

# 1

## INTRODUCTION

### 1.1 Background

As part of the 1999/2000 Skin Cancer Control Project, the NSW Department of Health commissioned The Cancer Council to develop, in consultation with appropriate partners, a Skin Cancer Prevention Strategic Plan for New South Wales for 2001 to 2005.

The Skin Cancer Prevention Strategic Plan 2001–2005 is the second planning document developed by The Cancer Council in consultation with the Health Promotion Unit, NSW Department of Health, representatives from Area Health Services, Health Promotion Coordinators and key stakeholders in sun protection in New South Wales.

The first, **Health Promotion Strategic Plan for Skin Cancer Control in New South Wales 1995–2000**, outlined priorities for intervention and the respective roles of The Cancer Council, NSW Health Department and other key stakeholders in the primary and secondary prevention of skin cancer. This document has provided a valuable resource for the development and implementation of state and regional skin cancer prevention plans and served as an excellent foundation for skin cancer control strategic planning in NSW.

The new Strategic Plan has been developed in the context of the past achievements and existing programs in skin cancer prevention in NSW. It has also attempted to take into account salient environmental, organisational and community issues relating to skin cancer control over recent years at national, state and regional levels. **Skin Cancer Prevention Strategic Plan 2001–2005** reflects an ongoing commitment to a coordinated and strategic approach to the prevention of skin cancer in NSW.

### 1.2 Purpose

Skin Cancer Prevention Strategic Plan 2001–2005 is designed to support the work of individuals and organisations involved in the planning and implementation of skin cancer prevention programs throughout the state. It aims to ensure a comprehensive skin cancer prevention program in New South Wales in which the activities conducted at state and regional level are complementary. It is intended that the Strategic Plan will:

- establish clear direction, goals and strategic priorities for programs at state and regional level
- provide guidelines for the development of regional action plans
- facilitate coordination of state and regional activities
- assist in establishing and maintaining partnerships supportive of skin cancer prevention
- disseminate best practice in skin cancer control interventions
- optimise use of available resources for skin cancer prevention
- provide a framework for monitoring and evaluation of programs
- maximise the impact of skin cancer initiatives undertaken
- attract commitment to and funding for skin cancer prevention programs in NSW.

## 1.3 About this document

As background to the Strategic Plan, a brief overview of the scientific and epidemiological rationale for skin cancer prevention programs is provided in Section Two. The third section of the document outlines the policy context for the NSW Strategic Plan and explains the basis for the selection of the priority population groups, settings and types of strategies proposed by the plan.

An overview of the Strategic Plan is presented on p.56 of Section Four. It provides a summary of the long term and intermediate outcomes required to reduce the incidence and mortality of skin cancer in NSW. The introduction to this section provides an aid to the interpretation of the overview and to the more detailed Strategic Plan which follows.

The most important practical application of the Skin Cancer Prevention Strategic Plan 2001–2005 will be in supporting the development of other planning documents in skin cancer programs throughout the state. It is the Strategic Plan itself, in Section Four, which has been the focus of most of the consultation processes during the development of the document. Its format and content have been designed to be compatible with those used in regional planning.

The Strategic Plan indicates strategic priorities in terms of target population groups, settings that provide the best opportunities for successful interventions, priority strategies and desirable partnerships to achieve change. Strategies proposed by the plan have been organised according to their contribution to achieving environmental, organisational or broader community outcomes favourable to reducing sun exposure. Recommendations for regional or area based activities are accompanied by an outline of the proposed state level support for these approaches. The final section of the document presents a framework for monitoring and evaluation of skin cancer prevention in New South Wales.

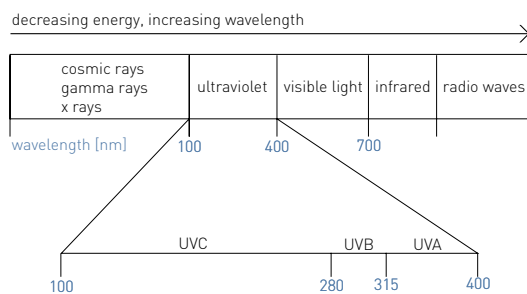
Although the Strategic Plan sets priorities and nominates preferred strategies, partners and outcomes, it is recommended that all users take into account specific local needs and opportunities for skin cancer prevention when formulating regional and local plans. Selecting priorities and strategies consistent with those outlined in this document for regional planning, however, means that local activities are more likely to be complemented and supported by statewide media campaigns, resources and programs.

# 27 RATIONALE

## 2.1 Solar ultraviolet radiation and health

Australia's proximity to the equator means that solar ultraviolet radiation (UVR) levels in NSW are very high.<sup>1</sup> Like radiowaves, visible light, infrared radiation and X-rays, UVR is a form of electromagnetic energy (Figure 1). Unlike visible light and the heat from infrared radiation, UVR cannot be seen or felt.

**Figure 1**  
The electromagnetic spectrum



There are three energy bands of UVR distinguished by their wavelength ranges:

UVA	315–400nm*
UVB	280–315nm
UVC	100–280nm

Longer wavelengths of solar UVR are more likely to penetrate the earth's atmosphere. Therefore, while most UVA transmits freely to the earth's surface, most UVB (85–95%) and virtually all UVC is absorbed by ozone and atmospheric gases.<sup>2</sup>

The biological effects of UVR also vary according to wavelength. Although only 5 to 15% of UVB penetrates the earth's atmosphere, these shorter wavelengths of UVR carry larger amounts of energy and are likely to cause more damage to human tissues than UVA.<sup>1</sup>

Levels of UVB reaching the earth's surface vary far more than UVA levels. This energy band is strongly influenced by changes in cloud cover and factors that influence the amount of atmosphere through which UVR passes before reaching the earth (such as time of day, season, and altitude). Factors influencing levels of UVR are summarised in Table 1.

**Table 1**  
Factors affecting ambient solar UVR levels

Factor	Influence
Geographic location	UVR levels (especially UVB) decrease with increasing distance from the Equator.
Time of day	The single most important factor affecting UVR levels is the height of the sun in the sky. UVR levels are highest around solar noon. UVB varies more during the day than UVA.
Season	UVR levels are higher in summer than in winter because the sun is higher in the sky and the path UVR has to traverse through the atmosphere is shorter. Also, during the southern hemisphere's summer, the earth's orbit brings it closer to the sun than it does during the northern hemisphere's summer. The effect is that the southern hemisphere receives 7% more UVR than the northern hemisphere does during its summer.
Clouds	The amount of cloud in the sky can have a substantial effect on UVR levels, particularly UVB. Heavy cloud can reduce UVB to less than 5% of that present under clear skies. Scattered cloud has a variable effect, with the levels rising and falling as clouds pass in front of the sun.
Surrounding environment	Environments that contain highly reflective surfaces such as snow and sand are usually characterised by high indirect UVR levels.
Altitude	UVR levels increase by approximately 4% with every 300 metres of altitude. <sup>3</sup> Locations at high altitudes can have significantly higher UVB levels than those at sea level.
Stratospheric ozone	Ozone makes up only a small proportion of the earth's atmosphere but is an important factor in the absorption of UVR, especially UVB. Ozone in the stratosphere (upper atmosphere) absorbs most of the UVB entering the atmosphere before it reaches the earth's surface. UVA is not significantly affected by ozone levels.

Sources:  
Gies PH, Roy CR, Toomey S, McLennan A. **Protection against solar UV radiation.** Sun Protection Seminar Papers. Australian Cancer Society, 1997.

IARC Monograph on the Evaluation of Carcinogenic Risks to Humans. **Solar and Ultraviolet Radiation 1992;** 55:95-122.

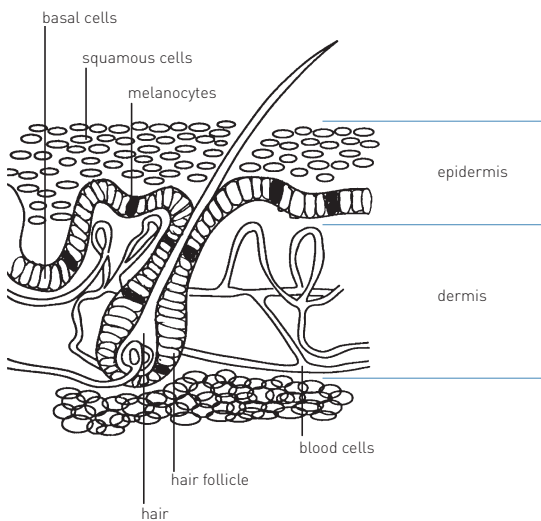
\* One nanometre (nm) = 10<sup>-9</sup> m

## 2.1.1 UVR and the skin

Penetration of UVR into the epidermis (outer layer) and dermis of the skin (see Figure 2) may produce changes in the skin's structure, function and appearance.

Ultraviolet radiation stimulates production of the pigment melanin by specialised cells (melanocytes) in the epidermis. Small packets of melanin (melanosomes) are taken up by skin cells above the basal layer providing a degree of protection from UVR for the cells below. The genetically determined amount and distribution of melanin gives skin its characteristic colour or, as in the case of freckled skin, pattern of pigmentation. Skin type will also determine its response to UVR; how long it takes to redden and how early in life damage and ageing become apparent.

**Figure 2**  
Structure of human skin



Exposure to UVR has both short and long term consequences for human skin. Acute exposure may cause sunburn that becomes evident as redness (erythema) accompanied sometimes by local inflammation or blistering. Over time, exposure to the sun may cause premature ageing of the skin characterised by wrinkles and altered pigmentation. UVR induced loss of skin tone is thought to be the result of damage to the collagen and elastin proteins which support the skin.<sup>4</sup>

The association between sunlight and skin cancer has been documented in medical literature for more than a century. Marks<sup>5</sup> cites a German paper from 1894 in which sunlight was nominated as a factor contributing to non-melanocytic skin cancer (NMSC). Extensive epidemiological and clinical research in Australia and overseas has since confirmed that solar ultraviolet radiation is responsible for the vast majority of skin cancer.<sup>2,6</sup>

The most common types of skin cancer resulting from UVR exposure arise in cells in the basal layers of the skin (basal and squamous cell carcinomas), and, less frequently, from melanocytes (melanoma).

When UVR penetrates the epidermis, it is capable of damaging the genetic components (DNA) of the cells in the skin. Although the UVB band of the solar spectrum is thought to be primarily responsible for UV-induced DNA damage in the skin, both UVA and UVB have been recognised by the International Agency for Research on Cancer as possessing carcinogenic properties.<sup>2</sup>

It is thought that UVR probably plays a dual role in the pathogenesis of skin cancer producing both the original DNA mutation and subsequent tumour promotion.<sup>7</sup> Mutations of the p53 tumour suppressor gene may be induced by UVR rendering the gene ineffective in its usual role of detecting and arresting abnormal cell growth. Studies from around the world have consistently revealed mutations of this gene in non-melanocytic skin cancer cells although alterations to p53 do not seem to be implicated in the causation of melanoma.

There are undoubtedly other genes and other factors involved in the development of skin cancer. Ultra-violet radiation is also thought to be responsible for the generation of short-lived but very destructive free radicals that may do further damage to the cell's DNA. Also, with age, our ability to repair DNA damage induced by sunlight is reduced.<sup>8</sup>

### 2.1.2 UVR and immune function

Recent research has also been examining the effect of UVR on the immune system. Although immunosuppression can be detected in the skin for several hours following exposure to UVR, the role of the immune system in preventing or managing skin cancer has not been clearly established by research.<sup>7,9</sup> In his review of the health effects of solar UVR, Armstrong<sup>10</sup> notes that the effect of UVR on human immunity may have implications for susceptibility to infection, the effectiveness of immunisation and the activation of latent virus infections (such as cold sores).

### 2.1.3 UVR and the eyes

Eye disease caused by UVR may impair vision, and, in some cases, cause blindness. Damage to the tissues of the eye may also occur as a result of acute or prolonged exposure to UVR. Exposure to UVR may cause:

- painful eye inflammation eg. snow blindness
- a growth over the cornea (pterygium)
- cloudiness of the lens (cataract)
- cancer on the surface of the eye and nearby tissues.

### 2.1.4 UVR and Vitamin D

Vitamin D is produced in the skin through the photochemical effect of UVR on cholesterol. The vitamin plays a role in calcium absorption into bones contributing to normal bone growth and maintenance. Although Vitamin D deficiency may occur in people confined indoors and dark skinned people living in northern Europe, it is not usually a problem for those who normally spend some time outdoors, even if they attempt to protect themselves well from the sun.<sup>10</sup>

**On balance, the risks of exposure to UVR far outweigh its limited benefits. Reducing exposure to UVR is one of the key components of any skin cancer prevention program.**

## 2.2 Skin cancer rates and trends

In Australia, skin cancer dominates cancer incidence data. New cases of skin cancer outnumber all other forms of cancer by more than three to one.<sup>11</sup> Currently, there are around 1,300 deaths each year from the disease.

Although Australia still has the highest incidence of skin cancer in the world, recent trends provide cause for optimism. The incidence of basal cell carcinoma and melanoma in younger people (under 55), especially among women, have begun to level off and, in some age groups, begun to decline.<sup>12</sup> This is the first time such a trend has been noted in any country. The pattern of change is consistent with the trends that would be expected to result from the impact of skin cancer prevention programs undertaken in this country over the last two decades.

### 2.2.1 Melanoma

Excluding non-melanocytic skin cancer (NMSC), malignant melanoma is the third most common cancer in Australian females and ranks fourth in males.<sup>13</sup> It affects almost 7,000 Australians each year, causing around 900 deaths.<sup>14</sup> Five year survival rates are around 90% and higher at earlier stages of detection.

Between the 1950s and 1980s, deaths from melanoma increased fourfold in males and doubled in females.<sup>11</sup> Recent trends, however, provide the first evidence of an impact of prevention programs, with a levelling off in incidence and declining mortality in some sections of the population. The strongest declines in incidence have occurred amongst people under 55 years of age - who would be the most likely to have benefited from prevention programs.<sup>15</sup>

### Melanoma in NSW

Data collected by the Cancer Council in 1997<sup>16</sup> indicate there were 2,807 new cases of melanoma of the skin (1,634 males, 1,173 females) in NSW. There were 358 deaths from the disease - 247 men and 111 women.

Melanoma ranked second highest in incidence among all registerable cancers in women and third for men. For mortality, melanoma ranked thirteenth and ninth respectively. Age standardised incidence rates (Australia 1991) were 52.5 and 37.1 per 100,000 population for males and females respectively. Based on these figures, around 1 in 23 males (4.5%) and 1 in 36 females (2.8%) could expect to develop melanoma by the age of 75 years.

NSW melanoma incidence rates for 1988–92 (33.1 males, 25.6 females per 100,000) were at least twice those in other countries except for non-Maoris in New Zealand (25.0 males, 29.8 females).

### Trends

Between 1987 and 1997, age-standardised incidence rates for melanoma rose by 17% among males but there was no significant trend for women. There were also no changes in mortality rates for either sex.

Generally, recent data trends demonstrate a stabilising of incidence and mortality, particularly in younger women.<sup>17</sup> Between 1983 and 1995, melanoma incidence increased by an average of 4.1% each year in males compared with only 0.3% in females. Mortality for men increased by 1.7% a year but decreased by 0.8% in women. Most of the increases in incidence during this period occurred in older males. There were significant falls in incidence rates amongst young adult women while rates in young men were steady.

### Age and gender

Around fifty percent of melanomas are diagnosed in people under 50 years of age – and occasionally in adolescents. In 1997, melanoma was the most common cancer in males aged 20–54 years and in females 15–34. Incidence rates were similar in both sexes up to the age of 50, after which, the increase in incidence was greater in men (See Table 2).

Mortality rates in older men were also much higher than those of women of similar age. Taking into account differences in age distribution, men in NSW were 1.5 times more likely to develop cutaneous melanoma than women, and 2.7 times more likely to die from it.

**Table 2**  
Number of new cases of melanoma in NSW 1997  
by age and gender

Age (yr)	0-34	35-44	45-54	55-64	65-74	75-84	85+
Male	127	175	274	311	442	257	48
Female	154	175	225	191	196	172	60

From: Coates MS, Armstrong BK. *Cancer in New South Wales. Incidence and mortality 1997*. Sydney: NSW Cancer Council, 2000.

### Survival

For men and women diagnosed with cutaneous melanoma between 1980 and 1995, five year survival rates were 89% and 95% respectively.

### Socio-economic status

For both sexes, incidence of melanoma tends to be lower in Local Government Areas (LGAs) of lower socio-economic status.

### Regional variation

Within NSW, there are considerable regional variations in melanoma rates.<sup>18</sup> For both sexes, high incidence rates occur along the coast and these rates are also generally higher in the north of the State. The Hunter, Central and North Coast have the highest melanoma rates. Incidence rates are lower in inner, south-western and western suburbs of Sydney and, for men, lower in most inland areas of the state.

**High LGAs** **Males** Byron, Coffs Harbour, Gosford, Great Lakes, Lake Macquarie, Lismore, Newcastle, Tweed and Warringah  
**Females** Coffs Harbour, Grafton, Kyogle, Lake Macquarie, Lismore, Newcastle and Port Stephens.

**Low LGAs** **Males** Ashfield, Blacktown, Burwood, Canterbury, Fairfield, Holroyd, Liverpool, Marrickville, Parramatta, Shellharbour and Young.  
**Females** Ashfield, Auburn, Bankstown, Blacktown, Canterbury, Fairfield, Holroyd, Liverpool, Marrickville, Parramatta, Penrith and Strathfield.

### Projections of incidence

Estimates of melanoma incidence in future years may be helpful for health program planning and priority setting. These figures are based on mathematical modelling of current trends and may be proved inaccurate if changes occur in exposure

to risk factors and screening methods. Tables 3 and 4 provide projected figures for melanoma incidence and number of new cases in NSW for 2001 and 2006.

**Table 3**  
Projected age-standardised incidence rates\*  
of cancer in NSW for 2001 and 2006

New cases	Sex	Actual rate		Projected rate	
		1996	2001	2006	2006
All cancers	Male	473.3	423.8	442.2	
	Female	326.8	335.4	343.3	
Cutaneous melanoma	Male	47.1	59.4	68.3	
	Female	31.0	37.1	40.4	

\* per 100,000 people, 1991 Australian population used as standard

**Table 4**  
Projected number of new cases of cancer in NSW  
for 2001 and 2006

Cancer site	Sex	Actual no.		Projected no.	
		1996	2001	2006	2006
All cancers	Male	14,447	14,380	16,705	
	Female	11,783	13,230	14,910	
Cutaneous melanoma	Male	1,458	2,075	2,600	
	Female	1,068	1,320	1,580	

Tables 3 and 4 adapted from Coory M, Armstrong B. Cancer incidence projections for Area and Rural Health Services in NSW Sydney: NSW Cancer Council, 1998.

## 2.2.2 Non-melanocytic skin cancer

Basal cell carcinomas (BCC) and squamous cell carcinomas (SCC) are collectively described as non-melanocytic skin cancer (NMSC). Data on NMSC is not routinely collected by Australian cancer registries. A number of studies have estimated the incidence by screening and follow up of malignancies in several localities.<sup>19, 20, 21</sup> This data, however, may not be representative of the general Australian community and the methodology is not suited to national surveillance.

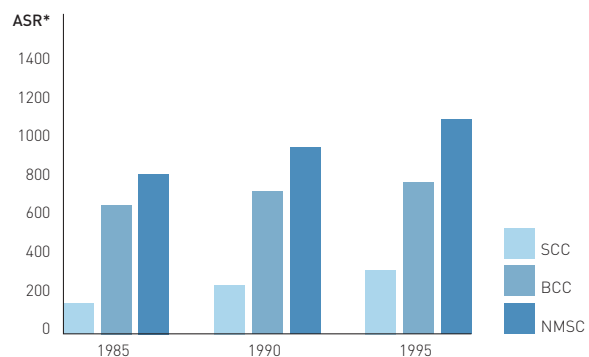
A national random household survey to estimate NMSC incidence was first conducted in 1985.<sup>11</sup> The protocol was replicated in 1990<sup>22</sup> and 1995 allowing trend analysis over the ten year period.<sup>23</sup> Among the methodological limitations of these surveys is that they document only the number of NMSC treated medically. Consequently, they may underestimate the true incidence of NMSC because of the probable high prevalence of untreated skin cancer in the

community. The results, therefore, may be influenced not only by true changes in incidence but also by the proportion of the affected population seeking treatment.

Each year, approximately 345,000 new cancer cases are diagnosed in Australia. A large proportion of these (approximately 270,000) are NMSC.<sup>24</sup> Non-melanocytic skin cancer is the most common type of cancer in Australia for both males and females. NMSC rates are eight times the next most common male cancer (prostate) and seven times the next most common female cancer (breast).

The incidence of NMSC continues to rise at 4–5% per annum; at higher rates in males than females.<sup>22</sup> Over the ten year period covered by the series of national surveys (1985–1995),<sup>23</sup> SCC rates increased by 93%; BCC by 19%. The ratio of BCC to SCC has changed from 4:1 in 1985 to 2.5:1 in 1995 (Figure 3).

**Figure 3**  
Australian NMSC rates  
1985–1995



\*Age standardised rate: per 100,000 person years, using world population

Although total NMSC incidence continues to rise, there have been reductions in BCC incidence observed in younger age groups (Figure 4). SCC rates increased in all age groups over all three surveys but were relatively greater at older ages.

**Figure 4**  
Change in the national age specific BCC rates 1985–1995

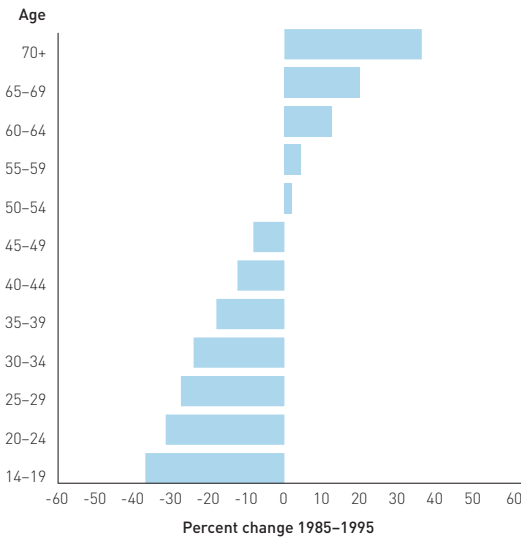


Figure adapted from Staples M, Marks R, Giles G. Trends in the incidence of NMSC treated in Australia 1985-1995; are primary prevention programs starting to have an effect? *Int J Cancer* 1998; 78:144-148.

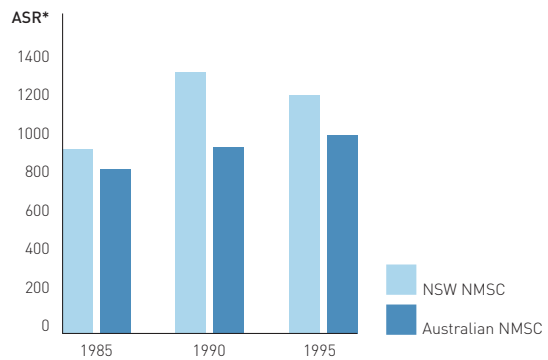
Despite the fact that NMSC can usually be cured if treated early (1% case-fatality rate),<sup>25</sup> mortality has been increasing since the late 1980s. Currently, there are around 400 deaths in Australia each year from the disease.<sup>26</sup>

Predicted long-term decreases in stratospheric ozone are also expected to increase UVB radiation in the earth's southern mid-latitudes by about 15% early in the twenty-first Century. It has been estimated that for every 1% sustained decrease in ozone, there will be a 1.7% long-term increase in basal cell carcinoma and a 3.0% increase in squamous cell carcinoma of the skin. The increase in melanoma of the skin is less certain, but would probably be at least 0.4%.<sup>27</sup>

### Non-melanocytic skin cancer in NSW

Data derived from the national surveys on NMSC indicate that NSW has higher NMSC rates than the general Australian population.<sup>28, 29</sup> NSW, however, has not followed the national trend of continual increase in NMSC rates over the 10 year period of the surveys; 1995 figures show a downturn in NMSC figures (Figure 5).

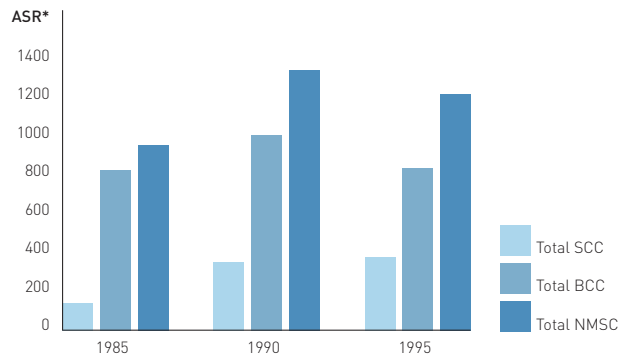
**Figure 5**  
NSW and Australian NMSC rates 1985–1995



\*Age standardised rate: per 100,000 person years, using world population

Although national data demonstrate declining BCC rates in younger age groups, the overall BCC rate continues to rise. In NSW, however, the overall BCC rate dropped between 1990 and 1995 – almost returning to 1985 figures (Figure 6). There is insufficient data available to accurately determine in which age groups the declining BCC rates have occurred.

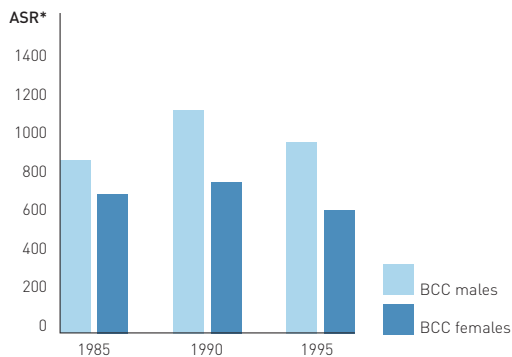
**Figure 6**  
NSW NMSC rates 1985–1995



\*Age standardised rate: per 100,000 person years, using world population

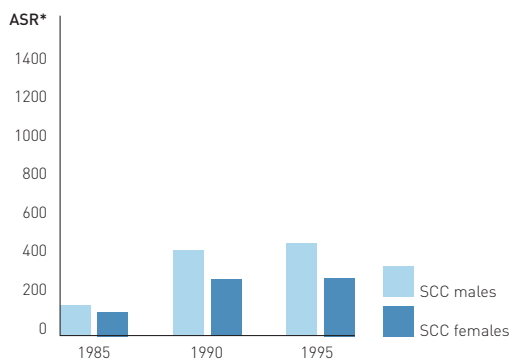
NSW males have consistently higher rates of both BCC and SCC than females. The decrease in BCC rates in NSW observed in 1995 occurs in both sexes (Figure 7). In both men and women, there were smaller increases in SCC rates between 1990 and 1995 than occurred between the first two national surveys (Figure 8).

**Figure 7**  
NSW BCC rates  
1985-1995



\*Age standardised rate: per 100,000 person years, using world population

**Figure 8**  
NSW SCC rates  
1985-1995



\*Age standardised rate: per 100,000 person years, using world population

## 2.3 Cost to the community

The Australian health system spends more money on the diagnosis and treatment of skin cancer than on any other cancer.

In 1993-94, the estimated total direct health system costs of all cancers in Australia was \$1,905 million.<sup>30</sup> Treatment of skin cancer was estimated to cost the Australian community around \$300 million each year – substantially more than any other cancer. Non-melanocytic skin cancer made the largest contribution with an estimated total direct health system cost\*\* of \$232.1 million; melanoma cost \$65.5 million.

This data does not include NMSC that is treated but not diagnosed histologically. Nor do these figures take into account the costs of much larger numbers of people being treated for solar keratoses; the prevalence of which is estimated to be at least 10 times that of NMSC.<sup>11</sup>

The extensive burden placed on the health system and community by skin cancer has led to evaluation of the costs and benefits of preventive programs. There appear to be substantial economic benefits for our health system in investing in skin cancer prevention programs; particularly those which combine media campaigns supported by regional strategies.<sup>15</sup> Reviews of the cost-effectiveness of skin cancer prevention programs indicate that a comprehensive national primary prevention program would be excellent value.<sup>31</sup>

Because of its high prevalence and cost to the community, well-established aetiology and the availability of effective preventive strategies, control of skin cancer has been nominated as one of the Australia's national health priority areas.<sup>13, 26, 28, 31, 32</sup>

\*\* Direct costs include costs of hospital inpatient and outpatient services, nursing homes, medical services, pharmaceuticals, allied health services, research, other institutional and administration but do not include ambulance services, community health services, medical aids or appliances. Indirect costs include those associated with lost productivity due to sickness and premature death and intangibles such as pain, suffering, anxiety and bereavement.

## 2.4 Risk factors

A preliminary step in reducing the incidence of any disease is to identify factors that influence the likelihood of the condition occurring. In the case of skin cancer, an individual's risk appears to be determined by a combination of inherited characteristics and behaviour or lifestyle relating to exposure to UVR.

### 2.4.1 Skin type and colouring

Skin with little melanin is more vulnerable to both acute and chronic damage by UVR. People with light complexions, fair or red hair, green or blue eyes are more likely to get skin cancer when exposed to UVR.<sup>33,34</sup> Typically, poor tolerance to the sun is indicated by skin that tends to burn and become freckled rather than tan in response to exposure to UVR.

Although people with skin that never tans have five times the risk of NMSC compared to people whose skin always tans, skin cancer rates among the group whose skin always tans (at 407/100,000 person years)<sup>22</sup> are still high enough to warrant regular sun protection measures.

People with black skin are well protected by their very high skin melanin levels. In the United States, skin cancer rates among black Americans are 20 times lower than those in whites and very low in people of Asian origin.<sup>35</sup> In areas with high UVR levels, it is sensible for everyone, regardless of skin type, to take protective measures to minimise the risk of skin cancer.

### 2.4.2 Naevi

Genetic factors, in combination with exposure to UVR and, in particular, exposure during childhood, are thought to determine an individual's number of melanocytic naevi (moles).<sup>36</sup> Epidemiological research has indicated that the number of naevi is the most important known phenotypic risk factor for developing melanoma.<sup>37,38</sup> A study in a Caucasian population in NSW aged 15–84 years found the risk of melanoma to be 12 times higher in those with more than 100 naevi than in people with less than ten.<sup>39</sup>

Indeed, it is possible that the role of childhood sun exposure in causing melanoma may be through the development of naevi.<sup>40</sup> It has been suggested that naevi may evolve from melanocytes with mutations induced by UVR.<sup>34</sup>

### 2.4.3 Solar keratoses

These lesions have been described as a major risk factor for skin cancer (especially NMSC). The link is through their similar association with sun exposure and risk, albeit low, of malignant transformation.<sup>41</sup>

### 2.4.4 Family history

Although the literature in this area does not consistently demonstrate a relationship between risk of melanoma and family history of the disease, much of the research involved has relied on anecdotal reporting for family members. Research combining the analysis of eight case-control studies<sup>42</sup> has found that an individual's risk of melanoma is approximately twice that of the general population if they have an affected first degree relative. This association is independent of age, naevus count, hair or eye colouring and freckling.

People with a family history of skin cancer, especially melanoma, appear to be at higher risk and should be encouraged to take effective personal protection measures and be alert for skin changes associated with cancer.

### 2.4.5 Age

The incidence of skin cancer (both melanoma and NMSC) increases exponentially with age. Over time, accumulated exposure to UVR (for the initiation and promotion of tumors) and declining ability to repair UV-damaged DNA are thought to contribute to an increased risk of skin cancer.<sup>7</sup>

## 2.4.6 Exposure to UVR

The association between sun exposure and skin cancer is supported by numerous animal and epidemiological studies<sup>25,43,44,45</sup> demonstrating that:

- tumours usually occur on skin most exposed to sunlight
- skin cancer increases in frequency with increasing length of exposure and age
- skin cancer is more common amongst light-skinned individuals with fair or red hair and who have a tendency to burn
- skin cancer is more common in people living at lower latitudes with higher ambient UVR levels (among people with the same skin type)
- trends in skin cancer incidence have reflected changes in social attitudes towards tanning and patterns of sun exposure
- people who migrate from countries with low UVR to Australia as adults have lower risk of skin cancer.

It has long been accepted that skin cancer rates, particularly NMSC, are related directly to total cumulative exposure to UVR, however, the relationship between sun exposure and skin cancer may not be so simple. Increasingly, research indicates that amount of exposure, UVR dose per exposure, timing of exposure and skin type are all important factors in the pathogenesis of skin cancer.

The intermittent exposure hypothesis postulates that the occurrence of some types of skin cancer is determined as much by the pattern of sun exposure as by total accumulated dose. Intermittent exposure to UVR (ie infrequent, high dose exposure of untanned skin sufficient to cause sunburn), especially in childhood, may be an important factor in the causation of BCC and melanoma.

Although squamous cell carcinoma occurs most frequently amongst people with outdoor occupations and usually occurs on the sun-exposed areas of the head and neck,<sup>44</sup> both melanoma and BCC may occur on parts of the body with infrequent exposure to UVR. Kricker and associates<sup>46</sup> have replicated the findings of other researchers which fail to find an association between BCC risk and total lifetime exposure to UVR. Their results indicate that, in the dose-response

relationship for UVR and BCC, a point is reached, beyond which, risk of BCC does not increase with further sun exposure. There was some evidence that risk of BCC was linked to higher levels of non-working days exposure.

For melanoma, recreational and intermittent exposure and history of sunburn seem to be stronger determinants of risk than either total or occupational exposure.<sup>2,47,48</sup> These findings have implications for public health measures to reduce skin cancer if there is a possibility that some members of the community shift from regular to intermittent patterns of exposure to UVR.

Although the association between sun exposure and skin cancer is well established, further research is needed to clarify the precise relationships between genetic factors, skin type and patterns of exposure to UVR contributing to skin cancer. Discerning the relationship between exposure and skin cancer is frequently complicated by the difficulty in reliably documenting exposure.

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