the *rate* of achievement gain before the installation of a particular lighting system differed from the *rate* of gain after the installation, it is impossible to claim that the lighting altered academic achievement.

Even if one were to ignore what might seem to some readers a discussion of statistical subtleties, and one simply compares the actual data to the published claims, the conclusions of Hathaway *et al* (1992) and Hathaway (1994) are not supportable. It is difficult to reach any conclusion other than that the data are misrepresented by the summarized claims related to achievement scores and other variables. For example, in the Abstract summarizing the results (p. 11), Hathaway (1994) states that "students under full-spectrum fluorescent lamps with ultraviolet supplements developed fewer dental cavities and had better attendance, achievement, and growth and development than students under other lights". This statement clearly indicates that UV supplementation improves scores on these variables. The issue of achievement scores is addressed by the summary data presented in Fig. 5 (p. 22), (Hathaway, 1994). The achievement gain for the UV-supplemented site was 1.96 grades, while the highest achievement rate was actually that of a non-UV-supplemented classroom (Site 2: 2.25 grades). Another non-UV-supplemented site had a gain of 1.88 which likely would not differ statistically from the UV-supplemented site's score. The statement in the Abstract is clearly false in its implications with respect to UV supplementation effects on academic achievement (as it is for school attendance). Unfortunately, such deceptive summaries are often the only information made available to the public or to school administrators.

A careful analysis of the so-called nutrition data and the data on rates of formation of dental caries also reveals that the results provide no support whatsoever for a role for UV supplementation in producing the outcomes observed. The results are much more parsimoniously attributable to pre-existing differences among study sites, for which the data provide suggestive evidence. Such an inadequate study and flawed analysis are equally incapable of demonstrating that UV supplementation is ineffective; the data, in effect, prove nothing at all. It remains possible, of course,

that UV supplementation has some beneficial effects, but the assertion that the Hathaway *et al* (1992) study provides substantial evidence for such effects is not tenable.

A third study, by Wohlfarth and Sam (1981) is also characterized by major problems in design and execution. Among the numerous serious flaws in the study were the following: the people scoring the children's behaviour were aware of the treatments throughout; the teachers were fully aware of what the study hoped to find; the supposed "effects" of the initial change in lighting and colour persisted unaltered when the colour and lighting reverted to the original condition; an unspecified number of confounded changes were made simultaneously to room design, furnishings, colour and lighting; the claimed effects of lighting were as clear in two blind children as in five sighted children; and the statistical analyses of the data were too poorly described to be comprehensible, but they appeared to be quite inappropriate. Clearly, such studies provide no experimental support for a role of lighting in modulating behavioural problems in children. Instead, the results of such studies amount merely to uninterpretable anecdotes. It would be reckless to base public health or educational policy decisions on such anecdotal claims.

Appendix B

Windowless Classrooms

A study of children in classrooms with different lighting conditions (Küller and Lindsten, 1992) observed only one physiological difference among children in these rooms. The actual functional significance of the differences observed in cortisol excretion in one season is completely unknown: there is no indication whether the differences reflect a harmful, beneficial, or neutral feature of the physiology of the children in the windowless classroom lacking full-spectrum lighting. In addition, these endocrine results are very difficult to interpret because only a single morning value was obtained for each child in each season. Because of the pronounced diurnal variation in cortisol secretion, and the possibility that its secretion is timed or regulated differently in different seasons, it is imperative to obtain more complete daily profiles and more than a single sample per season from each child.

What is most remarkable about this study, however, is that the authors largely ignore the fact that the four rooms differed dramatically in the *amount* of illumination provided, as well as the spectrum of illumination. One windowless classroom had intensities of 200-250 lux at all times of year, while a classroom with a large skylight had winter intensities of 300-750 lux and spring values of 650-6950 lux (Küller and Lindsten, 1992). Any effects observed might be attributable to the dramatic seasonal differences in intensities in some rooms and not in others (a 30-fold difference in the extreme case). The potential effects of the "natural" spectrum of illumination cannot be assessed if they are confounded by dramatic differences in lighting intensities. If the differences observed among children in these classrooms were demonstrated to be reliable, the light intensities experienced in the different classrooms should be considered a potential causal factor.

Leaving aside the issue of the cause of the one observed difference in endocrine activity, there is no hint that this difference had any negative impact on the children or reflected any pathological condition. Despite the complete lack of evidence for beneficial effects of window illumination for children in this study, and despite the failure to even address a critical confound in the study, a footnote indicated that following the study and as a consequence of it, the two windowless classrooms were reconstructed in order to introduce windows. It may be that putting windows into classrooms is a good idea, or a bad one (Boyce, 1981), but to base a decision on reconstructing classrooms on the flimsy evidence of this study is not supportable. Without commenting on whether a windowless classroom is desirable, it seems clear that personal beliefs about these issues will guide decisions in the absence of relevant and reliable data.

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