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CLINICAL RESEARCH

2017 CAO Congress
Poster Session Abstracts

PRACTICE MANAGEMENT

An Optometrist's Guide
to Protection from Burglary

CLINICAL REVIEW

Sun Safety and the Eyes



CANADIAN ASSOCIATION OF OPTOMETRISTS
ASSOCIATION CANADIENNE DES OPTOMÉTRISTES

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On the Cover

The effects of UV exposure
begin in childhood.



B. Ralph Chou, MSc, OD, FAAO
Editor-in-Chief/Rédacteur en chef

The reappearance of the UV index in Canadian weather forecasts heralds the beginning of the summer season of outdoor activities. This year, the Canadian Dermatology Association (CDA) has declared June 5 to 11 “Sun Awareness Week”, during which it will jointly issue public service announcements with the Canadian Ophthalmological Society (COS) reminding the Canadian public to protect their eyes and skin from damaging solar UV radiation. There is also a website devoted to sun protection for outdoor workers (sunsafetyatwork.ca).

Canadian Optometry has not been left out of the loop. In late April 2017, Canadian optometrists participated in a workshop on UV protection for millennials in Ottawa. This followed participation in a National Steering Committee for Consensus on Content for Sun Safety Messages. The Steering Committee published a paper in the Canadian Journal of Public Health in 2016, which laid out the consensus on sun safety messaging and the process used to get there.

Dr. Ben Giddens, who was the OAO representative, provides a review of the facts on sun safety and the eyes. Although Canadians protect themselves from solar UV radiation in the summer, they really should do this all year. Dr. Giddens reminds us that solar UV radiation isn't only dangerous to the eyes and skin when summer skies are clear. Skiers have tanned or sunburned faces after a day on the slopes, and photochromic lenses activate under cloudy skies for a reason.

Also in this issue you will find the abstracts for the poster session at this year's CAO Congress. The posters reflect the wide range of today's optometric research. I hope to see many of you at the posters. ●

Le retour de l'indice UV dans les bulletins de météo canadiens annonce le début de la saison estivale des activités en plein air. Cette année, l'Association canadienne de dermatologie (ACD) a déclaré la semaine du 5 au 11 juin « Semaine de la prudence au soleil », au cours de laquelle elle publiera des communiqués d'intérêt public de concert avec la Société canadienne d'ophtalmologie (SCO) afin de rappeler aux Canadiens qu'ils doivent protéger leurs yeux et leur peau contre les rayons solaires UV dangereux. Il y a également un site Web consacré à la protection solaire pour les personnes qui travaillent à l'extérieur (sunsafetyatwork.ca).

Le milieu de l'optométrie canadien n'est pas resté à l'écart. À la fin du mois d'avril 2017, les optométristes canadiens ont participé à un atelier sur la protection contre les UV pour la génération Y à Ottawa. Cette participation faisait suite à un engagement au sein d'un comité directeur national dont l'objectif était de parvenir à un consensus sur le contenu des messages portant sur la protection solaire. En 2016, ce comité a publié dans la Revue canadienne de santé publique un article qui présentait ce consensus et le processus ayant permis d'arriver à celui-ci.

Le Dr Ben Giddens, qui représentait l'Association des optométristes de l'Ontario (AOO) au sein du comité, passe en revue les faits sur la protection solaire et les yeux. Les Canadiens se protègent contre les rayons solaires UV en été, mais ils devraient le faire toute l'année durant. Le Dr Giddens nous rappelle que les rayons solaires UV ne sont pas seulement dangereux pour les yeux et la peau en été lorsque le ciel est dégagé. On peut voir le bronzage – les coups de soleil même – sur les visages des skieurs après une journée sur les pentes; et ce n'est pas sans raison que les lentilles photochromiques s'activent sous des ciels couverts.

Vous trouverez également dans ce numéro les résumés pour la séance d'affiches du congrès de l'ACO de cette année. Les affiches reflètent la grande variété de sujets sur lesquels les chercheurs en optométrie se penchent en ce moment. J'espère vous voir nombreux à cette séance. ●

Sun Safety and the Eyes

Ben Giddens BSc, OD
Optometrist
Family Eye Care Services

Abstract

The profession of optometry has a duty to advise the public about the short- and long-term adverse ocular effects of ultraviolet exposure, and the lifelong protective measures that should be adopted. This paper describes the most common ocular sequelae of, and provides a contemporary understanding of the mechanisms behind, ultraviolet damage. Emphasis is placed on how the eye and eyelids differ from the skin in terms of why and when the eye needs protection.

PURPOSE

Exposure to solar ultraviolet radiation (UVR) can harm the skin, eyes and immune system.^{1,2} Although the public should be encouraged to take measures to protect itself from UVR, such measures were last reviewed for consensus by all the relevant health care providers and public health officials in Canada in 1994. As a result, the “National Steering Committee for Consensus on Content for Sun Safety Messages” was formed with the mandate of promoting sun safety to the public. The Committee invited a team of national representatives to establish a common understanding of the science of sun and UVR damage and to provide succinct messaging about UVR protection that all of the participants could accept. Cancer Care Ontario asked the Ontario Association of Optometrists (OAO) to participate, and OAO acted as a national representative for the profession of optometry. In February, 2016, the final consensus statement manuscript was sent to the Canadian Journal of Public Health.

This process led to an awareness that optometry in Canada needs to play a bigger role in promoting sun safety for the eye, beyond the scope of the national consensus statements. This paper aims to present background science and research about the mechanism of ocular and periocular UVR damage to assist primary care optometrists in dispensing sound advice to patients.

In Canada, we do enjoy our sunny days and, as optometrists, casual conversation with our patients usually touches on the weather. However, we don’t often discuss the potential harm of UVR, evidence of sun damage to our patients’ eyes, or lifelong protective measures to adopt. As a rule, optometrists should habitually discuss these matters with our patients.

WHAT IS UV?

The broader electromagnetic spectrum includes the visible spectrum, which ranges from shorter-wavelength blue light (beginning at around 400nm) to longer-wavelength red light (ending at around 700nm). The wavelength bandwidths of UVR are commonly specified as 220 to 280nm for UVC, 280 to 320nm for UVB and 320 to 380nm for UVA. The World Health Organization (WHO), European Council of Optometry (ECO) and others have adopted 400nm as the upper level of UVA (TS UV EP).¹ The UV that reaches the Earth’s surface is comprised of about 95% UVA and 5%UVB. UVC is effectively filtered by the atmosphere, and is not considered a threat to the skin or the eyes because the recognition of stratospheric ozone depletion in the 1980’s led to a ban of ozone-depleting substances.³ Without this ban, it has been estimated that UV indices of 30 (see below) would have been recorded by the year 2060.⁴ Although the ozone layer is on track to recovery, estimates vary as to how long this will take, and we are currently still exposed to higher levels of UVB than pre-ozone-depletion.⁵

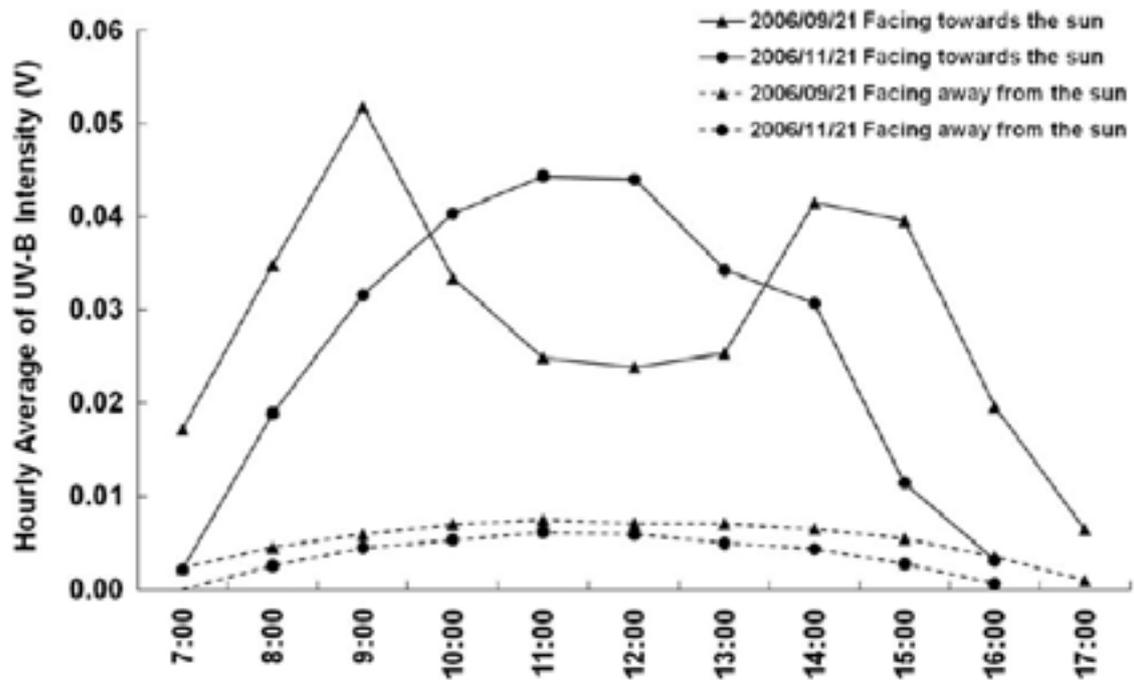
The UV index is used as a daily measure of the UV hazard in Canada. This index is based on the minimal erythema dose (MED) that produces a just-noticeable erythema (reddening) of the skin. Although skin sensitivity varies with skin pigmentation when the UV index is 3 or less, the average Caucasian will have a MED time of about 1 hour. For a UV index of 10, the MED time drops to about 15 minutes.

HOW DOES UV REACH THE EYE?

Since people typically don't look directly at the sun, there has been some discussion as to whether the UV index is a useful guideline for determining ocular risk due to solar radiation on any given day. Sasaki et al.⁶ demonstrated that the UV index is not accurate for assessing potential damage to the eye, and that sun protection is probably needed over more hours in a day for the eye than for the skin.

Sasaki used cranial mannequins with embedded sensors and measured the UVB dose from sunrise to sunset on Sept 21st and Nov 21st, 2006. The mannequin heads were maintained in a walking posture, but were moved throughout the day so that they faced (but did not look directly at) the sun as it arced from east to west. The study was done in Japan at a latitude of 36 degrees, whereas Canada's southern border varies from 43 degrees in Ontario, to the 49th parallel, in Western Canada. The study concluded that, for most of the year, the peak exposure time for the eye was between 8 and 10am and 2 and 4pm, not when the sun is at its zenith and UVB is at its maximum (Figure 1). The study also showed that from fall to winter, although the total UV exposure for a flat, horizontal surface drops by 29%, the exposure for the eye drops by only 8%.⁶

Figure 1: Hourly average of UVB intensity (in Volts) in the central eye when facing towards and away from the sun. From Sasaki et al. 2011 (Ref. 6).



The sky appears to be blue because blue light is selectively scattered by the atmosphere (Rayleigh scattering). Shorter wavelengths of UVR are more strongly scattered by the atmosphere, and the eye is exposed to varying degrees of UVR from all directions. In fact, more UV reaches the eye from scattered sunlight than from direct sunlight.⁷ It is possible to sit in the shade of a tree all day and still exceed a safe amount of (indirect) exposure.⁸ Remember, if someone sits in the shade but can still see blue sky, UVR is reaching their eyes. The sense of protection from direct

shade is probably due to the drop in heat. Long-wavelength infrared radiation provides radiant heat, since it comes directly from the sun and does not scatter in the atmosphere as does UV, so people can develop a false sense of safety when they step into the shade. Sliney offered a good way to think about UV exposure: “If one could see only in the UV-B spectrum, a clear sunny sky would appear to be a sun barely visible through a very heavy fog or haze.”⁷

Although thick cloud cover can attenuate solar UVB, scattering from the sides of cumulus clouds close to the solar disk can enhance UV exposure by up to 20%.⁹ In addition, clouds near the horizon opposite the sun can reflect UV and increase the total exposure to a level higher of UV than if the sky was clear.¹⁰ The influence of clouds on UV is still an important area of study. The amount, height, type and thickness of clouds and their effects on UVA versus UVB are all variables that can either enhance ocular risk or diminish it to almost zero.^{11,12}

Reflected UV can be particularly hazardous to the eye in some environments. For example, fresh snow is very reflective of UVB. A study of UVB reflectance at a variety of American locations around the 40th parallel demonstrated that fresh snowfall can reflect between 50 and 80% of the incident UVB. A study in New Zealand at the 45th southern parallel showed that the enhancement of UVB irradiance in midwinter from snow reflectance can exceed 30%.¹³ Sand can reflect between 8 and 18% of UVB and concrete paving, wooden walkways and water also enhance the ambient UVB.¹⁰ The relevance of reflectance always needs to be considered with respect to the posture of the eye. As an example, people usually do not get photokeratitis while sunbathing despite the fact that the minimal dose of UVR required to cause photokeratitis is less than that required to cause a sunburn.¹⁰ However, when an individual walks along while looking at the ground in front of them, the reflected UV does more ocular damage than the sun directly above.

If the surrounding landscape is flat and treeless, the eye will be exposed to much more UVR from the sky above the horizon than if the landscape had buildings, trees, or other geographical features that obscured the sky,¹⁰ which suggests that people who live on the Canadian prairie need more UVR protection for their eyes on sunny winter days.

AREAS OF DAMAGE

Spectral energy increases exponentially as the wavelength decreases: the potential for tissue damage with exposure to UVR of 300nm is 600X that at 325nm.¹⁴ Similarly, visible blue light has more potential for harm than visible red light. We recently provided evidence of damage to the skin and retina at wavelengths in the visible spectrum up to 490nm.¹⁵⁻¹⁷

SKIN

How does damage to the skin occur? DNA readily absorbs the higher energy of UVB; this causes direct and instant structural alterations.¹⁸ UVB is the cause of sunburns and was previously faulted as the main cause of skin cancer. It is now accepted that longer-wavelength UVA is also mutagenic, and research at Yale may have recently identified the mechanism. About half of the DNA alterations due to UV exposure were shown to be caused by energetic by-products of chemical chain reactions occurring in melanocytes that absorb UVA.^{19,20} Clearly, we need protection from both UVA and UVB.

The most superficial layer of the skin (epidermis) is comprised of several layers of squamous cells. The junction between the epidermis and deeper dermis is comprised of a layer of basal cells separated by occasional melanocyte cells. The cancers that can occur in these cells are named according to the cells in which they originate: melanomas are much more life threatening than squamous (SCC) or basal cell carcinomas (BCC).

Skin cancer is the most common cancer in Canada, with approximately 6500 cases of malignant melanoma and 76,000 cases of non-melanoma diagnosed in 2014.²¹ These figures are approximately equivalent to the number of lung, breast, colorectal and prostate cancers combined. It is estimated that 64% of melanoma and 90% of non-melanoma skin cancer is caused by UV damage.^{22,23} Even though skin cancer is one of the most preventable cancers, the incidence of melanoma in Canada is increasing: from 1986 to 2010, the incidence rose by 2% per year in men and 1.5% per year in women.²¹

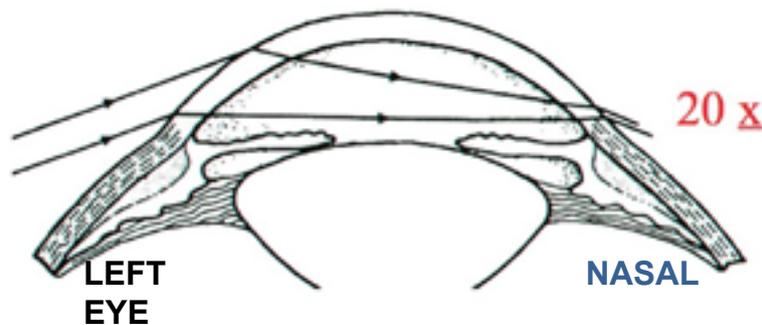
Both melanoma and non-melanoma (NMM) cancers are found on the eyelids; indeed, it is commonly stated that 5-10% of total skin cancers are found on the eyelid.²⁴ In 2011, 6.5% of all BCC and 1.3% of all SCC diagnosed in Canada were found on the eyelid.²¹ In addition, more than 50% of NMM were found from the neck up. It is clear that a proper hat and UV protective eyewear are necessary beginning at an early age.

OCULAR SURFACE INVOLVEMENT

Pterygia

Chronic UV damage to the ocular surface is most commonly seen in the form of pterygia, whereas acute trauma comes in the form of welder’s flash and snow blindness from UVB exposure. Coroneo deserves most of the credit for explaining the peripheral light focusing (PLF) effect, also known as the Coroneo effect (Figure 2).²⁵ UV light incident on the temporal cornea will focus through the anterior chamber onto the internal basal stem cells of the nasal limbus, increasing in intensity by approximately 20-fold. Direct sunlight would not otherwise be able to reach these deeper stem cells because it would be blocked by the superior limbal cells.²⁵⁻²⁷ Coroneo postulated that UV-damaged basal stem cells could form several types of new cells that could cross the limbal barrier and invade the cornea. He and others have subsequently produced a large volume of research aiming to establish the pathogenic mechanism by which epithelial cells then become involved in the fibrosis, angiogenesis and hyperplasia characteristic of pterygia.²⁵ Due to the tumour-like features of pterygia, a histopathological study of 100 excised pterygia was performed. The analysis provided evidence that pterygium is a disease of stem cells and also showed that pre-neoplastic disease such as primary acquired melanosis and ocular surface squamous neoplasia can co-exist with pterygia. The authors concluded that all excised pterygia should be subjected to histological evaluation.²⁸

Figure 2: From Coroneo 2011 (Ref. 25).



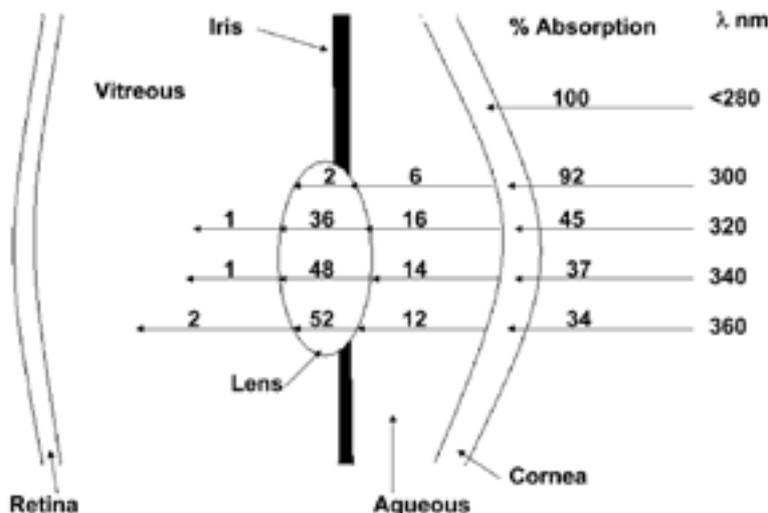
Pinguecula

Although pinguecula are believed to be associated with UVR exposure, a causal relationship has not been established.² An important study of people who worked on the water of Chesapeake Bay in Maryland showed a clear association between UVR exposure and an increased risk of pterygium and climatic droplet keratopathy, but only a weak association with pinguecula.²⁹ However, the known traits of elastoplasia and elastodystrophy found in pterygium and sun-induced skin damage are also found in pinguecula.³⁰

Cornea

The human cornea transmits all of the visible wavelengths of the EMR spectrum, but absorbs most of the shorter-wavelength UVB and essentially 100% of UVC.³ This absorption can lead to two different types of corneal response to UVR (Figure 3).³¹ Photo-keratitis is a superficial punctate keratopathy that is due to acute exposure to UVB. Also known as snow-blindness, it usually occurs after excessive exposure to UVB; while skiing, at altitude, or at the beach. Climatic droplet keratopathy is more commonly associated with chronic UVA and UVB exposure. Although it is more commonly seen in the tropics, it is also encountered in the Canadian Arctic where people are exposed to high levels of reflected UV.^{3,32}

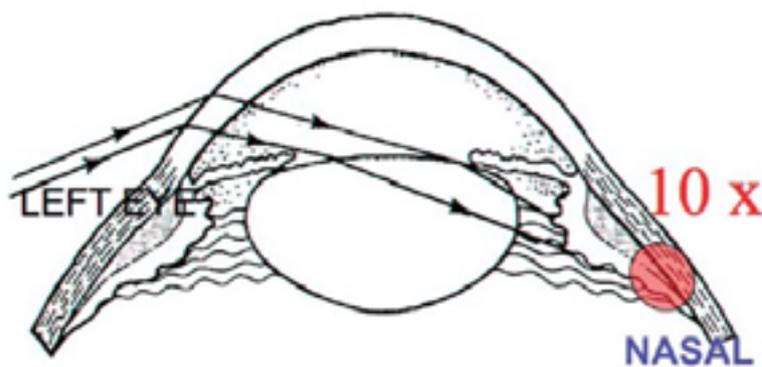
Figure 3: From Lucas 2011 (Ref. 31).



Cataract

The most common cataract seen by optometrists is the age-related nuclear sclerotic cataract (ARN). It begins as a yellowing of the lens and ultimately develops a darker smoky appearance that necessitates surgery. Although it is generally accepted that ARN cataracts are caused by oxidation of the lens, the role of UVR in enhancing progression is more speculative. After people reach middle age, a naturally occurring lenticular pigment is slowly converted into xanthurenic acid, which then begins to absorb UVR and forms harmful oxidative particles. At the same time, there is an age-related decrease in the production of anti-oxidant groups that allows lens proteins to become more damaged, causing further clouding of the lens.³³⁻³⁵ It is well accepted that cortical cataract is caused by UVR exposure. The Coroneo effect, which was established to demonstrate the formation of pterygia, also helps explain the common location of inferior-nasal cortical cataracts (Figure 4).²⁵ Temporal UV exposure, from a different angle than that which causes pterygium, enables the cornea to focus the UVR through the pupil and onto the inferior-nasal equatorial cells of the human lens. The equatorial region is the germinative area of the lens which likely results in the spoke-shaped cataract.^{25,36}

Figure 4: From Coroneo 2011 (Ref. 25).



Age-Related Macular Degeneration (AMD)

The initiation and progression of AMD are associated with genetic and environmental risk factors including age, cigarette smoking, white race, female gender, blue iris colour, obesity, nutritional factors and insufficient antioxidants in the diet.³⁷ A review of 465 articles by Sui et al. in 2012 concerning the association between AMD and sunlight exposure indicated that some individuals with higher levels of sunlight exposure are at a significantly increased risk of AMD.³⁸ Aging of the retinal pigment epithelial (RPE) cells and Bruch's membrane, impaired blood flow in the choriocapillaris and retinal exposure to UV and blue light have all been implicated in the process of degeneration.³⁹⁻⁴¹ The loss of RPE cells seems to lie at the core of AMD progression. RPE cells play a vital role in controlling inflammation caused by oxidative stress from various sources including constant exposure to light stimuli.^{2 42} Blue light has been shown to be the portion of the visible light spectrum that causes the most photochemical damage in animal RPE cells.² There are different types of oxidative particles that are collectively known as reactive oxygen species (ROS).^{37 39} The aerobic functioning of all human cells involves the production of ROS, but the retina is especially prone to the generation of ROS due to the high partial pressure of oxygen and exposure to UV and blue light.³⁷ Under normal circumstances, the retina responds to oxidative stress in part by increasing the production of antioxidants and breaking-down damaged proteins (proteolysis). If this system is overwhelmed, detrimental products such as intracellular lipofuscin and extracellular drusen can start to accumulate and cause visible evidence of AMD.⁴³

There is widespread support in the literature that UV and high-energy visible blue light are phototoxic to the retina and contribute to the detrimental process of AMD. However, UV exposure is not strongly touted as a primary cause of AMD because the yellowing of the aging crystalline lens blocks almost all UV transmission and optometrists usually see patients with AMD beyond middle age. A generally accepted figure in the literature is that the human lens will transmit 75% of near-UV light (300 to 400nm) until the age of 10, but by age 25, yellowing of the human lens will reduce the transmittance to 10%.⁴⁴ Although the human lens still transmits most of the visible blue light (400 to 500nm) at these ages, there is much greater absorbance with the elderly lens.^{33 44} We need to remind ourselves that AMD is usually a slowly progressive disease and generally takes decades before becoming visually disabling. Clearly, the maximum AMD-protective benefit of wearing UV-absorbing and visible blue light-blocking lenses begins in youth and diminishes with age.

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Uveal Melanoma

There is some evidence in the epidemiological literature that UV exposure may be a factor in primary malignant intraocular tumours in adults, although attempts to link outdoor work and chronic UV exposure to the development of uveal melanoma have been inconclusive.⁴⁵⁻⁵⁰ However, it has been shown that the carcinogenic effect of UV light on children, which can cause cutaneous melanoma, may be more important than that in adulthood.⁵¹ The relevant association is that the pathogenesis of uveal melanoma may be the same as that of cutaneous melanoma.

Eye Protection Considerations

All optometrists in Canada recognize the need for ocular UVR protection, and we need to address this issue routinely with our patients. Although many spectacle lens materials and coatings provide adequate UV blocking, a consideration of the frame in which the lenses will be mounted is also important. The work of Coroneo in demonstrating the damage caused by peripheral light coupled with the human tendency to turn away from the sun's direct rays tells us that we should attempt to fit our patients with close-fitting wrap-around frames as much as possible (Figure 5).⁵²

Figure 5: Close fitting wrap around frames



K. Clok (2012) Risk of UV exposure with spectacle lenses.

In addition to direct UV striking the eye from peripheral directions there is also a concern about the reflectance of UV from the back surface of lenses that do not wrap the face form and/or sit too far away from the eye. While clear lenses without an anti-reflective coating will reflect 4-6% of UVA and UVB from the back lens surface, lenses with anti-reflective coatings will reflect an average of 25% of most UV wavelengths.⁵³ This knowledge led Essilor SA (France) to develop and trademark E-SPF™. E-SPF provides a measure of the UV between 280 and 380nm that is transmitted directly through a lens plus the amount reflected from the lens back surface at an angle of 145 degrees:

$$E - SPF = \frac{1}{T_{UV0} + R_{uv145}}$$

For example, an E-SPF of 7 represents a low level of protection and an E-SPF of 50 represents a high level of protection.¹⁴ E-SPF refers only to the property of the lens and coating and does not take away the need for proper frame-fitting.

The same consideration regarding frame fit characteristics applies to pre-manufactured non-prescription sunglasses. Large frames with a wrap that are close-fitting provide the best protection. Canada does not have its own transmittance safety standard for sunglasses, and thus the American National Standard Institute (ANSI) Z80.3 label is seen most often: Class I lenses absorb at least 90% of UVA and 99% of UVB, while Class II lenses block at least 70% of UVA and 95% of UVB.⁵⁴ Packaging or labelling should be checked before dispensing any product to patients while remaining mindful that a significant amount of UV can reach the eye without passing directly through the lenses.^{55,56}

UV-blocking contact lenses offer good protection for the eye and should always be considered, especially for young patients who are more vulnerable to ocular UV damage, and for those engaged in outdoor activities. Of course, sunglasses would still be needed to protect the skin around the eye but at least the eye itself would have all-day protection while the contact lens is being worn.

OCULAR UV PROTECTION MESSAGE FOR THE PUBLIC

This article previously noted that the Sun Safety Committee sent a final document to the Canadian Journal of Public Health. The primary recommended protective action statements included comments about the UV index being highest between 11 am and 3 pm and that this was the time for maximum skin protection. Despite the urging of the eye care sub-committee representing optometry, ophthalmology, CNIB and other fields, a similar emphasis was not placed on the message that the eye and periocular tissue are most at risk outside these hours.

The American Cancer Society has provided the following statement:

“Ideally, all types of eyewear, including prescription glasses and contact lenses, should protect against UV rays. Some contact lenses are now made to block most UV rays. But, because they don’t cover the whole eye and surrounding areas, they are not sufficient eye protection when used alone.”⁵⁷

This Cancer Council Australia has provided the following statement:

“UV radiation exposure to the eyes is dependent on a number of factors and is not closely correlated to ambient UV levels and the UV index. Cancer Council Australia recommends protecting the eyes from UV at all times when outdoors during daylight hours.”⁵⁸

As primary eye care providers, optometrists need to routinely advocate for better UVR protection for their patients’ eyes. We see the deleterious effects of UV exposure and it is up to us to spread the message that the eye is at greater hazard throughout the day than the public would perceive from UV index messaging alone.

The eye care sub-committee as a whole suggested the following broad recommendation: “Under normal daylight conditions, wear sunglasses or prescription eyeglasses with UV-protective lenses when outdoors all year round.” Although this recommendation was not included in the final submission to the Journal of Public Health, something to this effect should be endorsed by provincial and national optometry associations, including the Canadian Association of Optometrists (CAO), and embraced by all of its members.

A final and telling observation that deserves consideration is that, of all the children who walk to and from school each day, rarely will a child wear sunglasses during this time. If this group is the most vulnerable due to their age and the time of day they are out walking, then there is much more work to do to protect them. ●

Perhaps one reason that we don’t see sunglasses on children and teens more often in Canada is that the ill effects of UV exposure are viewed as an adult disorder, whereas we know that it is a cumulative problem that begins with childhood exposure.

Another reason might be that sunglasses are perceived as merely an expensive status symbol, and parents don’t want to encourage consumerism in their children.

To promote the more widespread use of sunglasses, especially within the younger population, it would help if optometrists were to source and provide affordable yet fashionable sunglasses.

If there are any statements within the body of this article that you feel might resonate within your patient population, please feel free to cite them as a quote from the CJO and hang it on your office wall for the public to see.

REFERENCES:

1. Working Group IARC. *Risk of Skin Cancer and Exposure to Artificial Ultraviolet Light*. Lyon: World Health Organization International Agency for Research on Cancer, 2006.
2. Yam JC, Kwok AK. Ultraviolet light and ocular diseases. *Int Ophthalmol* 2014;34(2):383-400.
3. Oliva MS, Taylor H. Ultraviolet radiation and the eye. *Int Ophthalmol Clin* 2005;45(1):1-17.
4. Newman P, Oman L, Douglass A, et al. What would have happened to the ozone layer if chlorofluorocarbons (CFCs) had not been regulated? *Atmos Chem Phys* 2009;9(6):2113-28.
5. Cullen AP. Ozone depletion and solar ultraviolet radiation: ocular effects, a United Nations environment programme perspective. *Eye Contact Lens* 2011;37(4):185-90.
6. Sasaki H, Sakamoto Y, Schnider C, et al. UV-B exposure to the eye depending on solar altitude. *Eye Contact Lens* 2011;37(4):191-5.
7. Sliney DH. Geometrical assessment of ocular exposure to environmental UV radiation-implications for ophthalmic epidemiology. *J Epidemiol* 1999;9(6 suppl):22-32.
8. Parisi AV, Kimlin MG, Wong J, et al. Personal exposure distribution of solar erythemal ultraviolet radiation in tree shade over summer. *Phys Med Biol* 2000;45(2):349.
9. Mims III FM, Frederick JE. Cumulus clouds and UV-B. *Nature* 1994;371(6495):291.
10. Sliney DH. Physical factors in cataractogenesis: ambient ultraviolet radiation and temperature. *Invest Ophthalmol Vis Sci* 1986;27(5):781-90.
11. Aun M, Eerme K, Ansko I, et al. Modification of spectral ultraviolet doses by different types of overcast cloudiness and atmospheric aerosol. *Photochem Photobiol* 2011;87(2):461-9.
12. Parisi AV, Downs N, Turner J. Evaluation of the cloudy sky solar UVA radiation exposures. *J Photochem Photobiol B* 2014;138:141-5.
13. McKenzie R, Paulin K, Madronich S. Effects of snow cover on UV irradiance and surface albedo. *J Geophys Res* 1998;103:28785-92.
14. Krutmann J, Béhar-Cohen F, Baillet G, et al. Towards standardization

- of UV eye protection: what can be learned from photodermatology? *Photodermatol Photoimmunol Photomed* 2014;30(2-3):128-36.
15. Pfeifer GP, Besaratinia A. UV wavelength-dependent DNA damage and human non-melanoma and melanoma skin cancer. *Photochem Photobiol Sci* 2012;11(1):90-7.
 16. Remé C, Reinboth J, Clausen M, et al. Light damage revisited: converging evidence, diverging views? *Graefes Arch Clin Exp Ophthalmol* 1996;234(1):2-11.
 17. van Norren D, Gorgels TG. The action spectrum of photochemical damage to the retina: a review of monochromatic threshold data. *Photochem Photobiol* 2011;87(4):747-53.
 18. Schreier WJ, Schrader TE, Koller FO, et al. Thymine dimerization in DNA is an ultrafast photoreaction. *Science* 2007;315(5812):625-9.
 19. Sinha RP, Hader D-P. UV-induced DNA damage and repair: a review. *Photochem Photobiol Sci* 2002;1(4):225-36.
 20. Taylor J-S. The dark side of sunlight and melanoma. *Science* 2015;347(6224):824.
 21. Demers A, Sinclair, B., Chapter 7: Special Topic: Skin Cancers. *Canadian Cancer Statistics: Canadian Cancer Society*, 2014:96-7.
 22. Armstrong B, Kricger A. How much melanoma is caused by sun exposure? *Melanoma Res* 1993;3(6):395-401.
 23. Pleasance ED, Cheetham RK, Stephens PJ, et al. A comprehensive catalogue of somatic mutations from a human cancer genome. *Nature* 2010;463(7278):191-6.
 24. Cook BE, Bartley GB. Treatment options and future prospects for the management of eyelid malignancies: an evidence-based update. *Ophthalmology* 2001;108(11):2088-98.
 25. Coroneo M. Ultraviolet radiation and the anterior eye. *Eye Contact Lens* 2011;37(4):214-24.
 26. Hoover HL. Solar ultraviolet irradiation of human cornea, lens, and retina: equations of ocular irradiation. *Appl Opt* 1986;25(3):359-68.
 27. Podskochy A. Protective role of corneal epithelium against ultraviolet radiation damage. *Acta Ophthalmol Scand* 2004;82(6):714-7.
 28. Chui J, Coroneo MT, Tat LT, et al. Ophthalmic pterygium: a stem cell disorder with premalignant features. *Am J Pathol* 2011;178(2):817-27.
 29. Taylor HR, West SK, Rosenthal FS, et al. Corneal changes associated with chronic UV irradiation. *Arch Ophthalmol* 1989;107(10):1481-4.
 30. Austin P, Jakobiec FA, Iwamoto T. Elastodysplasia and elastodystrophy as the pathologic bases of ocular pterygia and pinguecula. *Ophthalmology* 1983;90(1):96-109.
 31. Lucas RM. An epidemiological perspective of ultraviolet exposure—public health concerns. *Eye Contact Lens* 2011;37(4):168-75.
 32. Cullen AP. Photokeratitis and other phototoxic effects on the cornea and conjunctiva. *Int J Toxicol* 2002;21(6):455-64.
 33. Roberts JE. Ultraviolet radiation as a risk factor for cataract and macular degeneration. *Eye Contact Lens* 2011;37(4):246-9.
 34. Roberts JE, Finley EL, Patat SA, et al. Photooxidation of lens proteins with xanthurenic acid: a putative chromophore for cataractogenesis. *Photochem Photobiol* 2001;74(5):740-4.
 35. Roberts JE, Wishart JF, Martinez L, et al. Photochemical studies on xanthurenic acid. *Photochem Photobiol* 2000;72(4):467-71.
 36. Lofgren S, Ayala M, Kakar M, et al. UVR cataract after regional in vitro lens exposure. *Invest Ophthalmol Vis Sci* 2002;43(13):3577-77.
 37. Tokarz P, Kaarniranta K, Blasiak J. Role of antioxidant enzymes and small molecular weight antioxidants in the pathogenesis of age-related macular degeneration (AMD). *Biogerontology* 2013;14(5):461-82.
 38. Sui G-Y, Liu G-C, Liu G-Y, et al. Is sunlight exposure a risk factor for age-related macular degeneration? A systematic review and meta-analysis. *Br J Ophthalmol* 2013 Apr;97(4):389-94.
 39. Lu L, Hackett SF, Mincey A, et al. Effects of different types of oxidative stress in RPE cells. *J Cell Physiol* 2006;206(1):119-25.
 40. Majji AB, Cao J, Chang KY, et al. Age-related retinal pigment epithelium and Bruch's membrane degeneration in senescence-accelerated mouse. *Invest Ophthalmol Vis Sci* 2000;41(12):3936-42.
 41. Tanito M, Kaidzu S, Anderson RE. Delayed loss of cone and remaining rod photoreceptor cells due to impairment of choroidal circulation after acute light exposure in rats. *Invest Ophthalmol Vis Sci* 2007;48(4):1864-72.
 42. Cheung LK, Eaton A. Age related macular degeneration. *Pharmacotherapy* 2013;33(8):838-55.
 43. Blasiak J, Petrovski G, Veréb Z, et al. Oxidative stress, hypoxia, and autophagy in the neovascular processes of age-related macular degeneration. *Biomed Res Int* 2014;2014:768026.
 44. Barker F, Brainard G. The direct spectral transmittance of the excised human lens as a function of age. *US Food and Drug Administration Report* 1991.
 45. Gallagher RP, Elwood JM, Rootman J, et al. Risk factors for ocular melanoma: Western Canada Melanoma Study. *J Nat Cancer Inst* 1985;74(4):775-8.
 46. Lutz J-M, Cree I, Sabroe S, et al. Occupational risks for uveal melanoma results from a case-control study in nine European countries. *Cancer Causes Control* 2005;16(4):437-47.
 47. Pane AR, Hirst LW. Ultraviolet light exposure as a risk factor for ocular melanoma in Queensland, Australia. *Ophthalm Epidemiol* 2000;7(3):159-67.
 48. Seddon JM, Gragoudas ES, Glynn RJ, et al. Host factors, UV radiation, and risk of uveal melanoma: A case-control study. *Arch Ophthalmol* 1990;108(9):1274-80.
 49. Shah CP, Weis E, Lajous M, et al. Intermittent and chronic ultraviolet light exposure and uveal melanoma: A meta-analysis. *Ophthalmology* 2005;112(9):1599-607.
 50. Tucker MA, Shields JA, Hartge P, et al. Sunlight exposure as risk factor for intraocular malignant melanoma. *N Engl J Med* 1985;313(13):789-92.
 51. Holman CAJ, Armstrong BK. Cutaneous malignant melanoma and indicators of total accumulated exposure to the sun: an analysis separating histogenetic types. *J Natl Cancer Inst* 1984;73(1):75-82.
 52. Sakamoto Y, Kojima M, Sasaki K. Effectiveness of eyeglasses for protection against ultraviolet rays. *Jpn J Ophthalmol* 1999;43(6):566-7.
 53. Citek K. Anti-reflective coatings reflect ultraviolet radiation. *Optometry* 2008;79(3):143-8.
 54. Guidance document for nonprescription sunglasses; Centre for Devices and Radiological Health, FDA; Department of Health and Human Services: Division of Ophthalmic Devices, 1998:1-14.
 55. Rosenthal FS, Bakalian AE, Lou CQ, et al. The effect of sunglasses on ocular exposure to ultraviolet radiation. *Am J Public Health* 1988;78(1):72-4.
 56. Sliney DH. Eye protective techniques for bright light. *Ophthalmology* 1983;90(8):937-44.
 57. American Cancer Society: Cancer and Sunlight exposure: Available at: <http://www.cancer.org/cancer/cancercauses/sunanduvexposure/sun-and-uv-exposure->, 2016.
 58. Cancer Council Australia: National Cancer Control Policy; Position Statement-Sun Exposure and Vitamin D Risks and Benefits: Available at:http://wiki.cancer.org.au/policy/Position_statement_-_Risks_and_benefits_of_sun_exposure, 2016.