Prog Biophys Mol Biol. Author manuscript; available in PMC 2012 August 01.

Published in final edited form as:
Prog Biophys Mol Biol. 2011 December ; 107(3): 349–355. doi:10.1016/j.pbiomolbio.2011.08.010.

Childhood exposure to ultraviolet radiation and harmful skin effects: Epidemiological evidence

Adèle C. Green[a,b,*], **Sarah C. Wallingford**[b], and **Penelope McBride**[a]

[a]Cancer and Population Studies Group, Queensland Institute of Medical Research, Herston Road, Brisbane, Queensland 4029, Australia

[b]School of Translational Medicine, Manchester Academic Health Science Centre, University of Manchester, Oxford Road, Manchester M13 9PT, UK

Abstract

We review the general amount and patterns of exposure to solar ultraviolet (UV) radiation that children and teenagers experience and the spectrum of UV-related skin damage that can occur as a result. Data about the amount of solar UV received by children and teenagers are relatively few but suggest that around 40–50% of total UV to age 60 occurs before age 20. Among white children, those with the palest complexions suffer the most damage. Comparisons of prevalence and incidence of outcomes in children and teenagers sharing common ancestry, but living at different latitudes, show that prevalence rates of photoaging and melanocytic naevi are higher in Australian compared with British children, and similarly for melanoma. Genetic risk for the majority of the melanomas in teens is a function of genes controlling naevus propensity and pigmentation in the skin. High numbers of naevi and freckles, red hair, blue eyes, inability to tan, as well as a family history are the primary determinants of melanoma among adolescents. Beyond the signs of skin damage seen in children are the latent effects observed later in adulthood. Childhood is believed to be a susceptible window for long-term harmful effects of UV, as evidenced by clear differences in skin cancer risk between child and adult migrants from high to low latitudes. Effective UV radiation protection from childhood is necessary to control both immediate and long-term harmful effects on children's skin.

Keywords

Childhood; Ultraviolet radiation; Melanoma; Skin cancer

1. Introduction

Through outdoor activities and recreation children can experience moderate to high levels of exposure to ambient solar ultraviolet (UV) radiation in the course of their daily lives. The organs most susceptible to UV-related damage are the skin and eyes, and this paper reviews available epidemiological evidence regarding harmful skin outcomes of solar UV exposure during childhood and teenage years. Children are at very low risk of UV-related skin damage compared with adults, but the most common UV-related skin diseases occurring in adults may be observed in the first two decades of life as well, namely photoaging (seen as premature mild skin wrinkling in teenagers) and specific pigmentary signs of UV exposure

*Corresponding author. Cancer and Population Studies Group, Queensland Institute of Medical Research, Herston Road, Brisbane, Queensland 4029, Australia. Tel.: +61 7 3362 0234; fax: +61 7 3845 3503. Adele.Green@qimr.edu.au (A. C. Green), sarah.wallingford@manchester.ac.uk (S.C. Wallingford), penmcbride@hotmail.com (P. McBride). .

Europe PMC Funders Author Manuscripts

such as freckling and development of melanocytic naevi (moles). After severe UV damage, benign keratinocytic skin tumours, actinic keratoses, and malignant skin tumours: melanoma, and basal cell and squamous cell carcinomas (BCC and SCC) develop, though among skin cancers, only melanoma occurs to any measurable incidence in childhood. On the other hand childhood appears to be a period in life when people are susceptible to initiation of latent harmful effects of UV manifest decades later in adulthood (Balato et al., 2007) (Thomas et al., 2007). Here we review the epidemiology of childhood actinic skin damage and conclude with evidence mostly from migrant studies of the role of childhood UV exposure in the development of adult skin cancers, in particular melanoma.

2. Ultraviolet radiation exposure in childhood and adolescence

Several epidemiological studies have provided estimates of sun or UV exposure during childhood and adolescent years, and the relative contribution of childhood UV to lifetime exposure. A few of these studies have relied on modelling and most estimates have been obtained from volunteers and/or selected groups.

An extensive review of early life sun exposure which summarised the mean UV exposure, proportion of the ambient UV radiation received and time spent outdoors daily based on 29 studies published between 1990 and 2005 (from Australia, Europe, Japan, Mexico, UK and USA), found the range of mean daily hours outdoors was 1.5 to 5.1 (Wright and Reeder, 2005). Among adolescents, weekend exposures of 3 hours per day for boys and 2 hours per day for girls was reported in various eastern Australian locations (Fritschi, 1993); approximately 1 to 4 hours a day outdoors between the hours of 10am and 4pm in summer were reported in US teenagers (Davis et al., 2002); around 4 hours mean daily summer sun exposure for young children in Sweden was noted (Boldeman et al., 2004); and almost 4 hours outdoors daily was recorded in young French adults during summer vacations (Autier et al., 1999).

Lifecourse sun exposure to age 59 years in Australia has also been analysed in a community-based study in southeast Queensland (McBride, 2009). Average daily duration of sun exposure through successive life periods was recalled by some 1150 randomly selected, Australian-born residents in the township of Nambour and was supplemented by information from prospective sun exposure diaries (McBride, 2009). The average number of hours spent daily in the sun in childhood was 4.1 for boys and 3.2 for girls in this population, higher than reported by Gies et al. (1998) in volunteer Queensland school children who spent between 1.7 and 3.0 hours median daily time in the sun.

Hours of sun exposure are a crude surrogate for amount of latitude-dependent solar UV exposure which is far more difficult to estimate. McBride (2009) estimated solar UV radiation using meteorological records of measured erythemally-weighted UV radiation during relevant calendar years in Nambour (adjusted for season and time of day of exposure), assuming people receive around 30% of measured erythemally-weighted solar UV radiation (Gies et al., 1998; Godar, 2005). Although childhood and teen years represent only 33% of the lifetime to 60, McBride (2009) estimated that the Nambour study cohort before age 20 had received around 50% of their total UV exposure to age 60. This estimate is broadly consistent with two other studies in southeast Queensland (Parisi et al., 2000, 1999) but differs materially from the approximate 25% before age 20 years of life exposure (to age 77) that was found for a Danish volunteer population (Thieden et al., 2004). However, since Danish adult exposure was averaged from findings in two distinct adult populations (62 indoor hospital workers and 22 golfers) this is likely to have diminished the accuracy of the Danish estimate as well as its generalisability. In a model proposed by Stern et al. (1986) the base case assumption was that around 50% of lifetime UVR exposure was

received before age 18 for an American (Stern et al., 1986) which is closer to the more rigorous Australian estimate than the Danish.

Thus in general the evidence suggests that annual exposures in childhood and early adulthood are similar and exceed middle adulthood (Parisi et al., 1999). As in all stages of life, before age 20 females spend less time in the sun than males, though the sex difference is less before age 20 than after (Gies et al., 1998; Godar, 2005).

3. Benign skin effects in children

3.1. Photoaging

In addition to dermatological assessment of the visible clinical signs of skin photoaging (Green et al., 2011), changes can be assessed by skin surface microtopography using silicone casts of the skin of the back of hand reflecting the level of underlying UV-induced deterioration of dermal elastin fibres (Battistutta et al., 2006). Skin damage scores range from 1 (least) to 6 (highest) with progressive flattening and creasing of the skin surface with increasing UV damage.

In otherwise healthy white-skinned children living in places of high ambient solar UV radiation such as Australia, early clinical evidence of photodamage in the form of photoaging is detectable as young as the early teens. Silicone casts of the skin of teenagers aged 13–15 years in 4 cities in eastern Australia showed 40% to 70% had mild skin damage (Fritschi et al., 1995) With continuing high ambient sun exposure during mid to late teens and early twenties, prevalence and severity of photoaging climbs steeply such that young men aged 20–29 yrs in the Queensland population show a 72% prevalence of moderate to severe photoaging and young women, 47% prevalence (Green, 1991). The severity of photoaging increases rapidly after the age of 30, with an overall average of 14% significant increase in odds of having higher grades of photoaging for every added year of age (Green et al., 2011) Other community-based studies of Australian adults show similar prevalence rates (Lucas et al., 2009), far higher than in European adults. In a hospital-based study in Leiden, The Netherlands, only 7% of 24–49 year old people attending an ophthalmology outpatient clinic had moderate to severe elastosis, clinically assessed by dermatologists (Kennedy et al., 2003) compared with around 35% of those aged 20–54 years in Queensland with equivalent clinical signs (Green et al., 2011).

Using the same surface microtopography assessment methods as in the Queensland study, Fritschi et al. (1995) found that schoolchildren aged 13–15 years in Scotland had prevalence rates of mild skin damage of 33% compared with the 40–70% in Queensland.

3.2. Melanocytic naevi

Estimates of naevus prevalence in the last two decades among white-skinned children living in countries at relatively high latitudes, such as Germany, show low numbers: a median count of 3 naevi at 2 years of age, climbing to a median of 10 at age 5 years, to 17 at 7 years (Bauer et al., 2005). These counts are substantially lower than those seen in very young children living at low latitudes such as in tropical Queensland, where median counts are around 20 at age 2; 60 at age 5; and 70 at age 6 years (Harrison et al., 1994).Kelly et al. (1994) and English et al. (2006) also showed that the level of ambient UV exposure in early childhood is positively and significantly associated with naevus prevalence in young Australian children.

More focused latitudinal differences have been confirmed in international comparative studies. When the presence of melanocytic naevi of a diameter of 2 mm or more on the body was assessed by the same methods in children aged 8–9 of similar ethnic origin living in

low-latitude Brisbane, Australia and in the high-latitude West Midlands region of England, naevus prevalence was almost 7-fold higher in the Brisbane children (Green et al., 1988). Fritschi et al. (1994) also performed a comparative study of melanocytic naevi in the 13–15 year olds in Australia and Scotland to test the hypothesis that children living in subtropical and tropical environments have more naevi than those of similar ethnicity living in temperate countries. Children in Brisbane had significantly ($P < 0.05$) more naevi over 2 mm in diameter on the right arm than those in Glasgow after adjusting for background complexion characteristics. The difference in the geometric mean number of naevi on the arm alone was much greater among boys (7.7 and 4.4 in Brisbane and Glasgow respectively) than among girls (7.3 and 6.7 respectively). Taken together, these studies support the role of early-life sun exposure in determining prevalence of naevi in childhood.

4. Skin cancers in children

Since the incidence of BCC and SCC among children and teenagers in general is negligible, only sporadic melanoma in children and teenagers has been considered in detail for the purposes of this paper.

4.1. Melanoma incidence

Cutaneous melanoma is rare in children and adolescents with only 2–3% of all cases observed in patients under age 20 years (Zhu et al., 1997). Consistent with melanomas among older age groups, rates increase with age among young people. While less than 0.5% of melanomas occur in the first decade of life (Karlsson et al., 1998; Zhu et al., 1997), a relatively sharp rise in cases occurs following puberty such that 15–19 year olds account for 85% of melanomas diagnosed in under 20 year olds (Baade et al., 2011).

As in adults, countries in the southern hemisphere have been noted to have much higher melanoma rates in children and young adults, compared with their northern counterparts. From 1987 to 1994 rates in Australia were 1/million in under 10 year olds and 30/million in 10–14 year olds (Baade et al., 2010). In comparison, rates in Northern England from 1968 to 1995 were 0.7/million in under 15 year olds and 9.6/million in 15–19 year olds (Pearce et al., 2003). Not only is there a latitudinal gradient between countries for childhood and adolescent melanoma, the increasing trend holds within countries as well. In American 15–19 year olds melanoma incidence increased 85% from 1973–2006 and was higher in southern registries (23.9/million) than in northern ones (14.5/million) (Hamre et al., 2002). There are also slight differences in the site of presentation of melanoma, by country. While trunk melanomas predominate in all males in this age group (Baade et al., 2010; Karlsson et al., 1998; Pearce et al., 2003), females in England in particular have an excess of lower limb tumours, so much so that the overall increases in rates have been driven mainly by increases at this site (Pearce et al., 2003).

Although the general trend over several decades has been for increasing melanoma rates, there have been a few reports of recent decreasing incidence. Rates among Australian children aged 10–14 years old significantly decreased by 8.5% per year from mid-1990's to mid-2000's (Whiteman et al., 1995). Similarly, Swedish population-based data reported a decline in incidence to 3.6/million in 1993–2002 from 5/million in the previous decade (Karlsson and Fredrikson, 2007). This, however, is not the case for countries that have laggedin implementing targeted primary prevention schemes, such as England, where the rates continue to increase at an alarming rate (approximately 4% per year) and recent rates (1982–2006) are higher than ever (5.2/million in males and 9.8/million in females in under 25 year olds) (Wallingford et al., 2010).

4.2. Melanoma mortality

Survival from melanoma in children and adolescents has significantly improved over time. A study in Sweden (1973–1992) showed overall mortality was 13% (median survival time of 3 years 7 months), with younger people showing higher rates of mortality (16% in 10–15 year olds versus 23% in 16–19 year olds) (Karlsson et al., 1998). More recently, however, melanoma survival in Swedish children and adolescents has been reported to be as high as 90% (1993–2002) (Karlsson and Fredrikson, 2007). Survival in under 25 year olds in England has also shown significant improvement from 1968–1995 with 5-year sex-specific survival at 64% in males and 75% in females (Pearce et al., 2003). When population-based data are pooled across Europe (1878–1989), children under 15 years show survival rates of 80% for boys and 78% for girls with head/neck, legs and arms having better survival than trunk, neck and scalp (Conti et al., 2001). In the US, overall survival in cases under 19 years of age relative to the general population was 89% from 1973 to 1996 (Hamre et al., 2002).

5. Host and environmental determinants of skin cancer in children

5.1. Skin and pigmentary characteristics

As for adults, differences in melanoma risk in children according to racial skin colour are clearly seen. In the National Cancer Database for all US hospital-based oncology patients (1985–2003), non-Hispanic white children comprised around 90 to 95% of all melanoma cases from aged 5 to 19 years (Lange et al., 2007). Within white children up to age 15, there have been very few population-based epidemiological studies of constitutional risk factors for incident primary melanoma. The largest was conducted in Queensland in the mid-1990s (Whiteman et al., 1997b), based on 61 cases notified to the Queensland Cancer Registry in the period 1987 to 1994. Personal information was collected from cases, age- and sex-matched controls and parents, and skin examinations were performed. Children with heavy facial freckling and inability to tan after sun exposure were at increased risk for melanoma. After adjusting for these and other factors, the strongest determinant of melanoma risk found among these Queensland children was the presence of more than 10 naevi greater than 5 mm in diameter, which independently increased the child's risk by 7-fold (Whiteman et al., 1997b).

Similar findings were reported in the only known population-based case–control study of risk factors for melanoma in adolescents (15–19 years), again conducted in the high-risk state of Queensland based on approximately 200 cases and a similar number of controls (Youl et al., 2002). The strongest independent risk factor associated with melanoma in adolescents was again the presence of many naevi, specifically more than 100 naevi of 2 mm or more in diameter, which increased melanoma risk by around 40-fold. Red hair, blue eyes, inability to tan after prolonged sun exposure and heavy facial freckling were also risk factors (Youl et al., 2002).

5.2. Genetic susceptibility

5.2.1. Melanoma—Of all melanoma cases, 5% to 12% arise in people with a family predisposition for the disease (Berg et al., 2004; Nagore et al., 2005; Soufir et al., 2004; Tsao et al., 2000). Young age at diagnosis is associated with familial melanoma, along with presence of dysplastic naevi and development of multiple primary tumours (Nagore et al., 2005). Knudson's hypothesis suggests that cancers arising at very young ages may result from mutations to key regulatory genes passed through the germline (Soufir et al., 2004; Whiteman et al., 1997a). Germline mutations in CDKN2A gene and to a lesser extent CDK4, have been found in familial melanoma patients (Berg et al., 2004; Nagore et al., 2005; Soufir et al., 2004; Tsao et al., 2000; Whiteman et al., 1997a). In families carrying these mutations, the median age at diagnosis is lower than for nonfamilial melanomas

(Nagore et al., 2005). However, a low prevalence of CDKN2A mutations among early-onset melanoma cases occurring in childhood or adolescence has been shown in several studies, with only 1 or 2 cases observed, a much lower number than expected (Berg et al., 2004; Nagore et al., 2005; Tsao et al., 2000; Whiteman et al., 1997a). As a result, the primary genes associated with familial melanoma do not appear to have a role in the development of childhood and adolescent cases, though they do suggest that genetic alterations involved in early-onset disease have yet to be fully identified (Berg et al., 2004; Nagore et al., 2005; Whiteman et al., 1997a). It is further possible that the high genetic susceptibility shown for melanomas in this group could be related primarily to naevus count (Berg et al., 2004; Nagore et al., 2005; Soufir et al., 2004; Tsao et al., 2000; Whiteman et al., 1997a).

In addition, there are several genetic conditions which predispose affected individuals to melanoma, BCC and SCC. Melanomas in xeroderma pigmentosum patients often appear at early ages (median 8 years), but show similarities to melanomas of the elderly, the majority being of lentigo maligna subtype and often located on head/neck area, a body site associate with chronic UVR exposure (Spatz et al., 2001). Melanomas in these patients are associated with accumulation of unrepaired DNA, as in the elderly whose DNA lesions are instead the result of long accumulated UV exposure (Spatz et al., 2001). Mutations in the OCA2 gene in children with oculocutaneous albinism, the most common form of albinism, have also been associated with increased melanoma susceptibility (Jannot et al., 2005).

5.2.2. Basal cell and squamous cell carcinomas Most keratinocytic cancers occur on highly sun-exposed body—sites as a result of genetic susceptibility interacting with excessive UV exposure. BCC and SCC are very rare among healthy white-skinned children and adolescents despite relatively high UV exposure levels being attainable in tropical and subtropical locations. Hence these tumours are seen in those children with a predisposing genetic condition such as xeroderma pigmentosum, naevus sebaceous, and basal cell naevus syndrome (Efron et al., 2008; Orozco-Covarrubias et al., 1994). Vitiligo and albinism are also considered predisposing factors, but this may be due to the high degree of UV-induced damage to skin that occurs in these conditions (Efron et al., 2008). Actual incidence rates of keratinocytic cancers in under 15 year olds have rarely been reported, though one estimate of BCC incidence in the under 15 years age group is 1.9/10 000 patients (Orozco-Covarrubias et al., 1994).

5.3. Solar ultraviolet radiation exposure

5.3.1. Naevi—Associations between UV exposure during early life and naevi on the skin, suggested by ecological studies of latitude of residence in early life and naevi in children (section 3.2), have been borne out in several cohort studies. Anatomic sites of sunburn reported by parents of a cohort of white children aged 5–6 years in Colorado, USA, were examined in relation to sites of melanocytic naevi at age 7 (Dodd et al., 2007). In 2 years of prospective reports, most children (69%) were sunburnt once or more, and the face, shoulders and back were the sites most frequently sunburned. Naevus density was highest on the face, neck and lateral forearms and total number of sunburns was associated with total naevus prevalence. Site-specific sunburns were significantly associated with naevus prevalence on the back ($P = 0.03$ for three or more sunburns) but not on the face, arms or legs (Dodd et al., 2007). Non-Hispanic white boys had significantly more naevi than did girls from 6 years of age, and this difference was due to more small naevi and naevi on chronically exposed body sites (Crane et al., 2009). In another prospective study of naevi in a US cohort of elementary children in northeast USA, high total naevus count was related to spending 5–6 h outdoors between 10 AM and 4 PM weekly, and to a history of painful sunburns (Oliveria et al., 2009). In a longitudinal study of melanocytic naevi in Queensland, adolescents aged 12 and 13 years whose mean whole-body nevus count at baseline was

130.1 (SD = 69.9) were followed up for 5 years (Darlington et al., 2002). At end=of follow-up the mean count had risen to 215.5 (SD = 127.1) and in this group, shoulder and back counts were consistently higher than face and neck counts. Subjects with heavy shoulder freckling had increased naevus counts on all sites investigated, with a means ratio for whole-body counts of 1.11 (95% CI, 1.03–1.19) compared with those without shoulder freckling. Habitual midday sun exposure rather than holiday sun exposure was also a significant determinant of melanocytic naevi in these adolescents (Darlington et al., 2002). Because of the importance of naevi in early life as a determinant of risk of melanoma development, repeated standardised surveys of naevus density in children have been advocated as a means of monitoring future melanoma risk in the population at large (Pfahlberg et al., 2001).

5.3.2. Melanoma—In the only available case–control study of melanoma in childhood (Whiteman et al., 1997b) neither acute nor chronic exposure to solar UV radiation was associated with increased melanoma, suggesting that, since most Queensland children experience high levels of sun exposure in early life, there was insufficient variability in any of the markers of UV exposure to discriminate the small number of cases from controls. The findings also suggest that genetic factors play a relatively greater role in very early childhood melanoma rather than sun exposure. The case–control study of melanoma in adolescence (Youl et al., 2002) showed a slightly higher proportion of cases than controls reporting more than 10 episodes of peeling sunburns, and there was a statistically significant trend to increasing risk of melanoma with increasing numbers of peeling or blistering sunburns.

6. Childhood as a susceptible life stage for adult UV radiation effects

Besides UV-induced skin damage manifest in children, harmful longer-term effects occur, that are latent until later in adulthood. It is possible that sunlight exposure during childhood and adolescence confers a greater increase in risk of melanoma compared with risk incurred by exposure at older ages. This theory is based on the plausible notions of heightened susceptibility of young melanocytes to initiation of UV carcinogenesis through alteration of melanocyte DNA by sun exposure and blistering sunburns during youth given that peak melanocytic activity and naevogenesis occurs in early life (Balk, 2011). The structure of juvenile skin shows the position of vellus hair follicles (containing stem cells that give rise to melanocytes inter alia) to be closer to the exposed skin surface than stem cells in adult hair follicles, so that the same amount of surface UV exposure may result in increased UV dosage and DNA damage to the cells that give rise to melanocytes than in older skin, contributing to increased childhood susceptibility to melanomagenesis (Gomez Garcia et al., 2011).

6.1. Migrant data

Whiteman et al. (2001) examined evidence from migrant studies and found that as people migrated from countries with low ambient solar radiation to countries with high solar UV at successively older ages, their melanoma risk decreased compared with those who arrived in early childhood (younger than 10–15 years). On the other hand, those who migrated in very early childhood from countries with high to those with low ambient exposure, were at similar risk of melanoma to native-born residents after living at least a year at the low latitude (Whiteman et al., 2001) thus supporting the notion that childhood is a susceptible period for UV carcinogenesis (Gomez Garcia et al., 2011).

6.2. Case–control studies

Reliance on personal reports of childhood sun exposure as in case–control studies of melanoma, does not give consistent and reliable enough data to assess the question of

heightened susceptibility in childhood (Autier and Dore, 1998; Whiteman et al., 2001). In a case–control study in Belgium, Germany and France in the early 1990s it was seen that melanoma risk associated with a given amount of sun exposure during adulthood increased with rising levels of sun exposure during childhood. This increase in risk was higher than a simple addition of melanoma risk associated with sun exposure during childhood and adulthood. On the other hand, high sun exposure during childhood was a significant risk factor for melanoma only if there was substantial sun exposure during adult life (Autier and Dore, 1998). Other researchers, including authors of a meta-analysis, have also noted that multiple sunburns increase melanoma risk irrespective of their timing in life (Dennis et al., 2008; Pfahlberg et al., 2001). Recently, in a molecular analysis of melanoma tumour tissue from US cases in a case–control study, BRAF and NRAS somatic mutations were compared with ambient solar UV exposures throughout life estimated using residential histories and satellite-based UV data. When ambient UV was analysed by decades of age, BRAF-mutant tumours were characteristic of patients with high ambient UV exposure in the first 2 decades of life, and were distinct from tumours with NRAS-mutations, which were linked with high exposure at ages 50 and 60 years (Thomas et al., 2007). These results further support the belief that UV irradiance influences melanoma risk differently based on the age of UV exposure. The authors also concluded that their observations of BRAF mutations associated with early-life UV exposure provided further evidence in support of childhood sun protection for melanoma prevention (Thomas et al., 2007).

6.3. Artificial UV radiation

Artificial UV exposure of young people under 20 years has increased in recent decades especially among young women, through the use of tanning facilities. The effect of artificial UV exposure on melanoma rates has been discussed separately in this issue (Gandini et al., in press).

7. Primary prevention

Worldwide, strategies for primary prevention have been mainly focused on reducing sunburn and sun exposure overall in white-skinned people of all ages in an attempt to diminish their skin cancer risk. These strategies include encouraging the use of hats, appropriate clothing and sunscreen, as well as avoidance of the sun, particularly at peak hours of the day, and avoidance of artificial forms of UV radiation such as sunbeds (Stanton et al., 2004). Children and adolescents have been major target groups in these prevention efforts (Stanton et al., 2004). Furthermore, although lifetime estimates of UV exposure have been variable, most estimates show that it is proportionately greater than expected in children and teens (McBride, 2009).

Among both children and adolescents, sunscreen is the most frequently used method of sun protection (Robinson et al., 2000; Stanton et al., 2004). The protective behaviours of children are highly dependent on the behaviours and advice of their parents, even after they begin to take greater responsibility for themselves (Fisher et al., 1996). Though their knowledge about skin cancer and associated risk factors is high (Robinson et al., 2000), adolescents are generally more reluctant than children to use sun protection. Females also tend to value having a tanned complexion (Stanton et al., 2004) more than males, which contributes to their highly prevalent use of sunbeds (Schneider and Krämer, 2010). Though parental influence on teens' sun protection practices becomes less important as peer influence increases, they still have a positive effect on adolescents' sun behaviours (Fisher et al., 1996).

Australia has been at the forefront of initiating primary prevention initiatives given the extremely high incidence of all types of skin cancers. Recent declines in the incidence of

Europe PMC Funders Author Manuscripts

both melanoma (Whiteman et al., 1995) and keratinocytic (Staples et al., 1998) cancers illustrate the success of public health interventions such as Australia's "Slip! Slop! Slap!" campaign. Declines in melanoma rates have also been noted in Sweden (Karlsson and Fredrikson, 2007), where a recent study among 7-year old children found an increase in active sun protection by their parents between 2002 and 2007 and a decline in the mean total number of naevi per child (Karlsson et al. 2011). With continued encouragement of protective behaviours both in children and adolescents, and in their parents, the hope is that increasing precaution will be taken by all of these groups to avoid harmful exposure to UV radiation at early ages and prevent adverse health outcomes later in life.

References

Autier P, Dore JF, EPIMEL and EORTC Melanoma Cooperative Group. European Organisation for Research and Treatment of Cancer. Influence of sun exposures during childhood and during adulthood on melanoma risk. Int. J. Cancer. 1998; 77:533–537. [PubMed: 9679754]

Autier P, Dore JF, Negrier S, Liénard D, Panizzon R, Lejeune FJ, Guggisberg D, Eggermont AMM. Sunscreen use and duration of sun exposure: a double-blind, randomized trial. J. Nat. Cancer Inst. 1999; 91:1304–1309. [PubMed: 10433619]

Baade P, Green A, Smithers M, Aitken J. Trends in melanoma incidence among children: possible influence of sun protection programs. Expert. Rev. Anticancer Ther. 2011; 11:661–664. [PubMed: 21554037]

Baade PD, Youlden DR, Valery PC, Hassall T, Ward L, Green AC, Aitken JF. Trends in incidence of childhood cancer in Australia, 1983–2006. Br. J. Cancer. 2010; 102:620–626. [PubMed: 20051948]

Balato N, Gaudiello F, Balato A, Monfrecola G. Sun habits in the children of Southern Italy. J. Am. Acad. Dermatol. 2007; 57:883–887. [PubMed: 17939938]

Balk SJ. Ultraviolet radiation: a hazard to children and adolescents. Pediatrics. 2011; 127:e791–e817. [PubMed: 21357345]

Battistutta D, Fourtanier A, Green A, Pandeya N, Strutton G, Tison S. Skin surface topography grading is a valid measure of skin photoaging. Photodermatol. Photoimmunol. Photomed. 2006; 22:39–45. [PubMed: 16436180]

Bauer J, Büttner P, Wiecker TS, Luther H, Garbe C. Effect of sunscreen and clothing on the number of melanocytic Nevi in 1,812 German Children Attending Day Care. Am. J. Epidemiol. 2005; 161:620–627. [PubMed: 15781951]

Berg P, Wennberg AM, Tuominen R, Sander B, Rozell BL, Platz A, Hansson J. Germline CDKN2A mutations are rare in child and adolescent cutaneous melanoma. Melanoma Res. 2004; 14:251–255. [PubMed: 15305154]

Boldeman C, Dal H, Wester U. Swedish pre-school children's UVR exposure – a comparison between two outdoor environments. Photodermatol. Photoimmunol. Photomed. 2004; 20:2–8.

Conti EMS, Cercato MC, Gatta G, Ramazzotti V, Roscioni S. Childhood melanoma in Europe since 1978: a population-based survival study. Eur. J. Cancer. 2001; 37:780–784. [PubMed: 11311654]

Crane LA, Mokrohisky ST, Dellavalle RP, Asdigian NL, Aalborg J, Byers TE, Zeng C, Baron AE, Burch JM, Morelli JG. Melanocytic nevus development in Colorado children born in 1998: a longitudinal study. Arch. Dermatol. 2009; 145:148–156. [PubMed: 19221259]

Darlington S, Siskind V, Green L, Green A. Longitudinal study of melanocytic nevi in adolescents. J. Am. Acad. Dermatol. 2002; 46:715–722. [PubMed: 12004313]

Davis KJ, Cokkinides VE, Weinstock MA, O'Connell MC, Wingo PA. Summer sunburn and sun exposure among US youths ages 11 to 18: national prevalence and associated factors. Pediatrics. 2002; 110:27–35. [PubMed: 12093943]

Dennis LK, Vanbeek MJ, Beane Freeman LE, Smith BJ, Dawson DV, Coughlin JA. Sunburns and risk of cutaneous melanoma: does age matter? A comprehensive meta-analysis. Ann. Epidemiol. 2008; 18:614–627. [PubMed: 18652979]

Dodd AT, Morelli J, Mokrohisky ST, Asdigian N, Byers TE, Crane LA. Melanocytic nevi and sun exposure in a cohort of colorado children: anatomic distribution and site-specific sunburn. Cancer Epidemiol. Biomarkers Prev. 2007; 16:2136–2143. [PubMed: 17932362]

Efron PA, Chen MK, Glavin FL, Kays DW, Beierle EA. Pediatric basal cell carcinoma: case reports and literature review. J. Pediatr. Surg. 2008; 43:2277–2280. [PubMed: 19040953]

English D, Milne E, Simpson J. Ultraviolet radiation at places of residence and the development of melanocytic nevi in children (Australia). Cancer Causes Control. 2006; 17:103–107. [PubMed: 16411059]

Fisher KJ, Lowe JB, Gillespie AM, Balanda KP, Baade PD, Stanton WR. The Relationship between Australian Student's Perceptions of Parental Behaviour, School Policies and Sun Protection Behaviour. Public Health Association of Australia. 1996

Fritschi, L. Assessment of sun exposure. Australian National University; Canberra: 1993. p. 162PhD

Fritschi L, Battistutta D, Strutton GM, Green A. A noninvasive measure of photoageing. Int. J. Epidemiol. 1995; 24:150–154. [PubMed: 7797337]

Fritschi L, McHenry P, Green A, Mackie R, Green L, Siskind V. Naevi in schoolchildren in Scotland and Australia. Br. J. Dermatol. 1994; 130:599–603. [PubMed: 8204467]

Gandini S, Boniol M, Autier P. Meta-analysis on artificial light and skin cancer. Progr. Biophys. Mol. Biol. in press.

Gies P, Roy C, Toomey S, MacLennan R, Watson M. Solar UVR exposures of primary school children at three locations in Queensland. Photochem. Photobiol. 1998; 68:78–83. [PubMed: 9679453]

Godar DE. UV doses worldwide. Photochem. Photobiol. 2005; 81:736–749. [PubMed: 15819599]

Gomez Garcia AM, McLaren CE, Meyskens FL. Melanoma: is hair the root of the problem? Pigment Cell Melanoma Res. 2011; 24:110–118. [PubMed: 20880199]

Green A, Sorahan T, Pope D, Siskind V, Hansen M, Hanson L, Leech P, Ball PM, Grimley RP. Moles in Australian and British schoolchildren. Lancet. 1988; 332:1487–1497.

Green AC. Premature aging of the skin in a Queensland population. Med. J. Aust. 1991; 155:473–478. [PubMed: 1921818]

Green AC, Hughes MCB, McBride P, Fourtanier A. Factors associated with premature skin aging (photoaging) before the age of 55: a population-based study. Dermatol. 2011; 222:74–80.

Hamre MR, Chuba P, Bakhshi S, Thomas R, Severson RK. Cutaneous melanoma in childhood and adolescence. Pediatric Hematol. Oncol. 2002; 19:309–317.

Harrison SL, Speare R, Wronski I, MacLennan R. Sun exposure and melanocytic naevi in young Australian children. Lancet. 1994; 344:1529–1532. [PubMed: 7983952]

Jannot AS, Meziani R, Bertrand G, Gerard B, Descamps V, Archimbaud A, Picard C, Ollivaud L, Basset-Seguin N, Kerob D, Lanternier G, Lebbe C, Saiag P, Crickx B, Clerget-Darpoux F, Grandchamp B, Soufir N, Melan C. Allele variations in the OCA2 gene (pink-eyed-dilution locus) are associated with genetic susceptibility to melanoma. Eur. J. Hum. Genet. 2005; 13:913–920. [PubMed: 15889046]

Karlsson MA, Wahlgren CF, Wiklund K, Rodvall Y. Parental sun-protective regimens and prevalence of common melanocytic naevi among 7-year-old children in Sweden: changes over a 5-year period. Br. J. Dermatol. 2011; 164:830–837. [PubMed: 21219291]

Karlsson P, Boeryd B, Sander B, Westermark P, Rosdahl I. Increasing incidence of cutaneous malignant melanoma in children and adolescents 12–19 years of age in Sweden 1973–92. Acta Dermato Venereol. 1998; 78:289–292.

Karlsson PM, Fredrikson M. Cutaneous malignant melanoma in children and adolescents in Sweden, 1993–2002: the increasing trend is broken. Int. J. Cancer. 2007; 121:323–328. [PubMed: 17372908]

Kelly JW, Rivers JK, MacLennan R, Harrison S, Lewis AE, Tate BJ. Sunlight: a major factor associated with the development of melanocytic nevi in Australian schoolchildren. J. Am. Acad. Dermatol. 1994; 30:40–48. [PubMed: 8277029]

Kennedy C, Bastiaens MT, Bajdik CD, Willemze R, Westendorp RGJ, Bouwes Bavinck JN, Study LSC. Effect of smoking and sun on the aging skin. J. Invest. Dermatol. 2003; 120:548–554. [PubMed: 12648216]

Lange JR, Palis BE, Chang DC, Soong SJ, Balch CM. Melanoma in children and teenagers: an analysis of patients from the national cancer data base. J. Clin. Oncol. 2007; 25:1363–1368. [PubMed: 17416855]

Lucas RM, Ponsonby AL, Dear K, Taylor BV, Dwyer T, McMichael AJ, Valery P, van der Mei I, Williams D, Pender MP, Chapman C, Coulthard A, Kilpatrick T. Associations between silicone skin cast score, cumulative sun exposure, and other factors in the Ausimmune study: a multicenter Australian study. Cancer Epidemiol. Biomark Prevent. 2009; 18:2887–2894.

McBride, P. Cutaneous squamous cell carcinoma and its determinants. University of Queensland; 2009. PhD

Nagore E, Montoro A, Oltra S, Ledesma E, Botella-Estrada R, Millan JM, Oliver V, Fortea JM, Guillan C. Age does not appear to be a major indicator of CDKN2A or CDK4 mutations in melanoma patients in Spain. Melanoma Res. 2005; 15:555–558. [PubMed: 16314743]

Oliveria SA, Satagopan JM, Geller AC, Dusza SW, Weinstock MA, Berwick M, Bishop M, Heneghan MK, Halpern AC. Study of nevi in children (SONIC): baseline findings and predictors of nevus count. Am. J. Epidemiol. 2009; 169:41–53. [PubMed: 19001133]

Orozco-Covarrubias MDLL, Tamayo-Sanchez L, Duran-McKinster C, Ridaura C, Ruiz-Maldonado R. Malignant cutaneous tumors in children: twenty years of experience at a large pediatric hospital. J. Am. Acad. Dermatol. 1994; 30:243–249. [PubMed: 8288784]

Parisi AV, Kimlin MG, Mulheran L, Meldrum LR, Randall C. Field-based measurements of personal erythemal ultraviolet exposure through a common summer garment. Photodermatol. Photoimmunol. Photomed. 2000; 16:134–138. [PubMed: 10885444]

Parisi AV, Meldrum LR, Wong JCF, Aitken J, Fleming RA. Lifetime ultraviolet exposure estimates for selected population groups in south-east Queensland. Phys. Med. Biol. 1999; 44:2947–2953. [PubMed: 10616147]

Pearce MS, Parker L, Cotterill SJ, Gordon PM, Craft AW. Skin cancer in children and young adults: 28 years' experience from the Northern Region Young Person's Malignant Disease Registry, UK. Melanoma Res. 2003; 13:421–426. [PubMed: 12883370]

Pfahlberg A, Kolmel KF, Gefeller O. Timing of excessive ultraviolet radiation and melanoma: epidemiology does not support the existence of a critical period of high susceptibility to solar ultraviolet radiation-induced melanoma. Br. J. Dermatol. 2001; 144:471–475. [PubMed: 11260001]

Robinson JK, Rigel DS, Amonette RA. Summertime sun protection used by adults for their children. J. Am. Acad. Dermatol. 2000; 42:746–753. [PubMed: 10775849]

Schneider S, Krämer H. Who uses sunbeds? A systematic literature review of risk groups in developed countries. J. Eur. Acad. Dermatol. Venereol. 2010; 24:639–648. [PubMed: 20015180]

Soufir N, Lacapere JJ, Bertrand G, Matichard E, Meziani R, Mirebeau D, Descamps V, Gerard B, Archimbaud A, Ollivaud L, Bouscarat F, Baccard M, Lanternier G, Saiag P, Lebbe C, Basset-Seguin N, Crickx B, Cave H, Grandchamp B. Germline mutations of the INK4a-ARF gene in patients with suspected genetic predisposition to melanoma. Br. J. Cancer. 2004; 90:503–509. [PubMed: 14735200]

Spatz A, Giglia-Mari G, Benhamou S, Sarasin A. Association between DNA repair-deficiency and high level of p53 mutations in melanoma of xeroderma pigmentosum. Cancer Res. 2001; 61:2480–2486. [PubMed: 11289118]

Stanton WR, Janda M, Baade PD, Anderson P. Primary prevention of skin cancer: a review of sun protection in Australia and internationally. Health Prom. Int. 2004; 19:369–378.

Staples M, Marks R, Giles G. Trends in the incidence of non-melanocytic skin cancer (NMSC) treated in Australia 1985–1995: are primary prevention programs starting to have an effect? Int. J. Cancer. 1998; 78:144–148. [PubMed: 9754642]

Stern RS, Weinstein MC, Baker SG. Risk reduction for nonmelanoma skin cancer with childhood sunscreen use. Arch. Dermatol. 1986; 122:537–545. [PubMed: 3707169]

Thieden E, Philipsen PA, Sandby-Moller J, Heydenreich J, Wulf HC. Proportion of lifetime UV dose received by children, teenagers and adults based on time-stamped personal dosimetry. J. Invest. Dermatol. 2004; 123:1147–1150. [PubMed: 15610527]

Thomas NE, Edmiston SN, Alexander A, Millikan RC, Groben PA, Hao H, Tolbert D, Berwick M, Busam K, Begg CB, Mattingly D, Ollila DW, Tse CK, Hummer A, Lee-Taylor J, Conway K. Number of nevi and early-life ambient UV exposure are associated with BRAF-mutant melanoma. Cancer Epidemiol. Biomarkers Prev. 2007; 16:991–997. [PubMed: 17507627]

Tsao H, Zhang X, Kwitkiwski K, Finkelstein DM, Sober AJ, Haluska FG. Low prevalence of germline CDKN2A and CDK4 mutations in patients with early-onset melanoma. Arch. Dermatol. 2000; 136:1118–1122. [PubMed: 10987867]

Wallingford SC, Alston RA, Green AC, Birch JM. Comparison of trends in the incidence of cutaneous melanoma in <25 year olds in England with older adults, 1979–2006. Pediatric Blood Oncol. 2010; 55:933.

Whiteman D, Valery P, McWhirter W, Green A. Incidence of cutaneous childhood melanoma in Queensland, Australia. Int. J. Cancer. 1995; 63:765–768. [PubMed: 8847130]

Whiteman DC, Milligan A, Welch J, Green AC, Hayward NK. Germline CDKN2A mutations in childhood melanoma. J. Nat. Cancer Inst. 1997a; 89:1460. [PubMed: 9326917]

Whiteman DC, Valery P, McWhirter W, Green AC. Risk factors for childhood melanoma in Queensland, Australia. Int. J. Cancer. 1997b; 70:26–31. [PubMed: 8985086]

Whiteman DC, Whiteman CA, Green AC. Childhood sun exposure as a risk factor for melanoma: a systematic review of epidemiologic studies. Cancer Causes Control. 2001; 12:69–82. [PubMed: 11227927]

Wright CY, Reeder AI. Youth solar ultraviolet radiation exposure, concurrent activities and sun-protective practices: a review. Photochem. Photobiol. 2005; 81:1331–1342. [PubMed: 16354111]

Youl P, Aitken J, Hayward N, Hogg D, Liu L, Lassam N, Martin N, Green A. Melanoma in adolescents: a case–control study of risk factors in Queensland, Australia. Int. J. Cancer. 2002; 98:92–98. [PubMed: 11857391]

Zhu N, Warr R, Cai R, Rigby HS, Burd DAR. Cutaneous malignant melanoma in the young. Br. J. Plastic Surg. 1997; 50:10–14.

Europe PMC Funders Author Manuscripts