Joint Canadian Tanning Association

JCTA Report on the Science of UV light

Sunlight, Sunbeds, Tanning Facilities and Phototherapy

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Executive Summary

Fulsome analysis of peer-reviewed studies suggests that commercial tanning facilities present negligible to no risk to tanning bed users, regardless of age, when overseen by trained professionals. Unfortunately, the political debate around tanning facilities has been tainted by the deliberate and misrepresentative extrapolation of data or data not being put forward deliberately by opponents of the industry who are intent on its demise, for reasons known only to themselves.

If anti-tanning lobbying groups against indoor tanning are so against UV exposure, what is not understood by this association and its members, is why the lobbying groups have not gone after phototherapy units in doctor's offices and sold freely to the general public, no medical prescription required. This type of equipment carries a higher increase risk than indoor tanning equipment in a professional tanning facility. Phototherapy equipment is also listed in the same Group 1 category as sunlight and UV emitting tanning devices by the World Health Organization.

Studies indicating a 75% increase in the incidence of melanoma as a result of the use of tanning beds before the age of 35 rely on data derived from the use of tanning equipment outside of tanning facilities, including equipment used in the home and equipment used for medical purposes. In fact, when data derived from home and medical tanning units is removed from these studies, the increased incidence of skin cancer is reduced to 6% when you include a Skin Type 1 person (always burn, never tan). What is never brought up in the discussion is the lifetime increase risk goes down to 15% still including all type of equipment and a skin type 1 person.

This 6% figure is reduced to nearly 0% when those having Type 1 skin types -- mostly fair skinned individuals who are susceptible to burning and who should never tan -- are removed from the data. Trained operators of commercial indoor tanning facilities are able to determine the skin type of prospective clients and determine who may be at risk, thus keeping these individuals from potential harm.

In addition, new genetic research has found that mutations leading to cancer found in 66% of melanoma are not related to UV exposure.

The WHO 2012 IARC Monograph 100D reports there is no associated risk of basal cell carcinoma (BCC) with the use of indoor tanning facilities. BCC make up approximately 80% all skin cancers in Canada. A positive association has been observed between the use of UV-emitting tanning devices and squamous cell carcinoma (SCC) of the skin, but the WHO does not call it a carcinogen risk. The 2013 Ontario Cancer report states that there is a limited connection between UV-emitting indoor tanning devices and SCC. Most research as it relates to SCC and UV exposure points to chronic and/or sunburning levels.

Opponents of UV light forget to inform the public and government that UV light is an environmental risk and the greatest risk with UV exposure is a person's; genetic makeup, skin type, number of moles and whether they have the ability to create a protective tan. It is this strong association that informs the JCTA's position that we are misdirecting our energy by fixating on age – as opposed to pre-existing genetic factors when designing cautionary messages to the public.

Using existing scientific reports and data, this report outlines and addresses many of the common misrepresentations that are leveled at the commercial indoor tanning industry by its opponents. Many of these same reports were utilized by the International Agency for Research on Cancer (IARC) of the World Health Organization (WHO) in making its determination in 2009 that UV emitting tanning devices (including home and medical units) be reclassified as a Group I carcinogen.

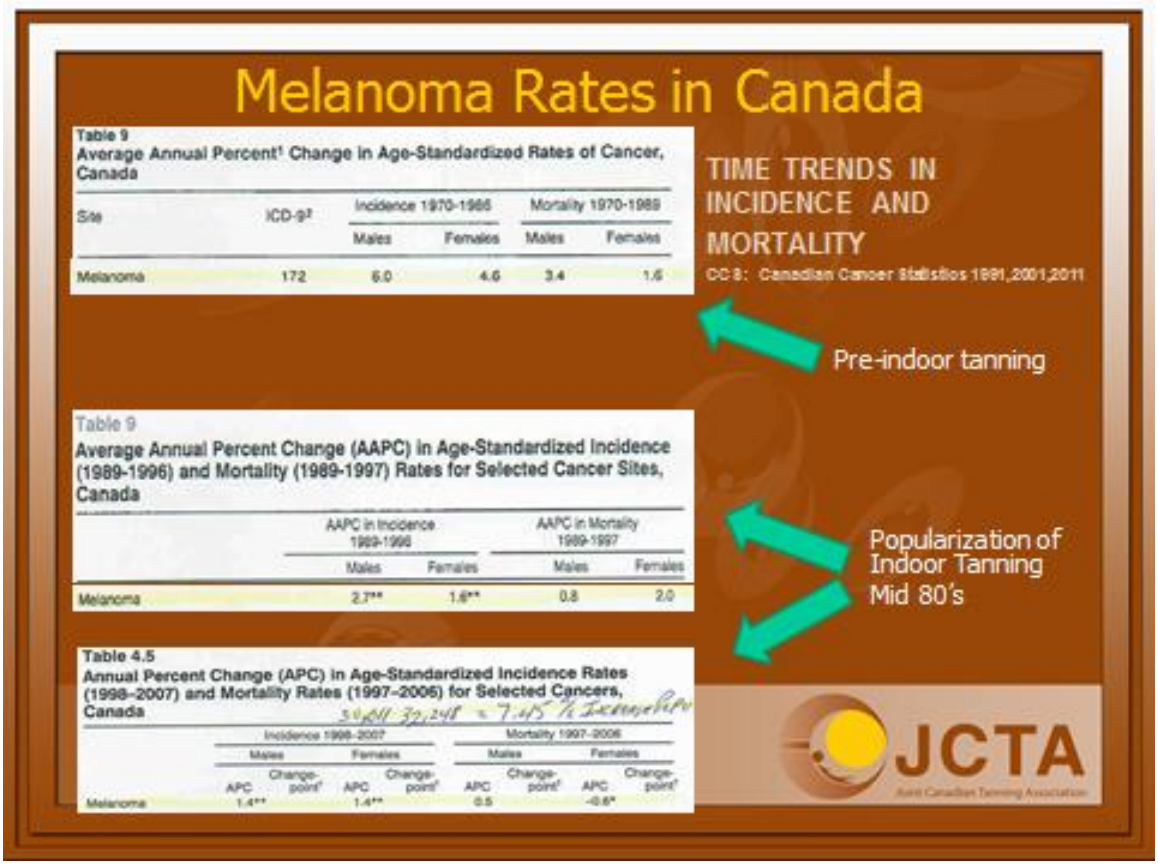
The Joint Canadian Tanning Association has proposed protective measures that mitigates the risk by having a trained and industry certified operator controlling the equipment, skin typing every client correctly using the Dr Fitzpatrick skin typing and ban on skin type 1 (always burn, never tan) people from UV indoor tanning, restricting teens, a ban on the use of self-serve (coin-op, swipe card & client controlled) equipment and the recording of complete client records. The research and information below will show this will enhance consumer protection and ensuring that the commercial indoor industry remains safe for all users.

The fundamental question is whether the facts will guide the debate about UV exposure and consider both the benefits and risks when regulating indoor UV tanning industry or if the political correctness, and whether the punitive agenda of a select few will be allowed to supersede rational discussion and discourse.

Overview

In 2009, the International Agency for Research on Cancer (IARC) of the World Health Organization (WHO) published in the Lancet medical journal that tanning beds were to be reclassified as a Group I carcinogen, which is the same group in which sunlight was placed in 1992 and also phototherapy units. Since then, lobbying by cancer organizations has succeeded in gaining wide support for government legislation banning people under the age of 18 from using tanning equipment in commercial tanning salons. The research specifically used by these lobbying groups is the research from IARC, which stated that using tanning equipment meant that people under the age 35 had a 75% increased risk of melanoma. Of crucial significance is that this increased risk is for all type of tanning equipment (home, medical and commercial) and not just for commercial tanning equipment. This report will show that lobbying groups have misrepresented the risk for commercial tanning equipment. Furthermore, we attempt to outline this misrepresentation in detail and provide a science-based response to common myths used by medical organizations in the media to sway government and public opinion.

The incidence of melanoma in Canada has slowed since the indoor tanning industry started in the early 1980s. The average annual percentage change in age-standardized rates of melanoma cancer have dropped from 6.0% for males and 4.6% for females for the period 1970-1986 to 2.7% for males and 1.6% for females for the period of 1989-1997 and most recently to 1.4% for males and 1.4% for females for the period 1998-2007.



Indoor tanning use in the U.S. population declined from 15% in 2008 to 6% in 2010.
Centers for Disease Prevention and Control. Use of indoor tanning devices by adults-- United States, 2010. MMWR 2012;61:323-6

Opponents of the commercial indoor tanning industry have been singular successful in efforts to portray the industry as unsafe. In the United States alone, the percentage of people using commercial indoor tanning facilities has declined from 15% to 6% in just two years, despite the fact that the indoor tanning industry is largely inconsequential in the big picture of skin cancer prevention. Yet the battle continues, largely because certain medical organizations misrepresent UV exposure and especially indoor tanning as the reason melanoma incidence is increasing.

By focusing on the small percentage of people who tan in a controlled indoor environment, medical organizations have failed to slow the melanoma epidemic, resulting in continued loss of life. In fact, the less UV exposure we seem to have, the more melanoma rates increase. People are working indoors and children are playing computer games, and spending less time outside. When they are outside, they are told to apply chemical sunscreens.

In addition, health organizations have failed to evaluate the real problem of melanoma, namely an increase in risk to older men. Older men are not large users of indoor tanning equipment.

Teen Bans, do they reduce the risk

There are also serious questions as to whether banning those under the age of 18 from indoor tanning facilities will actually reduce the incidence of skin cancer. France introduced an 18-year limitation for indoor tanning in May 1997, more than 15 years ago and was the first major jurisdiction to do so. A review of the publication "La situation du cancer en France en 2011" shows that melanoma for both men and women has increased steadily from 1980 with absolutely no change whatsoever from 1997 onward [page 78, figure 32]. In fact, the rates have increased markedly since 1997 by 35% for females and 62% for men. Evidently it seems like neither a tanning bed restriction nor an almost exponential growth in the sales of sun-protection cosmetics and sunscreens have had any effect on the melanoma incidences.

www.e-**cancer**.fr/.../7708-**la-situation-du-cancer-en-france-en-2011**

As to whether sunbed use actually increases the melanoma risk of younger users, a recent study in the United States reports that it actually lowers the melanoma risk. A large case-control study completed in 2011 in the US looked at sunbeds and sunlamps and their risk of causing melanoma. They found for females, "*use before age 20 yr, current use and years of use were not significant* after adjustments. *The estimated relative odds of melanoma was 0.8 for occasional users (<10 sessions) and 1.1 for more frequent users (10+ sessions)*." For males, the melanoma risk from sunbeds was 0.90 with no significant difference between occasional and frequent users.

Fears et al,. Sunbeds and sunlamps: who used them and their risk for melanoma. Pigment Cell Melanoma RES. doi: 10,1111/j.1755-148X.2011.00842.x

WHO IARC Studies – Review of Melanoma Risk by Sunbed Location

A common assertion is that the commercial tanning industry poses a substantial risk to sunbed users for all ages. In the same 2006/2012 WHO IARC report cited above, the overall increased risk of melanoma from using a sunbed for all ages of people, based on a review of 19 studies, was just 15%. This included home & medical units and incorporated Skin Type 1 individuals. This lower number is usually not stated in the media, and the public is left to infer incorrectly that their increased risk is still 75%, which would be inaccurate.

In addition, quoting a risk of 75% for people under 35 is misleading when referring to the melanoma risk for commercial tanning salons. Lobbying groups do not differentiate the risk for the actual location where the tanning equipment is located. At a commercial tanning facility, trained operators control the exposure time per session, the time between sessions, and the correct exposure time for the person based on their skin type, current tan, medical considerations and previous exposure schedule. People tanning at home can use the tanning equipment for as long as they want, whenever they want regardless of their skin type. This can lead to serious over exposure and risk of burning which results in higher melanoma risk.

Walter et al,. The association of cutaneous malignant melanoma with the use of sunbeds and sunlamps. American Journal of Epidemiology Vol. 131. No 2 (1990)
Chen et al., Sunlamp use and the risk of cutaneous malignant melanoma: a population-based case-control study in Connecticut, USA. International Journal of Epidemiology 1998;27:758-765

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The 75% increase in risk for people under age 35 reference is derived from the WHO IARC report from 2006, which reported that people under the age of 35 who tanned indoors had a 75% greater risk of developing melanoma. What was not reported was from where subjects in this study were getting their sunbed exposure – namely from home and/or medical units which made up more than 50% of the data, and not simply from commercial tanning facilities. When you extrapolate the data from each of the studies used by IARC and recalculate the risk by location of where the sunbed was actually used, you find that the risk was far higher for indoor tanning in both homes and medical facilities.

The cited increased risk for people under 35 from professional, commercial sunbathing equipment is only 6% (Papas) and this included exposure to a Skin Type 1 persons (always burn, never tan). By contrast, the corresponding risk of using sunbed equipment at home is 40% and the risk from medical units is 96%. In other words, medical units have 16 times the risk of professional tanning facilities. Further research into the IARC Report published by Dr. W. Grant found "*Removing skin type 1, those who are genetically predisposed to cutaneous malignant melanoma (CMM), showed no statistically significant link between ever use of indoor tanning facilities and melanoma exists"* (Grant 2009)

The JCTA did a review of the Papas research to find out what is the risk when you include home and commercial units, the increase risk moved from 1.06 to 1.19 when you include a Skin Type 1 person. Again the biggest increase risk was from phototherapy unit at 1.96

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Papas – Differential risk of malignant melanoma by sunbed exposure type (2011) Abstract and poster presented at the 3rd North American Congress of Epidemiology in Montreal June 21-24, 2011

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WHO IARC Working Group on artificial ultraviolet (UV) light and skin cancer. The association of use of sunbeds with cutaneous malignant melanoma and other skin cancers: A systematic review. Int. J. Cancer: 120, 1116-1122 (2006)

The 2006 WHO IARC study combined 7 studies in a meta-analysis for people under age 35 which reported a 75% increased risk for melanoma. The studies were: - Swerdlow 1988, Walter 1990, Westerdahl 1994, Chen 1998, Westerdahl 2000, Veierod 2003, and Bataille 2005. All of these studies had been previously published in peer reviewed publications. Not all 7 studies split out the location where the sun bed was used – home, commercial or medical and the associated melanoma related risks. For those that did and reported on their results, the findings were that sunbed use in commercial tanning salons did not cause a higher risk of melanoma. Here is what they actually reported in each of the individual studies:

Dr. Stephen Walter (McMaster University, Ontario, Canada) looked at the location where a sunbed was used and its impact on risk: *"Analysis of sunbed / sunlamp use according to location showed that home use is associated with a significant odds ratio for each sex, suggesting about a doubling of risk (table 7). Commercial sunbed/sunlamp use was more common in females, but their odds ratio is very close to 1. The commercial sunbed/sunlamp odds ratio* [OR] *for males is elevated, but does not attain statistical significance either. Although the numbers exposed in medical facilities were small, there was a significant risk elevation for females."*

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Table 7
Females - **Commercial Salon – OR 0.92** Home – OR 1.90 Medical Facility – OR 6.42

TABLE 7

Age-adjusted case-control odds ratios, by location of sunbed/sunlamp exposure

Location of exposure	Percent exposed at location		Odds ratio	95% confidence interval	*p* value
	Cases	Controls			
		Males			
Home	12.0	6.0	2.07	1.09–4.08	0.03
Commercial salon	9.8	6.0	1.75	0.89–3.51	0.11
Medical facility	2.5	2.8	0.88	0.27–2.83	1.00
		Females			
Home	14.4	8.0	1.90	1.11–3.31	0.02
Commercial salon	12.1	13.0	0.92	0.55–1.54	0.84
Medical facility	3.9	0.6	6.42	1.41–59.5	0.01

Walter et al,. The association of cutaneous malignant melanoma with the use of sunbeds and sunlamps. American Journal of Epidemiology Vol. 131. No 2 (1990)

The study by Chen from 1998 also reported a higher risk for home bed users. *"Subgroup analyses showed that sunlamp use was associated with a greater increase in risk for melanoma among those who used sunlamps at home."* The majority of sunbed users in the Chen study - 62% in fact, used sunlamps at home. *"A significant association was observed between sunlamp use at home and melanoma risk. For home users, ever use of sunlamps increased risk of melanoma with a crude OR of 1.63".* For sunlamps used in commercial settings such as tanning facilities, Chen reported – *"Sunlamp use in commercial settings was not associated with subsequent development of melanoma"*. Chen also analyzed the data by age group. They found that people under age 25 that used sunlamp's at home had double the risk of commercial tanning salons (see Table 3 below).

Chen reported – *"Sunlamp use in commercial settings was not associated with subsequent development of melanoma".*

Table 3 - Age at first use of sunlamp

<25 years – Commercial OR 0.63
<25 years – Home OR 1.79

Table 3 Case-control odds ratio (OR) and their 95% confidence intervals (CI) for sunlamp-related variables among 579 malignant melanoma cases and 468 population-based controls by location of sunlamp use, Connecticut, 1987–1989[a]

	Home				Commercial Settings			
Sunlamp variables	Cases	Controls	ORc (95% CI)[b]	ORa (95% CI)[c]	Cases	Controls	ORc (95% CI)[b]	ORa (95% CI)[c]
Ever used sunlamp								
No	483	417	–	–	483	417	–	–
Yes	96	51	1.63 (1.12–2.37)	1.40 (0.97–2.04)	44	44	0.88 (0.56–1.37)	0.79 (0.49–1.26)
Total number of sunlamp uses								
Never	483	417	–	–	483	417	–	–
<10	57	26	1.90 (1 16–3.09)	1.84 (1.11–3.06)	19	24	0.68 (0.37–1.28)	0.61 (0.47–0.81)
≥10	38	21	1.58 (0.91–2.74)	1.25 (0.71–2.20)	24	19	1.14 (0.61–2.13)	1.00 (0.52–1.91)
Linear trend[d]			$P = 0.68$	$P = 0.45$			$P = 0.78$	$P = 0.83$
Age at first use of sunlamp[e]								
Never	483	417	–	–	483	417	–	–
<25	57	26	2.03 (1.25–3.32)	1.79 (1.07–2.97)	14	16	0.77 (0.37–1.62)	0.63 (0.29–1 36)
25–45	21	15	1.19 (0.60–2.35)	0.92 (0.46–1.87)	18	16	1.01 (0.51–2.01)	1.07 (0.53–2.17)
>45	12	5	1.93 (0.67–5.58)	2.12 (0.69–6.47)	11	10	1.07 (0.45–2.54)	0.71 (0.29–1.75)

[a] Number of cases and controls do not always add up to total due to missing information.
[b] Crude odds ratio adjusted for sex and age.
[c] Odds ratio adjusted for sex, age, skin susceptibility index, and total recreational sun exposure index.
[d] Crude linear trend adjusted for sex, age, and ever used sunlamp (yes/no). Multivariate-adjusted linear trend adjusted for sex, age, cutaneous phenotype index, recreational sun exposure index, and ever used sunlamp (yes/no).
[e] Odds ratio adjusted for total number of sunlamp uses, in addition to variables mentioned above.

Chen et al., Sunlamp use and the risk of cutaneous malignant melanoma: a population-based case-control study in Connecticut, USA. International Journal of Epidemiology 1998;27:758-765

There are other facts to consider when looking at the combined melanoma risk for people under age 35 from these 7 studies:

- Swerdlow 1988
- older study, done in Scotland, greater percentage of Skin Type 1s with a greater risk of melanoma, with cases from 1979-84 when sunbed industry was still in its infancy
- report stated *"Risk analyzed by age at first exposure was somewhat greater for people first exposed before age of 30 compared with those at a later age, but overall relation to age was not significant"*

Swerdlow et al,. Fluorescent lights, ultraviolet lamps, and risk of cutaneous melanoma. BMJ Volume 297 10 September 1988

- Westerdahl 2000
- southern Sweden, greater percentage of skin type 1s vs Canada, plus exposure time was 30 minutes which was higher than in the use of Canadian equipment today
- Westerdahl did two studies from the same group of cases and controls, one on sunbed risk and one for sunscreen risk
- Westerdahl's Sunbed study found OR of 1.2 for "ever" use and 1.8 OR for regular sunbed use. In Westerdahl's Sunscreen study, with the same patients he found 1.3 OR for ever use and 1.8 OR for always use sunscreens. So sunbeds and sunscreens had the same Odds Ratio – 1.8 for regular users. The use of sunbeds (home or commercial) and chemical sunscreens have the same increase risk of melanoma.

Westerdahl et al,. Risk of cutaneous malignant melanoma in relation to use of sunbeds: further evidence for UV-A carcinogenicity. British Journal of Cancer (2000) 82(9), 1593-1599

Westerdahl et al,. Sunscreen use and malignant melanoma. Int. J. Cancer: 87, 145-150 (2000)

- Veierod 2003
- this was a prospective cohort study of 106,379 women from Norway and Sweden
- the IARC study used data from this study for women age 20-29 who tanned >1 time/month – OR 2.58. Not all the data was used to create the WHO IARC Conclusion, the increase risk would have been lower if all the data was used.
- IARC could have selected the data for age 10-39 RR 1.55 as this more broadly matched the criteria for under age 35 they were working with. IARC committed selection bias and cherry picked the worst case of RR 2.58 vs RR 1.55 to show an overall high risk for the meta-analysis
- in 2010 this study was updated for the 106,379 women and actual melanoma cases. When they recalculated the updated numbers the results for women age 20-29 who tanned >1 time/month had an RR of 1.53. That's a 40% drop in risk from the same study as what was used in the 2006 IARC report
- in addition, the 2010 updated study shows an RR of just 1.19 for people age 10 – 19 years and an RR of 1.38 for women aged 10 – 39 years

Table 5. Relative risks (RRs) and 95% confidence intervals (CIs) of cutaneous malignant melanoma according to solarium use during different age periods*

Age period and solarium use 2003	Frequencies, No. (%)	No. of cases	Age-adjusted RR (95% CI)	Multivariable RR† (95% CI)
10–19 years (n = 85 847)				
Never	84 182 (98)	152	1.00 (referent)	1.00 (referent)
Rarely or ⩾1 time/month	1665 (2)	4	1.65 (0.61 to 4.47)	1.52 (0.56 to 4.12)
			P = .36	P = .44
20–29 years (n = 89 142)				
Never	71 133 (80)	123	1.00 (referent)	1.00 (referent)
Rarely	11 618 (13)	19	1.16 (0.70 to 1.92)	1.11 (0.67 to 1.85)
⩾1 time/month	6391 (7)	18	2.32 (1.35 to 3.99)	2.58 (1.48 to 4.50)
			P_{trend} = .009	P_{trend} = .006
30–39 years (n = 87 890)				
Never	44 338 (50)	78	1.00 (referent)	1.00 (referent)
Rarely	28 383 (32)	51	1.03 (0.72 to 1.48)	0.93 (0.64 to 1.34)
⩾1 time/month	15 169 (17)	36	1.40 (0.93 to 2.10)	1.42 (0.93 to 2.16)
			P_{trend} = .15	P_{trend} = .19
40–49 years‡ (n = 41 409)				
Never	17 345 (42)	27	1.00 (referent)	1.00 (referent)
Rarely	14 514 (35)	33	1.46 (0.88 to 2.43)	1.39 (0.82 to 2.33)
⩾1 time/month	9550 (23)	22	1.48 (0.84 to 2.60)	1.67 (0.93 to 2.99)
			P_{trend} = .14	P_{trend} = .08
Combined, 10–39 years (n = 79 616)				
Never/rarely, 10–39 years	65 239 (82)	111	1.00 (referent)	1.00 (referent)
⩾1 time/month 10–19, 20–29, or 30–39 years	14 377 (18)	34	1.45 (0.98 to 2.14)	1.55 (1.04 to 2.32)
			P = .07	P = .04

Table 5. RRs and 95% CIs for cutaneous malignant melanoma according to solarium use in successive decades of age

Age period and solarium use 2010	Frequencies No. (%)	No. of cases	Age-adjusted RR (95% CI)	Multivariable* RR 95% CI
10-19 y (n = 85,210)				
Never	83,554 (98)	326	1.00	1.00
Rarely or ≥1 time/mo	1,656 (2)	7	1.30 (0.61-2.75)	1.19 (0.56-2.53)
			P_{trend} = 0.51	P_{trend} = 0.66
20-29 y (n = 88,478)				
Never	70,550 (80)	279	1.00	1.00
Rarely	11,560 (13)	41	1.15 (0.81-1.61)	1.08 (0.77-1.53)
≥1 time/mo	6,368 (7)	25	1.39 (0.90-2.14)	1.53 (0.99-2.38)
			P_{trend} = 0.13	P_{trend} = 0.09
30-39 y (n = 87,219)				
Never	43,863 (50)	166	1.00	1.00
Rarely	28,243 (32)	119	1.23 (0.96-1.56)	1.14 (0.89-1.45)
≥1 time/mo	15,113 (17)	71	1.41 (1.06-1.87)	1.49 (1.11-2.00)
			P_{trend} = 0.01	P_{trend} = 0.01
40-49 y (n = 41,022)				
Never	17,063 (42)	68	1.00	1.00
Rarely	14,449 (35)	65	1.12 (0.79-1.57)	1.14 (0.81-1.62)
≥1 time/mo	9,510 (23)	51	1.34 (0.93-1.93)	1.61 (1.10-2.35)
			P_{trend} = 0.12	P_{trend} = 0.02
Combined, 10-39 y (n = 79,042)				
Never in all decades, 10-39 y	37,991 (48)	137	1.00	1.00
Rarely but not ≥1 time/mo in any decade, 10-39 y	26,599 (34)	114	1.36 (1.05-1.75)	1.24 (0.96-1.61)
≥1 time/mo in one decade, 10-39 y	11,576 (15)	48	1.33 (0.95-1.86)	1.38 (0.98-1.94)
≥1 time/mo in two or three decades, 10-39 y	2,876 (4)	16	2.13 (1.25-3.64)	2.37 (1.37-4.08)
			P_{trend} = 0.004	P_{trend} = 0.003

NOTE: Poisson regression analysis. All statistical tests were two-sided. Analyses of solarium use at ages 40 to 49 y included only women ≥40 y when answering the questionnaire.

*Multivariable models included attained age, region of residence, pigmentation characteristics (hair color and skin color after heavy sun exposure in the beginning of the summer and after repeated sun exposure) and solar exposure (corresponding number of age-specific sunburns and weeks on annual bathing vacations).

Veierod et al,. A prospective study of pigmentation, sun exposure and and risk of cutaneous malignant melanoma in women. Journal of the National Cancer Institute, Vol. 95, No. 20, October 15, 2003

- Bataille 2005

- this study was the largest, a 5 country European study which found that the OR forever using a sunbed was 0.90 or a 10% reduced risk of melanoma
- the mean age of first use in the study was 24, so the majority of people in the study would have started indoor tanning below 35 years of age and the OR is close to 1.0, or no risk.
- the WHO IARC study selectively used the only age data reported in the study – Ever sunbed use before age 15 OR 1.82. This is but a sliver of the information contained in this study and clearly does not represent the melanoma risk for all people under age of 35 for this study. This is report bias as information for all people in the study under age 35 was not reported because it was not a risk
- The research concluded *"In conclusion, sunbed and sun exposure were not found to be significant risk factors for melanoma in this case-control study performed in five European countries"*

"In conclusion, sunbed and sun exposure were not found to be significant risk factors for melanoma in this case-control study performed in five European countries"

Bataille et al,. A multicentre epidemiological study on sunbed use and cutaneous melanoma in Europe. European Journal of Cancer 41 (2005) 2141-2149

In summary, the 7 studies used by the WHO IARC in their 2006 sunbed meta-analysis study all have major discrepancy issues and in no way present solid scientific evidence of an increased melanoma risk for people under age 35. Professional medical and research organizations should have first reviewed each of these individual studies in detail before blindly supporting the WHO recommendations. The health organizations should not ever quote the WHO IARC sunbed study of 75% risk for people under age 35, especially when referencing to the risk for commercial tanning facilities. The risk is just 6%.

In addition, all the studies had inconsistent data. It was not a compilation of 7 studies of data on people who were age 35 and under, who ever used a sunbed. There is a mixture of ages, and regular versus ever use of the equipment. In the majority of cases IARC falls short due to publication bias as research studies usually only report sub-analysis results for problems, not for OR results close to 1.0. Bataille is a prime example; they only reported separate results for ever sunbed use before age 15. Obviously they have data available for all ages but chose not to publish it in a chart. What is the risk of all age groups?

Swerdlow 1988 – Age <30 – Ever use of sunbed

Walter 1990 – Age <30 – Ever use of sunbed

Westerdahl 1994 – Age <30 – Ever use of sunbed

Chen 1998 – Age <25 – Ever use of sunbed

Westerdahl 2000 – Age <35 – Regular use of sunbed

Veierod 2003 – Age 20-29 - >1 time/mo (regular)

Bataille 2005 – Age <15 – Ever use of sunbed

The largest study used in the WHO IARC meta-analysis of 7 studies was a cohort study by Veierod 2003 and the study is following 106,366 women. In an updated issue of this cohort study in 2010, the study reported an age adjusted Relative Risk of 1.39 and a Multivariable Relative Risk of 1.53 or an adjusted increased risk of 53% down from the 2003 report which was a risk of 158%. The study reported that for women age 20-29, 70,550 who never used a tanning bed, and 279 of them got melanoma. That works out to an absolute risk of 279/70550=.0039546. For women 20-29y that used a tanning bed >1 time/mo, 6,368 women, 25 of them got melanoma. That works out to an absolute risk of 25/6368=.0039258. So the women who used a tanning bed actually had LESS absolute risk of melanoma than those who did not use a tanning bed.

Table 5 showing the data:

Table 5. RRs and 95% CIs for cutaneous malignant melanoma according to solarium use in successive decades of age

Age period and solarium use	Frequencies No. (%)	No. of cases	Age-adjusted RR (95% CI)	Multivariable* RR 95% CI
10-19 y (n = 85,210)				
Never	83,554 (98)	326	1.00	1.00
Rarely or ≥1 time/mo	1,656 (2)	7	1.30 (0.61-2.75)	1.19 (0.56-2.53)
			P_{trend} = 0.51	P_{trend} = 0.66
20-29 y (n = 88,478)				
Never	70,550 (80)	279	1.00	1.00
Rarely	11,560 (13)	41	1.15 (0.81-1.61)	1.08 (0.77-1.53)
≥1 time/mo	6,368 (7)	25	1.39 (0.90-2.14)	1.53 (0.99-2.38)
			P_{trend} = 0.13	P_{trend} = 0.09
30-39 y (n = 87,219)				
Never	43,863 (50)	166	1.00	1.00
Rarely	28,243 (32)	119	1.23 (0.96-1.56)	1.14 (0.89-1.45)
≥1 time/mo	15,113 (17)	71	1.41 (1.06-1.87)	1.49 (1.11-2.00)
			P_{trend} = 0.01	P_{trend} = 0.01
40-49 y (n = 41,022)				
Never	17,063 (42)	68	1.00	1.00
Rarely	14,449 (35)	65	1.12 (0.79-1.57)	1.14 (0.81-1.62)
≥1 time/mo	9,510 (23)	51	1.34 (0.93-1.93)	1.61 (1.10-2.35)
			P_{trend} = 0.12	P_{trend} = 0.02
Combined, 10-39 y (n = 79,042)				
Never in all decades, 10-39 y	37,991 (48)	137	1.00	1.00
Rarely but not ≥1 time/mo in any decade, 10-39 y	26,599 (34)	114	1.36 (1.05-1.75)	1.24 (0.96-1.61)
≥1 time/mo in one decade, 10-39 y	11,576 (15)	48	1.33 (0.95-1.86)	1.38 (0.98-1.94)
≥1 time/mo in two or three decades, 10-39 y	2,876 (4)	16	2.13 (1.25-3.64)	2.37 (1.37-4.08)
			P_{trend} = 0.004	P_{trend} = 0.003

NOTE: Poisson regression analysis. All statistical tests were two-sided. Analyses of solarium use at ages 40 to 49 y included only women ≥40 y when answering the questionnaire.
*Multivariable models included attained age, region of residence, pigmentation characteristics (hair color and skin color after heavy sun exposure in the beginning of the summer and after repeated sun exposure) and solar exposure (corresponding number of age-specific sunburns and weeks on annual bathing vacations).

WHO'S General Statement on Sunbeds in 2006 Report

There has been some suggestion that the World Health Organization issued a recommendation in 2009 to ban indoor tanning completely for those under 18. This is a generalized statement and all the more unfounded when one considers that the data from this report show tanning salons having the lowest risk, with home units having 7 times that risk with medical units having 16 times the risk of a professional salon. The risk is about who controls the equipment and ensuring that users are not allowed to sunburn. The WHO 2006 IARC Report also said in their executive summary *"Epidemiologic studies to date give no consistent evidence that use of indoor tanning facilities in general is associated with the development of melanoma or skin cancer."*

In 2009 the World Health Organization moved tanning equipment to Group 1, or carcinogenic to humans, the same category as the sun has been in since 1992. This was not as a result of any additional research on tanning equipment. The rationale appears to have been that UV from the sun and UV from sunbeds is the same thing. Most health organizations fail to mention that natural sunlight is a Group 1 carcinogen and the majority of the population is exposed to this type of UV light compared to tanning equipment. The WHO IARC report did not quantify how much UV exposure is carcinogenic, but they did identify risk factors for Skin Types. Also included in Group 1 carcinogens are birth control pills, phototherapy treatments and salted fish.

SUNLIGHT Risk factors according to WHO IARC Report 1992
Total Sun Exposure
10 studies - average was OR 1.75. There were 3 studies that were very high outliers. If removed the OR=0.89

Occupational Exposure
17 studies - average was OR 1.76. There were 3 studies that were very high outliers. If removed the OR=1.03

Intermittent Exposure
35 studies - average was OR 2.17. There were 4 studies that were very high outliers. If removed the OR=1.77

WHO 2012 IARC Monographs Radiation 100 D Report

There has been a number of update from the 2006 IARC report, the increase risks remained the same for all skin cancers, but there were further notations about those risks as it related to genetics, skin type and ability to tan. As well as the meaning of carcinogenic risk and further research on increased risk of phototherapy unit. Some interesting points made in the report are:

Page 1 – Note to Reader
-The term 'carcinogenic risk' in the IARC Monographs series is taken to mean that an agent is capable of causing cancer. The Monographs evaluate cancer hazards, despite the historical presence of the word 'risks' in the title.
Inclusion of an agent in the Monographs does not imply that it is a carcinogen, only that the published data have been examined. Equally, the fact that an agent has not yet been evaluated in a Monograph does not mean that it is not carcinogenic. Similarly, identification of cancer sites with sufficient evidence or limited evidence in humans should not be viewed as precluding the possibility that an agent may cause cancer at other sites IARC 2012

The evaluations of carcinogenic risk are made by international working groups of independent scientists and are qualitative in nature. No recommendation is given for regulation or legislation.

SOLAR AND ULTRAVIOLET RADIATION starts on page 35 and ends on page 90, references from page 90 to 102.

-The median annual exposure dose from artificial tanning is probably 20–30 times the MED.(page 39)

-A typical dose in a single course of UVB phototherapy can be in the range of 200–300 times the MED (page 39)

-Individuals exposed to lighting from fluorescent lamps may typically receive annual exposure doses of UVR in the range of 0–30 times the MED (Page 40)

- (CMM) However, 'chronic' or 'more continuous'exposure, which generally equated with 'occupational' exposure, and total sun exposure (sum of 'intermittent'+'chronic'), generally showed weak, null or negative associations. (page 41)

- First, outdoor workers are not at a substantially increased risk of melanoma (IARC, 1992; Armstrong & Kricker, 2001); second, outdoor workers tend to have a higher than- average ability to develop a tan (Green et al., 1996; Chang et al., 2009).(page 42)

- Page 46 - 52 – reduce risk of cancers with UV exposure

-The four studies on basal cell carcinoma did not support an association with the use of indoor tanning facilities (IARC, 2006a, 2007a). (page 48)

-PUVA has been reviewed previously by two IARC Working Groups and there is sufficient evidence that PUVA therapy is carcinogenic to humans (Group 1), causing cutaneous squamous cell carcinoma (IARC, 1986, 2012), (page 53)

-For those who had more than 100 UVB therapy treatments, the risks, relative to those who received 25 or less such treatments, were 1.22 (95%CI: 0.28–4.25) for basal cell carcinoma, 2.04 (95%CI: 0.17–17.8) for squamous cell carcinoma, and 1.02 (95%CI: 0.02–12.7) for melanoma.(page 61)

-The major steps of UV-induced immune suppression have been determined but it should be noted that, in many instances, these details were obtained following a single or a few exposures of a rodent model or human subjects to UVR and that the dose chosen was sufficient to cause burning. In addition, the source used to emit UVR frequently contained more than 50% UVB (wavelength 280–315 nm), considerably more than natural sunlight. (page 87)

http://monographs.iarc.fr/ENG/Monographs/vol100D/index.php

Credibility of Research

In an article published by Forbes magazine, Geoffrey Kabat discusses how activism distorts the assessment of health risks. 1. IARC considers experimental evidence of carcinogenicity but gives priority to human epidemiologic evidence. But – epidemiologic studies are subject to high rates of false positives. 2. Some of the working groups convened to assess a particular agent have included scientists who have carried out the studies on the agent under evaluation. It is fanciful to think that scientists who have a vital stake in a particular question can evaluate the evidence, including their own studies dispassionately. 3. IARC reaches its assessments by consensus. But this can mean that those who are more forceful and persuasive may influence the group decision making process. In addition, consensus implies a philosophic stance which has nothing to do with science. 4. Invocation of the precautionary principle focuses attention solely on the possibility of harm, often ignoring information about the dose to which people are exposed, avoiding consideration of benefits of the agent in question.

> *"epidemiologic studies are subject to high rates of false positives"*

We need to recognize that scientists are human and can be influenced by pressures and agendas that have nothing to do with science. People who know the answer and have an agenda are believers and advocates, and they should have no role in assessing the science.

Geoffrey C. Kabat is a cancer epidemiologist at the Albert Einstein College of Medicine and the author of Hyping Health Risks: Environmental Hazards in Daily Life and the Science of Epidemiology
http://www.forbes.com/sites/realspin/2012/11/20/how-activism-distorts-the-assessment-of-health-risks/

Absolute Risk vs Relative Risk

Much has been made of the oft-cited 75% increase in risk of developing melanoma but this figure should be viewed in its appropriate context. *"Melanoma is pretty rare and almost all the time, the way to make it look scarier is to present the relative change, the 75 percent increase, rather than to point out that it is still really rare,"* Dr. L. Schwartz, a general internist at Veterans Affairs Medical Center in White River Junction, Vt. While Dr. Ivan Oransky M.D. elaborated further *"Absolute risk just tells you the chance of something happening, while relative risk tells you how that risk compares to another risk, as a ratio. If a risk doubles, for example, that's a relative risk of 2, or 200 percent. If it halves, it's .5, or 50 percent. Generally, when you're dealing with small absolute risks, as we are with melanoma, the relative risk differences will seem much greater than the absolute risk differences. You can see how if someone is lobbying to ban something – or, in the case of a new drug, trying to show a dramatic effect – they would probably want to use the relative risk."*

http://www.healthjournalism.org/blog/2010/05/tanning-beds-what-do-the-numbers-really-mean/

"Melanoma is pretty rare and almost all the time, the way to make it look scarier is to present the relative change, the 75 percent increase, rather than to point out that it is still really rare," Dr. L. Schwartz, a general internist at Veterans Affairs Medical Center in White River Junction, Vt.

"Absolute risk just tells you the chance of something happening, while relative risk tells you how that risk compares to another risk, as a ratio. If a risk doubles, for example, that's a relative risk of 2, or 200 percent. If it halves, it's .5, or 50 percent. Generally, when you're dealing with small absolute risks, as we are with melanoma, the relative risk

Indoor Tanning is Addictive

Some make the accretion that indoor tanning is addictive. Sunshine, UV exposure causes the same effect. It's natural. It's why you feel good on a sunny spring day after a long winter. Tanning is not an addiction. It's an attraction. Humans have been biologically programmed to be attracted to UV light. We need it to live. To call it an addiction is like saying that people are addicted to oxygen or water. It just isn't accurate. Here's the core science that is being twisted: Ultraviolet light exposure produces endorphins in the skin — substances that literally make us feel good. Because sunshine is natural and humans need sunshine in order to be healthy, endorphin production is nature's way of telling us that sunshine is good. Just as food does the same thing. Tanning (color of the skin) doesn't actually produce endorphins. It is UV light that has this effect, and you do not have to produce a tan to create endorphins.

Solariums - Skin Cancer Risk vs Overall Cancer Reduction

So are people who use solariums increasing their total risk of cancer as most health organizations suggest? Or do solariums provide an overall reduction in total cancers? Consider these studies:

Solarium use was found to be inversely associated with breast cancer incidence in a large Swedish cohort study. The study reported *"reduced breast cancer risk consistently appeared among women who spent one week or*

more per year on sunbathing vacations between ages 10 and 29 years, or who used solarium between ages 10 and 39 years, after controlling for the other risk factors". "*A 15% statistically significant decreased risk of breast cancer was found for women whose skin colour was brown after chronic sun exposure, compared with those whose skin was light or never brown".* The study reported that 10-39 year old women who used a solarium and tanned >1 time/month in two or three decades, had a statistically significant 37% reduced risk of breast cancer incidence (HR 0.63 (0.41-0.96)).

Yang et al., Prospective Study of UV Exposure and Cancer Incidence Among Swedish Women. Cancer Epidemiol Biomarkers Prev; 20(7) July 2011

Solarium use was found to be inversely associated with breast cancer incidence in a large Swedish cohort study. The study reported *"reduced breast cancer risk consistently appeared among women who spent one week or more per year on sunbathing vacations between ages 10 and 29 years, or who used solarium between ages 10 and 39 years, after controlling for the other risk factors".*

Yang et al., Prospective Study of UV Exposure and Cancer Incidence Among Swedish Women. Cancer Epidemiol Biomarkers Prev; 20(7) July 2011

A study from Ontario, Canada of 972 breast cancer cases from the Ontario Cancer Registry were reviewed and matched to 1135 controls. They found that reduced breast cancer risks of 35% were associated with increased sun exposure from ages 10 to 19. They concluded "*We found strong evidence to support the hypothesis that vitamin D could help prevent breast cancer. Our results suggest that exposure earlier in life, particularly during breast development, may be most relevant*".

Knight JA, Lesosky M, Barnett H, Raboud JM, Vieth R. Vitamin D and reduced Risk of Breast Cancer: A Population-Based Case-Control Study. Cancer Epidemiol Biomarkers Prev 2007;16(3). 422-29 March 2007

This large population-based, case-control study of 3,101 people in Ontario, Canada found that increased time spent outdoors (>21 vs <6 hours/week) between May and September was associated with reduced breast cancer risk of 29% for woman in the teenage years, 36% reduction for age 20-30, 26% reduction for age 40 – 50 and a 50% reduction for women aged 60-74 years old. The authors concluded "*This study suggest that factors suggestive of increased cutaneous production of vitamin D are associated with reduced breast cancer risk*."

Anderson LN, Cotterchio M, Kirsh VA, Knight JA. Ultraviolet Sunlight Exposure During Adolescence and Adulthood and Breast Cancer Risk: A Population-based Case-Control Study Among Ontario Women. Am J Epidemiol. 2011 Aug 1;174(3):293-304. doi: 10.1093/aje/kwr091. Epub 2011 Jun 9.

A study of sunlight and breast cancer incidence in the USSR found that the states which were at a higher latitude and that received the least sunlight had 2-3 times higher incidence rate of breast cancer. The study concluded "*The pattern of increased breast cancer incidence in regions of low solar radiation in the USSR is consistent with the geographical pattern seen for breast cancer mortality in the US and worldwide*."

Gorham ED, Garland FC, Garland CF. Sunlight and Breast Cancer Incidence in the USSR. Int J Epidemiol. 1990 Dec;19(4):820-4.

This study from Ontario, Canada showed that woman with deficient vitamin D levels were 94% more likely to have distant recurrence of breast cancer and 73% more likely to die from the disease compared to those with sufficient vitamin D levels. The study concluded "Vitamin D deficiency may be associated with poor outcomes in breast cancer."

Goodwin PJ, Ennis M, Pritchard KI, Koo J, Hood N. Prognostic Effects of 25-Hydroxyvitamin D levels in Early Breast Cancer. J Clin Oncol. 2009 Aug 10;27(23):3757-63. doi: 10.1200/JCO.2008.20.0725. Epub 2009 May 18.

A large population-based case-control study in Mexico found that breast cancer risk was reduced by 47% for women who had 25(OH)D blood serum levels greater than 30 ng/ml (75 nmol/L) compared with those < 20 ng/ml (50 nmol/L). The study concluded *"our findings strongly support the hypothesis that higher vitamin D status may reduce risk of breast cancer in both pre and postmenopausal women in Mexico".*

Fedirko et al., Serum 25-Hydroxyvitamin D and risk of breast cancer: results of a large population-based case-control study in Mexican women. Cancer Causes Control DOI 10.1007/s10552-012-9984-

Solarium use was found to prevent other cancers as well.

The Yang study reported that 10-39 year old women who used a solarium and tanned >1 time/month in two or three decades, had a statistically significant 37% reduced risk of breast cancer incidence (HR 0.63 (0.41-0.96)). The study also showed positive incidence reductions with other cancers – 46% less ovarian cancer incidence, 51% less lung cancer incidence, and 79% less brain cancer incidence.

Yang et al., Prospective Study of UV Exposure and Cancer Incidence Among Swedish Women. Cancer Epidemiol Biomarkers Prev; 20(7) July 2011

A tanning bed does provide the UV dosage quicker than outdoor sunshine. A session in the average tanning equipment can run from 7 minutes to 20 minutes. The UV dosage that you would get would be similar in total amount. This would be similar to the exposure dose that you would get outdoors in the summer sun at noon for approximately 60 minutes.

The media often quote a study by Gerber that tanning beds emit 10-15 times more UVA than the midday sun. This has not been confirmed. In fact a study by Donald Smith reports that a high pressure tanning bed only emits 1.7 times more UVA than the sun. "*When valid data is compared, we find that a sunbed equipped with traditional low pressure sunlamps emits 20% less UVA than sunlight; a sunbed equipped with a new-era sunlamp emits only 30% (1.3 times) more UVA than sunlight; and a HID/high pressure sunbed emits only 70% (1.7 times) more UVA than sunlight. However, a PUVA booth like those used by the dermatology community emits 5.8 times (580%) more UVA than sunlight and a xenon solar simulator like those used for sunscreen testing emits 6.8 times (680%) more UVA than sunlight*." (Donald L Smith)

Gerber et al., Ultraviolet emission spectra of sunbeds. Photochemistry and photobiology, 2002, 76(6): 664-668
Patricia E. Reykdal, Donald L. Smith, Sunlight vs Sunbeds: The Truth About UVA

There appear to be no scientific studies that show that individuals are at higher risk if they receive UV exposure in a higher, quicker dosage, nor are there specific research references that we know of.

The real risk of sunbeds was published in the 2006 study by WHO IARC as a 15% lifetime increased risk of melanoma when you included a Skin Type 1 person (always burn, never tan), based on a meta-analysis of 19 studies.

WHO IARC Working Group on artificial ultraviolet (UV) light and skin cancer. The association of use of sunbeds with cutaneous malignant melanoma and other skin cancers: A systematic review. Int. J. Cancer: 120, 1116-1122 (2006)

In 2010, researchers from Lund University in Sweden found that the health benefits of sun exposure far outweighed the risks – as long as you sunbathe sensibly. Oncologist Hakan Olsson told a Swedish newspaper *'Our studies show that women with active sunbathing habits live longer.'* They reported their controversial finding after studying the effect of sun exposure on 40,000 Swedish women.

Lindqvist PG et al., Does an active sun exposure habit lower the risk of venous thrombotic events? A D-lightful hypothesis. J Thromb Haemost 2009; 7: 605-10

In 2008, Johan Moan from the University of Oslo in Norway published a paper – to try and answer the question – *"will increased sun exposure lead to net health benefits or risks?"* The study concluded *"These data indicate that increased sun exposure may lead to improved cancer prognosis and, possibly, give more positive than adverse health effects."*

Moan J. et al,. Addressing the health benefits and risks, involving vitamin D or skin cancer, of increased sun exposure www.pnas.org/cgi/doi/10.1073/pnas.0710615105

Professor Tim Oliver, Medical Oncologist, Barts and the London Hospital, UK – *"Current medical advice is to cover up in the sun, but I believe the health benefits of exposure to its UVA and UVB rays greatly outweigh the disadvantages, even if that means using a sunbed during winter months."*

http://www.dailymail.co.uk/health/article-1127175/Top-cancer-doctor-says-SHOULD-sunbed-session.html

In a 2012 review of sunbeds, vitamin D and health, researchers determined that tanning bed use would decrease 10 times as many cancers than they might contribute to. They reported "*Due to the fear of skin cancer, health authorities warn against sun and sunbed exposure. This policy, as well as the recommended vitamin D doses, may need revision.*" They elaborated further "*Tanning is thought to protect DNA and reduce carcinogenesis as indicated by the low skin cancer risk of dark-skinned people. 10 min of exposure to sunbeds, twice weekly, give similar vitamin D levels as a daily intake of 2000 IU of vitamin D and can bring a winter level of vitamin D up to a summer level (70-90 nmol/L), which may be optimal.*" Other points made; occupational exposure (farmers, fisherman) and regular weekend sun exposure are associated with decreased risk of CMM. UV exposure earlier in life is related to reduced overall and breast cancer. The study concluded "*The overall health benefit of an improved vitamin D status may be more important than the possibly increased CMM risk resulting from carefully increasing UV exposure.*"

Moan J, Baturaite Z, Juzeniene A, Porojnicu AC. Vitamin D, sun, sunbeds and health. Public Health Nutr. 2012 Apr;15(4):711-5. doi: 10.1017/S1368980011002801. Epub 2011 Oct 24

"Tanning is thought to protect DNA and reduce carcinogenesis"

Moan J, Baturaite Z, Juzeniene A, Porojnicu AC. Vitamin D, sun, sunbeds and health. Public Health Nutr. 2012 Apr;15(4):711-5. doi: 10.1017/S1368980011002801. Epub 2011 Oct 24

Skin Types

Dr. Fitzpatrick created a skin typing chart to reduce the risk of overexposure for phototherapy.

http://bradsmelanomafoundation.org/skin_type

http://r.b5z.net/i/u/10030785/i/Fitzpatrick_Skin_Types.jpg

UV risk is based on skin type. This is why the JCTA has been pushing for professional standards for provinces which would include a ban on skin type 1 individuals using sunbeds, regardless of their age.

The WHO IARC Report in 2006 states the following on skin types *"There is a considerable range of susceptibility of the human skin to the carcinogenic effects of UV radiation, and in humans, there is an estimated 1000-fold variability in DNA repair capacity after UV exposure (Hemminki et al., 2001).*

Susceptibility to sun-induced skin damage is closely related to pigmentary traits, and subjects having the following characteristics are at increased risk for developing a skin cancer (melanoma, SCC and BCC):

• Red hair, followed by blond hair, followed by light brown hair.

• Skin phototype (Fitzpatrick, 1988): subjects who always burn and never tan when going unprotected in the sun (skin phototype I) have a much higher risk for skin cancer than subjects who never burn and always develop a deep tan (skin phototype IV). Intermediate risk categories are subjects who always burn then develop a light tan (skin phototype II), and subjects who sometimes burn and always develop a tan (skin phototype III). Subjects of skin phototypes V and VI belong to populations with natural brown or black skin, and are resistant to sunlight.

• Freckles (ephelides) on the face, arms or shoulders. The skin cancer risk increases with increasing sensitivity to freckling.

• Skin colour: pale colour, followed by increasing depth of pigmentation.

• Eye colour: blue, followed by grey/green eyes, then by brown eyes.

World Health Organization (WHO), International Agency for Research on Cancer (IARC), Exposure to Artificial UV Radiation and Skin Cancer. IARC 2006 Page 9

This reinforces the fact that the risk factors for melanoma are genetic, with skin type and moles being solid indicators of susceptibility. The focus should be on people of all ages that represent the genetic risk. This is exactly what the JCTA has been asking for, namely an outright ban on users who are Skin Type I.

Understanding UV Exposure

Annual U.S. Available UV Exposure – Northern USA – 620,000 J

Indoor Worker – 25,000 J
Outdoor Worker – 75,000 – 225,000 J

Maximum Exposure for one UV sunbed session – 624 J

12 sessions = 4,200 J
30 sessions = 15,430 J

An indoor worker having 30 sunbed sessions/year would still be at least 40% less than the total UV exposure of an outdoor worker.

Sunbed session vs Day at the Beach

Sunbed session = 624 J
Day at the Beach = 4,013 J
Day at the Beach is 6.4 times the UV from one sunbed session

Godar DE. UV doses worldwide. Photochem Photobiol. 2005 Jul-Aug;81(4):736-49.

Skin Cancer

The most common Skin Cancers are – Basal Cell Carcinoma, Squamous Cell Carcinoma and Melanoma.

NMSC rates have remained the same or declined since 2005. 2005 would be about the time, if indoor tanning was a risk for NMSC, that it would start showing up on the incidence rates. Since NMSC take about 20 to 30 years to metastasize. For some reason these number fluctuate or maybe the words is readjusted.

Canadian Cancer Society statistics on Non-melanoma Skin Cancer in Canada (Estimates Only – no actual numbers)

2005 – 78,000	2006 – 68,000
2007 – 69,000	2008 – 73,000
2009 – 75,100	2010 – 75,500
2011 – 74,000	

The incidence of melanoma in Canada has slowed since the indoor tanning industry started in the early 1980s. Incidence rates according to the Canadian Cancer Society have dropped from 6 per 100,000 for males and 4.6 per 100,000 to 1.4 per 100,000 for both female and males over recent years.

"The relationship of UV radiation to development of skin cancer differs for melanoma and non-melanoma skin cancer and depends on the interplay of genetic susceptibility, the intermittent or chronic nature of time spent in the sun, and lifetime acquisition of sunburns"

Lazovich et al., Time to get serious about skin cancer prevention. Cancer Epidemiol Biomarkers Prev DOI:10.1158/1055-9965 EPI-12-0327

Basal Cell Carcinoma (BCC) – Makes up 80% of all Skin Cancers

A number of health organizations insinuate that UV exposure from sunbeds is one of the largest risks for Non Melanoma Skin Cancer. Numerous studies show that there is no relationship between sunbeds and Basal Cell Carcinoma (BCC).

The 2006 IARC WHO study that was peer reviewed and published on sunbeds also included a meta-analysis of studies for BCC. They reviewed 4 studies on sunbeds and BCC. They reported an OR of 1.03 or a 3% increased risk of BCC for those who have used a sunbed. This was not considered significant. *"For basal cell carcinoma, the studies did not support an association."*

> According to the 2006 & 2012 WHO IARC Reports *"For basal cell carcinoma, the studies did not support an association."*

IARC The association of use of sunbeds with cutaneous malignant melanoma and other skin cancers. A systematic review. Int. J. Cancer: 120, 1116-1122 (2006)
World Health Organization (WHO), International Agency for Research on Cancer (IARC), Exposure to Artificial UV Radiation and Skin Cancer. IARC 2006

The WHO IARC Monograph Radiation 100D Report from 2012 reviewed the most up to date current published studies on indoor tanning equipment and BCC. They reported "*The four studies on basal cell carcinoma did not support an association with the use of indoor tanning facilities (IARC, 2006a, 2007a).*"

http://monographs.iarc.fr/ENG/Monographs/vol100D/index.php

This confirms that there is no association between UV-emitting tanning equipment and BCC.

In fact some published research studies even show a reduction in BCC with the use of sunbeds. For example:

A study just released examined the relationship between sunlight exposure and risk of NMSC. Researchers found that lifetime tanning bed use, >10 times, provided a 36% reduced risk of BCC (OR 0.64). The study concluded "*A history of blistering sunburn (a measure of intermittent sunlight exposure) was associated with both BCC (OR = 1.96) and SCC (OR = 2.02).*

Iannacone et al., Patterns and timing of sunlight exposure and risk of basal cell and squamous cell carcinomas of the skin – a case-control study. BMC Cancer 2012, 12:417 doi:10.1186/1471-2407-12-417

A 1996 Canadian study with data from the Alberta Cancer Registry evaluated non-solar ultraviolet radiation and the risk of basal and squamous cell skin cancer. The study showed no evidence of elevated risk for BCC (Basal Cell Carcinoma) or SCC (Squamous Cell Carcinoma) among subjects exposed to various types of NSUVR (non solar ultraviolet radiation) including sunlamps and sunbeds.

Bajdik et al,. Non-solar ultraviolet radiation and the risk of basal and squamous cell skin cancer. British Journal of Cancer (1996) 73, 1612-1614

Regarding the role of artificial UV radiation sources, this study did not provide any evidence of increased BCC risk due to exposure to sunbeds or sunlamps. The absence of risk could be due to the fact that exposure to UV radiation is small compared with that from sunlight, and the latter is likely to overwhelm any effect due to

artificial sources of UV radiation. The results of most published studies aimed at investigating the relative importance of phenotypic traits, sun sensitivity and different indicators of sun exposure in the development of BCC are largely inconsistent.

Corona et al,. Risk factors for Basal Cell Carcinoma in a Mediterranean population. Arch Dermatol/Vol 137, Sep 2001

A study that evaluated Basal Cell Carcinoma in young women found that although women with BCC had almost twice as many tanning salon visits (152.2 vs 83.1) on average, this was not considered to be statistically significant. Young women with a BCC are more likely to have a past or current history of cigarette smoking and blistering sunburns.

Boyd et al,. Basal cell carcinoma in young women: an evaluation of the association of tanning bed use and smoking. J Am Acad Dermatol. 2002 May;46(5):706-9

- Types of UV exposure that affects BCC risk.

The relationship between UV and BCC is complex. A large study in the UK looking at risk factors for BCC found outdoor occupation was not a factor; thus, intermittent recreational exposure may be more important than chronic ultraviolet exposure which can lead to sunburn because a person has not pre-conditioned their skin for higher level of UV light.

Lear et al,. Risk factors for basal cell carcinoma in the UK: case-control study in 806 patients. J R Soc Med 1997;90:371-374

- Ability to Tan

A study from Australia found that *"People who reported that their skin tanned deeply had lower rates for both types of NMSC than people whose skin did not tan. Rates increased steadily with a decreasing tendency of the skin to tan. The increase from lowest to highest rates was about 2-2.5 times for BCC and 3.5-3.6 times for SCC."*

Staples et al,. Non-melanoma skin cancer in Australia: the 2002 national survey and trends since 1985. MJA 2006; 184:6-10

- Family History

A remarkably strong association emerged for family history of skin cancer, emphasizing the importance of genetic predisposition to BCC. Subjects reporting a family history of skin cancer had an extremely increased risk of BCC with an OR of 17.8 or a 17 times greater risk of BCC.

Corona et al,. Risk factors for Basal Cell Carcinoma in a Mediterranean population. Arch Dermatol/Vol 137, Sep 2001

- HPV

Not all skin cancers are caused from sun or UV exposure. Skin cancer from human papilloma virus (HPV) develops on genital skin in both men and women. It is estimated that half of all deaths from skin cancer other than melanoma are from genital skin cancer.

A study published in 2007 looked at non-melanoma skin cancer mortality rates in the United States from 1969 to 2000. It found that 40% of deaths were due to NMSC arising on genital skin. The study concluded *"These data suggest that greater emphasis could be placed on the risk of mortality from genital skin cancer".* It went on to say "*The magnitude of the public health burden is great; nevertheless, efforts on the part of the*

dermatology community to prevent human papilloma virus infection in the United States have been slight compared to similar effort to reduce excess exposure to UV light."

Lewis and Weinstock. Trends in Nonmelanoma Skin Cancer Mortality Rates in the United States, 1969 through 2000. Journal of Investigative Dermatology (2007) 127, 2323-2327

- Other Factors for BCC

Skin cancer patients whose childhood included periods of neglect or maltreatment are at a much greater risk for their cancers to return when they face a major stressful event, based on a new study. The research suggests that such experiences during a person's youth can set a lower level of immune response for life, which in turn might make them more susceptible to the kind of cancers that are often successfully fought by the immune system, so-called immunogenic tumors.

Fagundes et al,. Basal Cell Carcinoma. Stressful Life Events and the Tumor Environment. Arch Gen Psychiatry/Vol 69 (No. 6), June 2012

Squamous Cell Carcinoma (SCC) – Makes up 15% of all Skin Cancers

The UV role in squamous cell carcinoma is also complicated. UV exposure whether through outdoor sunshine or a sunbed is the same – 95% UVA and 5% UVB. A photon of light is a photon of light. The same risk of over-exposure and the same benefits (ie.Vitamin D production) of exposure.

A study completed from the Alberta Cancer Registry reported that age-adjusted crude odds ratios for phenotype and pigmentary factors demonstrate an increased risk of SCC (Squamous Cell Carcinoma) for subjects with light skin colour and red hair who burn rather than tan when first exposed to the sun and who are unable to develop a tan even after a week or more of exposure to sunshine. It also stated *"After adjustment for the mother's ethnic origin, hair colour, and skin colour, no association was seen with recreational sun exposure during childhood and adolescence".* It concluded: *"Thus, it might be hypothesized that subjects at risk of SCC are those who are phenotypically sensitive to the sun (fair skin, red hair, propensity to burn rather than tan in the sun), develop severe sunburns in childhood as initiating events in the sequence of development of malignancy".*

Gallagher et al,. Sunlight Exposure, Pigmentation Factors and Risk of Nonmelanocytic Skin Cancer II. Squamous Cell Carcinoma. Arch Dermatol 1995 Feb;131(2):164-9

". . . subjects at risk of SCC are those who are phenotypically sensitive to the sun (fair skin, red hair, propensity to burn rather than tan in the sun), develop severe sunburns in childhood as initiating events in the sequence of development of malignancy."

Gallagher et al,. Sunlight Exposure, Pigmentation Factors and Risk of Nonmelanocytic Skin Cancer II. Squamous Cell Carcinoma. Arch Dermatol 1995 Feb;131(2):164-9

A prospective study of 107,900 predominantly white women aged 30-55 years at baseline in 1976 (Nurses Health Study) found that red hair RR 2.0 and light brown hair RR 1.7 were associated with an increased risk of

SCC compared to dark brown hair. In addition, the actual number of severe burns appeared to be a more important factor – RR 2.4. Finally, current cigarette smokers showed a 50% increase in the risk of SCC compared with those who had never smoked RR 1.5.

Grodstein et al,. A prospective study of incident squamous cell carcinoma of the skin in the nurses' health study. J Natl Cancer Inst. 1995 Jul 19;87(14):1061-6

The JCTA guidelines for tanning salons recommend that people with Skin Type 1, that always burns and never tans, do not use a sunbed or UV tan. In fact, a tan will protect Skin Type 2 and above people from burning.

- Diet

Diet also plays an enormous role in the development of SCC. Dr. Homer Black from Baylor College completed a study in 1995 that found that patients who maintained a low fat diet of 21% fat, reduced their risk of non-melanoma skin cancer by over 90%.

Black H, et al, Evidence that a low-fat diet reduces the occurrence of non-melanoma skin cancer. Int. J. Cancer: 62, 165-169 (1995)

Research reported by Dr. Hughes and colleagues in the International Journal of Cancer indicated that of those people who had previously had at least one squamous cell carcinoma (SCC), those who had the highest intake of green leafy vegetables had only 45% of the risk of developing another. Furthermore, those with the highest intake of dairy had two-and-one-half times the risk.

Hughes et al,. Food intake and risk of squamous cell carcinoma of the skin in a community: The Nambour skin cancer cohort study. Int J Cancer 2006

- Ability to Tan

A study from Australia found that *"People who reported that their skin tanned deeply had lower rates for both types of NMSC than people whose skin did not tan. Rates increased steadily with a decreasing tendency of the skin to tan. The increase from lowest to highest rates was about 2-2.5 times for BCC and 3.5-3.6 times for SCC."*

Staples et al,. Non-melanoma skin cancer in Australia: the 2002 national survey and trends since 1985. MJA 2006; 184:6-10

- Other Factors

By 1977, psoralen and ultraviolet A (PUVA) was established as a highly effective medical treatment therapy for psoriasis. A follow-up on a 30 year prospective study found that exposure to more than 350 PUVA treatments greatly increases the risk of SCC.
Stern et al,. The risk of squamous cell and basal cell cancer associated with psoralen and ultraviolet A therapy: A 30-year prospective study. JAAD 20 January 2012

This type of phototherapy is listed as a Group 1 carcinogen by the WHO and is still used today no matter what age you are.

Arsenic is also a well-known Group 1 (IARC) carcinogen. Skin cancer is the most common malignancy associated with arsenic ingestion through drinking water from wells. Substantial evidence led the International Agency for Research on Cancer (IARC) to conclude that ingestion of inorganic arsenic can cause skin cancer.
IARC Monographs on the Evaluation of the carcinogenic Risk of Chemicals to Man: Some Metals and metallic Compounds, Vol 23. Lyon 980

A case-control study in New Hampshire, USA found that among individuals with toenail arsenic concentrations above the 97th percentile the adjusted odds ratios were 2.07 for SCC and 1.44 for BCC, compared with those with concentrations at or below the median.

Karagas et al,. Skin cancer risk in relation to toenail arsenic concentrations in a US population-based case-control study. Americal Journal of Epidemiology Vol. 153, No. 6 2001

A study just released in June 2012 reviewed the link between cutaneous human papillomavirus (HPV) infection and the risk of squamous cell carcinoma (SCC). The study *reported "SCC was significantly associated with seropositivity to any genus beta HPV type (OR=1.93; 95% CI=1.23-302)."* That's a 93% increased risk! The study concluded *"These findings support a role for cutaneous HPV as a co-factor in SCC carcinogenesis"*

Iannacone et al,. Case-control study of cutaneous human papillomaviruses in squamous cell carcinoma of the skin. Cancer Epidemiol Biomarkers Prev Published Online First June 15, 2012

Melanoma – Makes up 5% of all Skin Cancers

The dermatologist associations would have you believe that 90% of all skin cancers and melanoma are the result of UV exposure. That simply is not true. If that was true, than the more UV exposure you received, the higher your risk would be for melanoma.

However numerous melanoma studies have proven that chronic exposure, defined as continuous regular exposure, provides a reduced risk of melanoma. Dermatologists cannot explain this flaw in their logic.

The World Health Organization (WHO) has acknowledged that indoor workers — who get 3-9 times less UV exposure than their outdoor counterparts – get more melanomas.

The World Health Organization's web site in a statement on melanoma it states "*Tumour development may be linked to occasional exposure to short periods of intense sunlight, such as at weekends or on holiday. The higher incidence of malignant melanoma in indoor workers compared to outdoor workers support that notion."*

http://www.who.int/uv/faq/uvhealtfac/en/index2.html

The WHO in its "Ultraviolet radiation and human health – Fact Sheet No. 305, December 2009" reported – *"Between 50% and 90% of skin cancers are due to UV radiation",* indicating the high degree of uncertainty which exists around the interrelationship between UV radiation and skin cancer. Research indicates that it's the type of exposure that is the risk; intermittent and sun burning exposure.

World Health Organization (WHO) – Ultraviolet radiation and human health. Fact sheet No. 305, December 2009

Genetic Risk for Skin Cancer

Basal Cell Carcinoma BCC (80% 0f all skin cancers)

Only a fraction of individuals who have been exposed to increasing levels of solar UV radiation will develop BCC or SCC (NMSC), suggesting a genetic susceptibility to UV light-induced carcinogenesis in the general population. DNA repair capacity is under genetic control and was associated with a 62% increase risk of BCC and a 63% increase in SCC.

Wang et al,. Repair capacity for UV light-induced DNA damage associated with risk of nonmelanoma skin cancer and tumor progression. Clin Cancer res 2007;13(21) November 1, 2007

A study looking at DNA repair found *"Mutations in Hedgehog pathway related genes, especially PTCH1, are well known to represent the most significant pathogenic event in BCC. However, specific UV-induced mutations can be found only in approximately 50% of sporadic BCCs. Thus, cumulative UVB radiation <u>can not</u> be considered to be the single etiologic risk factor for BCC development."*

Rass et al,. UV damage and DNA repair in malignant melanoma and nonmelanoma skin cancer. Adv Exp Med Biol. 2008;624:162-78

Research here reports that carriers of two MC1R gene variants had an increased risk of squamous cell carcinoma OR 3.77, nodular basal cell carcinoma OR 2.26 and superficial multifocal basal cell carcinoma OR 3.43. Carriers of one MC1R variant had half the risk. When subjects were stratified by skin type and hair color, analysis showed that these factors did not materially change the relative risks. Therefore, we conclude that MC1R gene variants, fair skin and red hair are independent risk factors for nonmelanoma skin cancer. The study concluded "*These findings indicate that MC1R gene variants are important independent risk factors for nonmelanoma skin cancer*."

Bastiaens et al,. Melanocortin-1 Receptor Gene Variants Determine the Risk of Nonmelanoma Skin Cancer Independently of Fair Skin and Red Hair. Am. J. Hum. Genet. 68:884-894, 2001

Squamous Cell Carcinoma SCC (15% of all Skin Cancers)

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Bastiaens et al,. Melanocortin-1 Receptor Gene Variants Determine the Risk of Nonmelanoma Skin Cancer Independently of Fair Skin and Red Hair. Am. J. Hum. Genet. 68:884-894, 2001

Malignant Melanoma MM (5% of all Skin Cancers)

It is questionable how much UV plays a role in the development of melanoma. Davies reports that 66% of all malignant melanomas are due to BRAF mutations and UV does not play a role in BRAF mutations. Plus Whiteman reports UV exposure is also not involved in Acral melanoma – commonly found on palms, soles, nails and mucous membranes.

Davies et al,. Mutations of the BRAF gene in human cancer. Nature Vol 417 27 June 2002
Whiteman et al,. The melanomas: A synthesis of epidemiological, clinical, histopathological, genetic, and biological aspects, supporting distinct sub-types, casual pathways, and cells of origin. Pigment Cell Melanoma Res. Doi: 10.1111/j.1755-148X.2011.00880.x

It is questionable how much UV plays a role in the development of melanoma. Davies reports that 66% of all malignant melanomas are due to BRAF mutations and UV d*oes* not play a role in BRAF mutations.

"Sun exposure is commonly supposed to be the main cause of cutaneous malignant melanoma (CMM) in most populations. However, the matter is disputed, and we have reviewed the arguments for and against a causation. Several factors are probably involved, as exemplified by a relationship sometimes found between gross domestic product and CMM incidence. Intermittent sun exposure and severe sunburn in childhood are associated with an increased risk of CMM. CMM incidence rates per unit skin area are larger on trunk (intermittently exposed) than on head and neck, while the opposite is true for basal cell and squamous cell carcinomas. Occupational exposure (farmers, fishermen) and regular weekend sun exposure are associated with decreased risk of CMM. Sun exposure may even protect against CMM on shielded skin sites, and CMM arising on skin with signs of large UV exposure has the best prognosis. UV exposure earlier in life is related to reduced overall and breast cancer. It has also been observed that patients with the highest blood levels of vitamin D have thinner CMM and better survival prognosis from CMM."

Moan et al,. Vitamin D, sun, sunbeds and health. Public Health Nutrition doi:10.1017/S1368980011002801

A tan protects against UV-induced DNA damage in 3 ways. Increased pigmentation and cornification (skin thickening) guard against UV skin penetration. In addition, increased concentrations of vitamin D compound in skin resulting from UV exposure act to protect against DNA damage through the reduction of nitric oxide products and increase p53 expression, which facilitates DNA repair.

Mason et al,. Photoprotection by 1,25-dihydroxyvitamin D and analogs: Further studies on mechanisms and implications for UV-damage. Journal of Steroid Biochemistry & Molecular Biology 121 (2010) 164-168

A tan protects against UV-induced DNA damage in 3 ways.

A study looked at repair kinetics of UV-damaged DNA among healthy individuals and melanoma patients. They found a *"wide interindividual variation in DNA damage immediately after irradiation and its repair."* They concluded *"The worst-case scenario is that the differences between individuals are multiplicative, resulting in 1000-fold differences in sensitivity in the population, which would be likely to translate into differences in risks of skin cancer."*

Hemminki et al,. Ultraviolet radiation-induced photoproducts in human skin DNA as biomarkers of damage and its repair. IARC Sci Publ. 2001;154:69-79

At a professional, commercial tanning salon, trained operators review clients' skin type, past tanning history, and any medical conditions to develop an exposure time that does not burn the client. This is controlled exposure to a non-burning UV dose. The National Cancer Institute recommendation for primary prevention for melanoma is to avoid intense intermittent exposure to UV radiation. Sunburn is a marker of that exposure. Properly run tanning salons do not burn people, but rather gradually build up their tan so they are protected against intense intermittent exposure that they might encounter outdoors or on a sunny vacation.

National Cancer Institute - Genetics of Skin Cancer (PDQ)
http://www.cancer.gov/cancertopics/pdq/genetics/skin/HealthProfessional/page4

Melanoma is a very complicated disease. A true meta-analysis of melanoma risks was completed by Dr. Sara Gandini in 2005. Her team reviewed over 60 studies and summarized the data. They found the following risk factors for melanoma: large number of moles +589%, freckles +110%, red hair +264%, Skin Type 1 +109%, Family history +74%, sunburns +103%, and intermittent UV exposure +61%. The study found that "Chronic"

(defined as regular, continuous) sun exposure REDUCED the risk of melanoma by 5%. This is consistent with scientific studies of outdoor workers which show a higher, continuous, regular UV exposure results in a lower melanoma rate than indoor workers who get less UV.

Gandini S, et al., Meta-analysis of risk factors for cutaneous melanoma: I. Common and atypical naevi doi:10.1016/j.ejca.2004.10.015
Gandini S, et al., Meta-analysis of risk factors for cutaneous melanoma: II. Sun exposure doi:10.1016/j.ejca.2004.10.016
Gandini S, et al., Meta-analysis of risk factors for cutaneous melanoma: III. Family history, actinic damage and phenotypic factors doi:10.1016/j.ejca.2005.03.034

If you look at the Gandini study and look at four population-based case-control studies of a "well conducted" design which stated that controls with dermatological diseases had been excluded the results for chronic UV exposure were even better and statistically significant with a 36% reduced risk of melanoma (RR = 0.64, 95% CI: 0.51, 0.81)
Gandini S, et al., Meta-analysis of risk factors for cutaneous melanoma: II. Sun exposure doi:10.1016/j.ejca.2004.10.016

Herzfeld PM, Fitzgerald EF, Hwang SA, et al. A case-control study of malignant melanoma of the trunk among white males in upstate New York. Cancer Detect Prev 1993, 17, 601–608.

Holly EA, Aston DA, Cress RD, et al. Cutaneous melanoma in women. I. Exposure to sunlight, ability to tan, and other risk factors related to ultraviolet light. Am J Epidemiol 1995, 141, 923–933.

Holman CD, Armstrong BK, Heenan PJ. Relationship of cutaneous malignant melanoma to individual sunlight-exposure habits. J Natl Cancer Inst 1986, 76, 403–414.

White E, Kirkpatrick CS, Lee JA. Case-control study of malignant melanoma in Washington State. I. Constitutional factors and sun exposure. Am J Epidemiol 1994, 139, 857–868.

In 2004, Dr. Jason Rivers from the Division of Dermatology, University of British Columbia published a paper – Is there more than one road to melanoma. In it he discussed how outdoor workers have a decreased risk of melanoma compared to indoor workers. In addition, how some melanomas form on sun-exposed regions and others do not. The report concluded *"These findings strongly suggest that distinct genetic pathways lead to melanoma".*

Rivers JK. Is there more than one road to melanoma. Lancet 2004 feb 28;363(9410):728-30

Dr. A. Bernard Ackerman MD, recognized by the American Academy of Dermatology in 2004 as Master Dermatologist, wrote a book entitled – The Sun and the Epidemic of Melanoma: Myth on Myth, published in 2008. When reviewing the evidence on the role genetics plays in melanoma, Ackerman stated that *"in my opinion, those who spawn one or more melanomas have a disposition genetic to that malignant neoplasm and without it no amount of sunlight and no length of time of exposure to it is sufficient to galvanize proliferation of the abnormal melanocytes constituent of it*". He went on to say *"the majority of melanomas in Caucasians occur in skin that is free of solar elastosis, that is a sign of unquestionable damage by virtue of exposure excessive and for very long to sunlight".*

In fact, scientists cannot agree on how melanoma comes about. Is it total accumulated UV exposure, intermittent exposure or sun burning exposure? And how does melanoma develop when the UV hits the skin? Does UV cause mutations in genes or do they depress the capability of the skin to marshal an immune response against malignancy? If sunlight is responsible for melanoma, than melanoma would only occur on body sites that receive a lot of sun exposure, and they would be numerous – like freckles. This does not happen.

A Tan provides Photoprotection from skin cancer. A tan is just increased melanin in the skin. *"Melanoma occurs infrequently in type V-VI skin, suggesting that skin pigment plays a protective role [Fitzpatrick's Seventh Edition, 2008:1135]".* Dark skinned people get melanoma on the bottoms of their feet and palms of the hand.

Genetic play a key role in skin cancer; Moles, Nevi, dysplastic nevus. *"Factors genetic, alone, unrelated entirely to sunlight, could be solely responsible for melanoma."*

Dr. A. Bernard Ackerman MD, book – The Sun and the Epidemic of Melanoma: Myth on Myth published in 2008, Second Edition, page 154

MC1R Variants – partial loss of function mutation are associated not only with red hair, fair skin, and poor tanning, but also with increased skin cancer risk independent of cutaneous pigmentation. A study by Demenais found that carrying any one of the four most frequent MC1R variants was associated with an increased risk of melanoma. One variant increased the risk twofold, but having two or more variants increased melanoma risk nearly six fold.

Demenais et al,. Association of MC1R Variants and Host Phenotypes With Melanoma Risk in CDKN2A mutation Carriers: A GenoMEL Study. J Natl Cancer Inst 2010;102:1-16

Adele Green, former chairperson for the IARC 2006 sunbed study, published a research paper in 2011 investigating the risk factors for melanoma on the arms and legs vs trunk. The study concluded *"After multivariate analysis, the strongest risk factor for both limb and trunk melanomas was the presence of more than 10 naevi on the arm (odds ratio limb melanoma =41.4, 95% confidence interval 10.4–164)"* The research also showed that the inability to tan was associated with a higher risk of melanoma than people who could tan.

Green et al, Risk factors for limb melanomas compared with trunk melanomas in Queensland DOI: 10.1097/CMR.0b013e32834ec02f

Doubt has been cast on sunlight as the major causative factor for malignant melanoma. A study by Shipman in 2010 found that sunnier European countries have lower melanoma mortality. *"It is possible that the major factor affecting MM mortality is therefore the difference in skin colour between northern and southern Europe." "In conclusion, this study supports the notion that research in MM epidemiology should focus on identifying genetic, phenotypic and other environmental triggers for fatal MM."*

Shipman et al. Sunnier European countries have lower melanoma mortality. Clinical and Experimental Dermatology doi:10.1111/j. 1365-2230.2011.04024.x

"Cancers arise owing to the accumulation of mutations in critical genes that alter normal programmes of cell proliferation, differentiation and death. Here we report BRAF somatic missense mutations in 66% of malignant melanomas and at lower frequency in a wide range of human cancers."

"The highest frequency of BRAF mutations is in malignant melanoma. <u>This does not seem to be related to the effects of ultraviolet light</u>, the only known environmental risk factor for this disease. The T to A change at nucleotide 1796, which accounts for 92% of BRAF mutations in melanoma is distinct from the CC to TT or C to T changes associated with pyrimidine dimmer formation following exposure to ultraviolet light – these changes commonly found , for example in the TP53 gene in non-melanoma skin cancers."

Davies et al,. Mutations of the BRAF gene in human cancer. Nature Vol 417 27 June 2002

"The highest frequency of BRAF mutations is in malignant melanoma. This does not seem to be related to the effects of ultraviolet light"

A study analyzing somatic mutations in 147 melanomas did not detect UV damage signature mutations in acral, mucosal or ocular melanomas. In addition, BRAF mutations which are found in ~50% of cutaneous melanomas also were not UV induced and does not have the UV signature.

Krauthammer et al,. Exome sequencing identifies recurrent somatic RAC1 mutations in melanoma. Nat Genet. 2012 September ; 44(9): 1006-1014. doi:10.1038/ng.2359

"*The majority of melanomas that occur on skin with little evidence of chronic sun-induced damage (non-CSD melanoma) have mutations in the BRAF oncogene, whereas in melanomas on skin with marked CSD (CSD melanoma) these mutations are less frequent."* This study shows that MC1R variants are strongly associated with BRAF mutations in non-CSD melanomas. *"Tumours on skin with few or no histopathologic signs of CSD, as evidenced by the relative absence of solar elastosis in the surrounding skin, occur in younger individuals and have frequent mutations in the BRAF oncogene (non-CSD melanoma). By contrast, melanomas on skin with signs of CSD affect older individuals, have different patterns of chromosomal aberrations and have lower frequency of BRAF mutations (CSD melanoma)."*

"*We found that BRAF mutations were 6 to 13 times as frequent in those with at least one MC1R variant compared to those with no MC1R variants. The odds ratio increased from 7.2 for individuals with one MC1R variant to 17.0 for those with multiple variants compared to individuals with no MC1R variants. Moreover most BRAF mutations do not show the standard C > T signature of direct UVR induction."*
Landi et al,. MC1R Germline Variants Confer Risk for BRAF-Mutant Melanoma. Science Vol 313 28 July 2006

"69% of the melanomas in patients under the age of 55 y had BRAF mutant tumors, while only 35.3% were BRAF mutant in older patients."

Viros et al,. Improving Melanoma Classification by Integrating Genetic and Morphologic Features. PLoS Med;5:e120 2008

"Mutational activation of BRAF is the earliest and most common genetic alteration in human melanoma."

Dankort et al,. BRAFV600E cooperates with PTEN silencing to elicit metastatic melanoma. Nat Genet. 2009 May ; 41(5): 544-552. doi:10.1038/ng.356

"Our findings suggest that a significant proportion of melanomas arise from nevi. Furthermore, these results demonstrate that PI3K pathway activation serves as a rate-limiting event in this setting, acting at least in part by abrogating OIS (oncogene-induced senescence). The PI3K pathway was often activated through either decreased PTEN or increased AKT3 expression in melanomas relative to their adjacent nevi."

Vredeveld et al,. Abrogation of BRAFV600E-induced senescence by PI3K pathway activation contributes to melanomagenesis. Genes & Development 26:000-000 2012

Even back in 1985 researchers knew that melanoma was primarily related to genetics. Elwood in his Western Canada Melanoma study concluded *"Melanoma risk is also increased in association with a tendency to burn easily and tan poorly and with pigmentation characteristics of light hair and skin colour, and history freckles; the associations with sunburn and suntan are no longer significant when these other factors are taken into account. This shows that pigmentation characteristics, and the usual skin reaction to sun, are more closely associated with melanoma risk than are sunburn and suntan histories."*

Elwood et al - Sunburn, suntan and the risk of cutaneous malignant melanoma - The Western Canada Melanoma Study. Br. J. Cancer (1985), 51, 543-549

Occupational exposure was reviewed in a large study by Elwood in 1997. This study reviewed 20 studies and calculated the OR for heavy occupational exposure at 0.86 or a 14% reduced risk of melanoma if you worked

outdoors in the sunshine. He concluded *" a significantly reduced risk for heavy occupational exposure (OR 0.86)"* He went on to say *"It seems likely that the neutral or protective effect of heavy chronic exposure is related to protective mechanisms such as tanning and skin thickening, but this may not be the total explanation".*

Elwood et al, - Melanoma and sun exposure: An overview of published studies. Int. J. Cancer: 73, 198-203 (1997)

"Odds ratios associated with sun exposure are often no longer significant after adjustment for skin type, which supports a hypothesis that host response to ultraviolet radiation is more important than dose of sun exposure. Individuals with red hair and freckles, or multiple atypical naevi, with or without a family history of melanoma, should avoid sunbeds since their risks of developing both melanoma and non-melanoma skin cancer are already significantly increased. Having fair skin with poor ability to tan, or a freckled complexion with or without red hair, doubles a person's risk of melanoma. Naevi are the most powerful predictor of risk of melanoma. A meta-analysis of observational studies found that an individual who has more than 100 common naevi or more than two atypical naevi has a fivefold to 20-fold increased risk of melanoma (Gandini 2005)"

Bataille et al,. Melanoma – Part 1: epidemiology, risk factors and prevention. BMJ 29 November 2008 Volume 337

We're also discovering that skin tanning has a function in preventing the development of skin cancer. Researchers found that the protein p53, which plays a role in causing the skin to tan after sun exposure, also reduces the risk of melanoma. The ability to tan seems to be a protective factor against skin cancer. *"The number one risk factor for melanoma is an inability to tan,"* said Dr. David E. Fisher, director of the Melanoma Program at Dana-Farber. The study showed that p53, which is a tumor-suppressor protein in the skin, *"has a powerful role in protecting us against sun damage in the skin"* according to Fisher."

Cui et al., Central role of p53 in the suntan response and pathologic hyperpigmentation. Cell 128, 853-864, March 9, 2007

"The number one risk factor for melanoma is an inability to tan," said Dr. David E. Fisher, director of the Melanoma Program at Dana-Farber.

Genetics/Skin Type are a major risk for skin cancer with regards to UV exposure

A study published in the peer-reviewed journal Nature in 2012 found that people with pale skin, red hair, freckles and an inability to tan – the red hair/fair skin phenotype (MC1R variant) – are at the highest risk of developing melanoma, compared to all other pigmentation types. Minimal receptor activity, as in red hair/fair skin polymorphisms, produces the red/yellow pheomelanin pigment, whereas increasing MC1R activity stimulates the production of black/brown eumelanin. Pheomelanin has weak shielding capacity against ultraviolet radiation relative to eumelanin, and has been shown to amplify ultraviolet-A-induced reactive oxygen species. Unlike non-melanoma skin cancers, melanoma is not restricted to sun-exposed skin and ultraviolet radiation signature mutations are infrequently oncogenic drivers. Ultraviolet-radiation-independent events are likely to have a significant role.

The study introduced a conditional, melanocyte-targeted allele of the most common melanoma on coprotein, BRAF V600E, into mice carrying an inactivating mutation in the MC1R gene. They observed a high incidence of invasive melanomas without providing additional gene aberrations or ultraviolet radiation exposure. Selective absence of pheomelanin synthesis was protective against melanoma development. The study concluded "*These data suggest that the pheomelanin pigment pathway produces ultraviolet-radiation-independent carcinogenic contributions to melanomagenesis by a mechanism of oxidative damage.*" This explains why red hair/fair skin (Skin Type 1) people have a higher risk of melanoma. They don't have the type of melanin to protect a person from overexposure.

Mitra et al,. An ultraviolet-radiation-independent pathway to melanoma carcinogenesis in the red hair/fair skin background. Doi:10.1038/nature11624

A study looked at 4 distinct groups of melanoma; chronic sun-induced, Non-chronic sun-induced, acral and mucosal melanoma. Melanomas on skin without chronic sun-induced damage had frequent mutations in BRAF, and were typically found in people who have a large number of moles but fewer solar keratosis and occur at a younger age.

Curtin et al,. Distinct Sets of Genetic Alterations in Melanoma. N Engl J Med 2005;353:2135-47

A study reviewing risk patterns in carcinoma and melanoma of the skin in men found that subjects with an elevated number of naevi had a high and consistent risk increase for CMM of OR 8.4. Other key factors for melanoma included blonde hair, green eyes; however, high hours of outdoor work did not show an increased risk. SCC risk was found in people with blonde/red hair and green eyes who were poor tanners and those with high sun exposure. BCC risk resulted from people with blonde/red hair, green eyes and medium to high sun exposure. The study concluded "*our direct case-case comparison to mitigate a possible bias in comparing results from different studies confirmed previous findings on the association between pale eyes, naevi and CMM, compared to other skin cancers, and the increased risk of BCC for intermittent sun exposure when compared to the risk of SCC."* This study confirms moles and red hair/pale eyes are risk factors for skin cancer and that the protective tan that would be found on outdoor workers prevents melanoma risk.

Zanetti et al,. Comparison of risk patterns in carcinoma and melanoma of the skin in men: a multi-centre case-case-control study. British Journal of Cancer (2006) 94, 743-751. doi: 10.1038/sj.bjc.6602982

Understanding Melanin Pigmentation and MC1R Variants and Skin Cancer Risk

Melanin pigmentation protects the skin from the damaging effects of ultraviolet radiation (UVR). There are two types of melanin, the red pheomelanin and the black eumelanin, both of which are present in human skin. Eumelanin is photoprotective whereas pheomelanin, because of its potential to generate free radicals in response to UVR, may contribute to UV-induced skin damage. Individuals with red hair have a predominance of pheomelanin in hair and skin and/or a reduced ability to produce eumelanin, which may explain why they fail to tan and are at risk from UVR.

We now report the presence of MC1R gene sequence variants in humans. These were found in over 80% of individuals with red hair and/or fair skin that tans poorly but in fewer than 20% of individuals with brown or black hair and less than 4% of those who showed good tanning response.

Valverde et al,. Variants of the melanocyte-stimulating hormone receptor gene are associated with red hair and fair skin in humans. Nat Genet 1995 Nov;11(3):328-30

There appears to be a significant genetic contribution to melanoma susceptibility. Pigmentation and the cutaneous response to ultraviolet irradiation, which also appear to be largely under genetic control, are major risk factors for melanoma. There seem to be at least two possible pathways in which the MC1R may be influencing melanoma development: through controlling the switch from pheomelanin to eumelanin and hence determining the ability to protect against ultraviolet radiation; or alternatively, via the effects of MSH (melanocyte stimulating hormone) on melanocyte growth.

Valverde et al,. The Asp84Glu variant of the melanocortin 1 receptor MC1R is associated with melanoma. Human Molecular Genetics, 1996, Vol. 5, No. 10 1663-1666

This study confirms the association of MC1R gene variants with the occurrence of cutaneous melanoma and nonmelanoma skin cancer. The presence of the MC1R gene variant Asp84Glu appeared to impose the highest risk for cutaneous melanoma with OR of 16.1 and was strongly associated with both fair skin and red hair. The exact mechanisms underlying the increased risk of individuals carrying MC1R gene variants to develop melanoma are not known. It is conceivable that the increased risk of cutaneous melanoma in individuals carrying the MC1R gene variants is modulated by the effects of complete or partial inactivation of the MC1 receptor, which may modulate the effects of MSH (melanocyte stimulating hormone) on proliferation and differentiation of different cells or by hampering the immune defense against genetically changed melanocytes. The study concluded "*the presence of MC1R gene variants appears to play an important part in the pathogenesis of cutaneous melanoma, although the exact mechanism underlying this increased risk remains to be determined.*"

Kennedy et al,. Melanocortin 1 Receptor (MC1R) gene Variants are Associated with an Increased Risk for Cutaneous Melanoma Which is Largely Independent of Skin Type and Hair Color. J Invest Dermatol 117:294-300, 2001

A tendency to sunburn and inability to tan after sun exposure are major risk factors for both melanoma and non-melanoma skin cancer. The MC1R gene is closely associated with variation in the skin's response to ultraviolet radiation in most of the population who do not have red hair. The results suggest that the MC1R gene is of substantial importance as a susceptibility gene for sunburn, photoageing and skin cancer. The study concluded "*We suggest that MC1R gene status therefore determines sun sensitivity in people without red hair*."

Healy et al,. Melanocortin-1-receptor gene and sun sensitivity in individuals without red hair. Lancet 355:1072-1073, 2000

A study measured skin color on the inner side of the forearm using a spectrophotometric instrument. Women carrying Arg151Cys, Asp294His, Arg160Trp and Asp84Glu variants had a significantly higher reflectance in the red region, which indicates a lower level of functional melanin. This association was the most pronounced for women carrying Asp84Glu. The study concluded "*Our findings support the hypothesis that MC1R polymorphisms do not necessarily alter the skin color but should sensitize the skin to UV-induced DNA damage.*"

Latreille eta al,. MC1R gene polymorphism affects skin color and phenotypic features related to sun sensitivity in a population of French adult women. Photochem Photobiol. 2009 Nov-Dec;85(6):1451-8. doi: 10.1111/j.1751-1097.2009.00594.x.

In a large case-control study, patients with melanoma and non-melanoma skin cancer and subjects without a history of skin cancer were studied. Carriers of one or two MC1R gene variants had a 3 and 11-fold increased risk of developing ephelides (freckles) whereas the risk of developing severe solar lentigines was increased 1.5 and 2 fold. These associations were independent of skin type and hair color, and were comparable in patients with and without a history of skin cancer. The population attributable risk - 60% of the ephelides (freckles) in the population was caused by MC1R gene variants. The study concluded "*As nearly all individuals with*

ephelides (freckles) were carriers of at least one MC1R gene variant, our data suggests that MC1R gene variants are necessary to develop ephelides."

Bastiaens et al,. The melanocortin-1receptor gene is the major freckle gene. Human Molecular Genetics, 2001, Vol. 10, No. 16 1701-1708

A study of the general Irish population found that 75% of people contained a variant in the MC1R gene, with 30% of people containing two variants. The Arg151Cys, Arg160Trp, and Asp294His variants were significantly associated with red hair. The same three variants were also over-represented in individuals with light skin type. The study concluded "*The results show that the Arg151Cys, Arg160Trp, and Asp294His variants are of key significance in determining the pigmentary phenotype and response to ultraviolet radiation, and suggest that in many cases the red-haired component and in some cases fair skin type are inherited as a Mendelian recessive*."

Smith et al,. Melanocortin 1 Receptor Variants in an Irish Population. J Invest Dermatol 111:119-122

A study investigating the relationship between MC1R genotype to CMM risk found there was a strong relationship between MC1R variants and hair color and skin type. MC1R variants were found in 72% of the individuals with CMM whereas only 56% of the control individuals carried at least one variant. Three active alleles (Arg151Cys, Arg160Trp, and Asp294His), previously associated with red hair, doubled CMM risk for each additional allele carries OR 2.0. All 71 redheads in this study carried at least one MC1R variant and 45 carried two. 66% of all individuals carrying two alleles from the set including Arg151Cys, Arg160Trp, and Asp294His were redheads, the frequency of redheads dropped to 8% for other variants. Not all whites share identical risk for skin cancers such as CMM, and better understanding of the genetic basis of UV-sensitive skin types will greatly enhance targeting of skin cancer-prevention campaigns. The study concluded "*We have shown that carrying particular MC1R variants increases CMM risk in individuals whose darker complexion would normally be considered protective. In light-skinned individuals, the association between CMM and MC1R variants disappears once skin and hair-color phenotype are taken into account*."

Palmer et al,. Melanocortin-1 Receptor Polymorphisms and Risk of Melanoma: Is te Association Explained solely by Pigmentation Phenotype? Am. J. Hum. Genet. 66:176-186, 2000

A study from Italy set out to verify the association of MC1R variants and BRAF mutated melanomas. The study reported "*Patients with MC1R variants had a high risk of carrying BRAF mutations in melanomas (odds ratio OR = 7.0) that increased with the number of MC1R variants and variants associated with red hair. MC1R carriers had a 5 to 15 fold increased risk of BARF-mutant melanomas based on carrying one or two variants and regardless of signs of chronic solar damage.*" The study concluded "*This confirms that in the Italian population, MC1R variants are strongly associated with BRAF-mutant melanomas independently of the degree of solar damage in the areas adjacent to the melanoma lesions*."

Fargnoli et al,. MC1R Variants Increase Risk of Melanomas Harboring BRAF Mutations. Journal of Investigative Dermatology (2008) 128, 2485-2490; doi:10.1038/jid.2008.67

Disseminated melanoma has a dismal prognosis and is almost completely resistant to therapeutic modalities such as chemotherapy and radiotherapy. The 2002 discovery of activating mutations in the serine/threonine kinase BRAF in approximately 50% of all melanomas kick-started a targeted therapy "arms race", which in less than 10 years, led to the U.S. Food and Drug Administration (FDA) approval of the BRAF inhibitor vemurafenib. V600E-mutated BRAF is a bona fide melanoma oncogene, with its introduction leading to the malignant transformation of immortalized human melanocytes both in vitro and in vivo. BRAF mutations are not ultraviolet (UV) radiation signature mutations. This review concluded "*If progress continues as we expect, a future can be envisaged in which rationally designed BRAF inhibitor-based drug combinations may be able to*

significantly extend the life span of patients with BRAF mutant melanoma." BRAF mutations are in over 50% of all melanomas and are not a result of UV exposure. New drugs have been developed to help treat BRAF melanoma.

Kudchadkar et al,. Targeting Mutant BRAF in Melanoma. Current Status and Future Development of Combination Therapy Strategies. Cancer J 2012;18: 124-131

BRAF mutations are not ultraviolet (UV) radiation signature mutations.

Kudchadkar et al,. Targeting Mutant BRAF in Melanoma. Current Status and Future Development of Combination Therapy Strategies. Cancer J 2012;18: 124-131

A genome-wide association study identified three loci associated with melanoma risk. It reported "Increased intermittent exposure to UV radiation, rather than chronic exposure, is thought to be responsible for the continued increase in incidence in many populations." The study identified MC1R and TYR variants which are associated with pigmentation, freckling and cutaneous sun sensitivity, well-recognized melanoma risk factors. The study concluded "*these genetic variants show notable homogeneity of effect across populations of European ancestry living at different latitudes and show independent association to disease risk*."

Bishop et al,. Genome-wide association study identifies three loci associated with melanoma risk. Nat Genet. 2009 Aug;41(8):920-5. doi: 10.1038/ng.411. Epub 2009 Jul 5.

Phase 2 of the Bishop study was published in 2012. It reported "*Cutaneous melanoma is predominantly a disease of fair-skinned individuals. Risk factors include a family history, certain pigmentation phenotypes (notably the presence of fair skin, blue or green eyes, blond or red hair, sun sensitivity or an inability to tan) and increased numbers of melanocytic nevi*." The study went on to say "*the major common genetic determinants of risk in the populations considered were MC1R locus (associated with red hair, freckling and sun sensitivity) tyrosinase (TYR) gene variants which code for skin color and a region near CDKN2A and MTAP which is associated with number of melanocytic nevi*."

Barrett et al,. Genome-wide association study identifies three loci associated with melanoma risk Nat Genet. : 43(11): 1108-1113. doi:10.1038/ng.959

Risk factors include a family history, phenotypes and increased numbers of melanocytic nevi, sun sensitivity or an inability to tan

Barrett et al,. Genome-wide association study identifies three loci associated with melanoma risk Nat Genet. : 43(11): 1108-1113. doi:10.1038/ng.959

A study examined the development of cutaneous nevi and melanoma. A high number of melanocytic nevi are the most important known risk factor for cutaneous melanoma. The study found two loci associated with nevus count and also show these are each associated with increased risk of melanoma. The study concluded "*About one subject in 11 is homozygous for the variant at both loci with twice the number of nevi compared to those*

homozygous for the protective alleles, and double the risk for melanoma. These data provide the first evidence for common melanoma alleles whose effects are mediated through nevus number."

Falchi et al,. Loci at 9p21 and 22q13 harbour alleles for development of cutaneous nevi and melanoma. Nat Genet. 2009 August; 41(8): 915-919. doi:10.1038/ng.410

A study looked at melanocytic nevi, nevus genes and melanoma risk in the UK. Twin studies have provided strong evidence that the number of nevi is predominantly genetically determined, with a smaller effect of sun exposure. The study reported "There was no convincing relationship between either average daily exposure or sunburn and nevus number." The analysis confirmed the strong relationship between nevus number and melanoma risk, with a crude odds ratio (OR) for melanoma of 10.02 when comparing the top quartile with the lowest quartile of nevus count. The study concluded "*This paper confirms the importance of nevi in melanoma pathogenesis and increases understanding of their genetic determinants*."

Newton-Bishop et al,. Melanocytic nevi, nevus genes and melanoma risk in a large case-control study in the United Kingdom. Cancer Epidemiol Biomarkers Prev. 2010 Aug;19(8):2043-54. doi: 10.1158/1055-9965.EPI-10-0233. Epub 2010 Jul 20

A study looked at melanoma risk for CDKN2A mutation carriers and compared Australia with the UK. The estimated HR for melanoma carriers relative to the general population decreased with regions of increasing ambient ultraviolet (UV) irradiance being higher for the UK – 87 than Australia – 31. The study concluded "*Contrary to the strong association between UV radiation exposure and melanoma risk for the general population, CDKN2A mutation carriers appear to have the same cumulative risk of melanoma irrespective of the ambient UV irradiance of the region in which they live.*"

Cust et al,. Melanoma risk for CDKN2A mutation carriers who are relatives of population-based case carriers in Australia and the UK. J Med Genet. 2011 Apr;48(4):266-72. doi: 10.1136/jmg.2010.086538

"*, CDKN2A mutation carriers appear to have the same cumulative risk of melanoma irrespective of the ambient UV irradiance of the region in which they live.*"

Cust et al,. Melanoma risk for CDKN2A mutation carriers who are relatives of population-based case carriers in Australia and the UK. J Med Genet. 2011 Apr;48(4):266-72. doi: 10.1136/jmg.2010.086538

A new study of the FTO gene which is associated with obesity, found an association with melanoma. The variants associated with melanoma are at one end of the gene (intron 8) and show no association with BMI. The genetic variants in FTO are inherited from your parents and are not caused by UV exposure. The increased melanoma risk is 16%. The study concluded "*In addition to identifying a new melanoma-susceptibility locus, this is to our knowledge the first study to identify and replicate an association with SNPs in FTO not related to body mass index (BMI)*."

Iles et al,. A variant in FTO shows association with melanoma risk not due to BMI. Nat Genet. 2013 Apr;45(4):428-32. doi: 10.1038/ng.2571. Epub 2013 Mar 3.

A study evaluating Gossypin and its application for melanoma treatment provided a clear indication of the extent of BRAF gene mutations found in melanoma. Previous research has reported that BRAF mutations are not caused by UV exposure.

BRAF gene (BRAFV600E) exists in nearly 70% of human melanomas.
Among the genes that have been associated with melanoma development, the serine/threonine kinase BRAF and cyclin-dependent kinases (CDK) are very important. Of all, 60% to 70% melanomas harbor BRAF missense mutations that result in the substitution of glutamic acid for valine at amino acid 600 (BRAFV600E; ref. 5). This causes destabilization of the inactive kinase conformation by switching the equilibrium toward active form rendering BRAFV600E 500 times more active than wild-type BRAF (6, 7). The constitutively active BRAFV600E can stimulate MEK–ERK pathway in cancer cells, and hence targeted inhibition of BRAFV600E is ideal for the treatment of melanoma.

Bhaskaran S et al,. Gossypin as a Novel Selective Dual Inhibitor of v-raf Murine Sarcoma Viral Oncogene Homolog B1 and Cyclin-Dependent Kinase 4 for Melanoma. Mol Cancer Ther; 12(4); 1-12. doi: 10.1158/1535-7163.MCT-12-0965

Environmental Risks other than UV Exposure as it relate to Melanoma

Wennborg – Solvents RR 2.7

A study aimed to elucidate cancer occurrence in relation to occupational exposure found elevated incidence for malignant melanoma among female laboratory employees for whom use was reported of solvents SIR 2.73 and of selected IARC Group 2B agents SIR 3.15. The study concluded "*In general, there were few cases of cancer in this comparatively young cohort, but the findings give some indication of increased risks for malignant melanoma in female laboratory personnel after exposure to organic solvents or substances classified by IARC as possibly carcinogenic.*"

Wennborg et al,. Cancer incidence and work place exposure among Swedish biomedical research personnel. Int Arch Occup Environ Health 2001 Oct;74(8):558-64.

Do factors other than UV radiation play a role in CMM? Richard Gallagher of BC Cancer, recently (2010) investigated the role of PCB's and cutaneous malignant melanoma (CMM). He found strong associations between risk of CMM and plasma levels of non-dioxin-like PCB's – OR 7.02 or a 700% increase in risk. He concluded that his study results *"suggest that environmental factors other than UV radiation may play a role in genesis of CMM, and indicate that it may be productive to search for further agents which might increase risk".*

Gallagher et al – Plasma levels of polychlorinated biphenyls and risk of cutaneous malignant melanoma: a preliminary study. Int. J. Cancer: 000, 000-000 (2010)

Risk of alcohol for Skin Cancer

Alcohol is also listed as a Group 1 carcinogen by the WHO IARC monographs. A large case-control study in 2004 found that consuming 2.8 liquor drinks per week was associated with a 69% increased risk in developing melanoma. This risk would be present for all people of drinking age and over, not just people under age 35, and represents over 11 times the increased risk of using a commercial tanning facilities (6%). It is notable that health organizations and government have not chosen to warn people to stop drinking alcohol because it is a Group 1 carcinogen or implemented warning signs at liquor retailers as a result of the increased risk.

Millen et al,. Diet and Melanoma in a case-control study. Cancer Epidemiology Biomarkers Prev 2004;13(6):1042-51

Tobacco and Sunbed Risk

Cigarette smoking increases one's risk of any cancer by 2,000% -- 333 times greater than the relative risk increase that the IARC data reports for commercial sunbed users. Any comparison between tobacco and commercial tanning is both unfair and misrepresentative and smoking should never be compared to UV exposure. The numbers do show the vast different in the increase risk for what is listed in Group 1. Unjustified comparisons dilute the importance of the anti-smoking message and add dangerous credence to the misinformed notion that "everything causes cancer these days".

Cigarette smoking increases one's risk of any cancer by 2,000% -- 333 times greater than the relative risk increase that the IARC reports for commercial sunbed users.

Base Tans vs Chemical Sunscreens

The actual value of a natural base tan is an SPF of 6 when following the exposure schedule of tanning equipment. This is based on research studies where people start off with non-burning exposures that are increased by a factor of 6 as their tan becomes darker and strengthens and prevents burning.

Miller et al,. Reduction of the UV burden it indoor tanners through new exposure schedules: a pilot study. Photodermatol Photoimmunol Photomed 2006; 22:59-66
Caswell et al,. The kinetics of the tanning response to tanning bed exposures. Photodermatol Photoimmunol Photomed 2000; 16:10-14

The FDA in the USA and Health Canada require sunbed manufacturers to attach a label to the sunbed outlining a recommended exposure schedule by skin type and by week using the "erythema reference action spectrum". This schedule shows that a base tan is protective or you wouldn't be able to increase the exposure time. As the tanner progresses through the weeks and their tan builds, they are allowed to tan longer in the sunbed. This schedule is based on increasing sunbed exposure from 100j/m2 to 625j/m2 in a typical 4 week exposure schedule.

Studies have shown that people rarely apply the amount of chemical sunscreen that the chemical sunscreen manufacturers use to determine an SPF rating, which is a covering of 2.0 mg/cm2. People under-apply sunscreen with an average application of between 0.25 to 0.50 mg/cm2. The result is the actual SPF value that the average person gets from under applying an SPF 30 would be an SPF value of 2.3.

Letter from Don Smith, Non-Ionizing Radiation Research Institute, 2/24/2011 – PHOTOPROTECTION COMPARISON SUNSCREEN vs NATURAL TAN

When you compare a chemical sunscreen with an SPF rating of 30, under applied by the public which provides an SPF rating of 2.3 to a natural fully developed base tan with an SPF rating of 6, the natural tan would provide almost three times the protection from burning than an SPF 30 chemical sunscreen. A natural tan never has to be re-applied when you sweat or swim, it's always there when you need it. A natural tan does not involve exposing your body to any harmful chemicals. A natural tan provides far better burn protection when compared objectively to an SPF 30 sunscreen.

This research is supported by a new peer reviewed and published research paper by Lazovich from 2012. In the study she states: *"Determination of the sun protection factor (SPF) of a sunscreen product is based on applying 2 mg/cm2, but individuals typically apply less than half this amount (25). Under these conditions, sunscreen with an SPF of 15 is reduced to about 5, the same level of protection as a suntan (26)."*
Lazovich et al., Time to get serious about skin cancer prevention. Cancer Epidemiol Biomarkers Prev DOI:10.1158/1055-9965 EPI-12-0327

25. Neale R, Williams G, Green A. Application patterns among participants randomized to daily sunscreen use in a skin cancer prevention trial. Arch Dermatol 2002;138:1319-25.
26. Schneider J. The teaspoon rule of applying sunscreen. Arch Dermatol 2002;138:838-9.

A study published in 2011 by Lazovich found *"Our data suggest ranking other sun protection methods, such as clothing or sun avoidance, higher than sunscreen for reducing melanoma risk."*
Lazovich, Vogel, Berwick, Weinstock, Warshaw, Anderson 2011. Melanoma Risk in Relation to Use of Sunscreen or Other Sun Protection Methods. Cancer Epidemiol Biomarkers Prev; 1–11. _2011 AACR

In summary, a base tan has a value up to an SPF 6 which is higher than the actual value for an SPF 30 which is applied by the average person at a rate that is thinner than how it's tested and provides a protection of an SPF 2.3. A Base Tan give a person 6 times the protection that someone without a tan would have. Consider the following:

1. A Base Tan does not wash off.
2. A Base Tan does not need to be reapplied every 2 hours.
3. A Base Tan is the natural way of protecting yourself called photoprotection.
4. A Base Tan is a multiplier for chemical sunscreen.
5. A Base Tan does not come with chemicals, like Oxybenzone, that can harm you.

Miller et al,. Reduction of the UV burden it indoor tanners through new exposure schedules: a pilot study. Photodermatol Photoimmunol Photomed 2006; 22:59-66
Caswell et al,. The kinetics of the tanning response to tanning bed exposures. Photodermatol Photoimmunol Photomed 2000; 16:10-14

Letter from Don Smith, Non-Ionizing Radiation Research Institute, 2/24/2011 – PHOTOPROTECTION COMPARISON SUNSCREEN vs NATURAL TAN

Dr R Mason in a 2010 research paper showed that UV exposure creates photoprotection by melanin production and by thickening of the top layer of skin. Vitamin D production is also a photoprotective factor. These three factors, melanin production (tan), thickening of the skin and vitamin D production does not happen when someone applies a sunscreen with a SPF of 8 correctly to their skin.

Mason RS, Sequeira VB, Dixon KM, Gordon-Thomson C, Pobre K, Dilley A, Mizwicki MT, Norman AW, Feldman D, Halliday GM, Reeve VE. Photoprotection by 1,25-dihydroxyvitamin D and analogs: Further studies on mechanisms and implications for UV-damage. Journal of Steroid & Molecular Biology 121 (2010) 164-168

Dr B Gilchrest, in an article in the May 2011 issue of Dermatology World which is put out by the America Academy of Dermatology, admits that a chemical sunscreen rated at SPF100 would give an actual rating of a sun protection Factor (SPF) of 5 to 7 based on what the average person applies.

http://www.aad.org/dermatology-world/monthly-archives/2011/may/lets-not-call-it-the-sunshine-vitamin#allpages

A Tan provides Photoprotection from skin cancer. A tan is just increased melanin in the skin. *"Melanoma occurs infrequently in type V-VI skin, suggesting that skin pigment plays a protective role [Fitzpatrick's Seventh Edition, 2008:1135]"*. Dark skinned people get melanoma on the bottoms of their feet and palms of the hand.

Genetic play a key role in skin cancer; Moles, Nevi, dysplastic nevus. *"Factors genetic, alone, unrelated entirely to sunlight, could be solely responsible for melanoma."*

Dr. A. Bernard Ackerman MD, book – The Sun and the Epidemic of Melanoma: Myth on Myth published in 2008, Second Edition, page 154

- Westerdahl 2000
- southern Sweden, greater percentage of skin type 1s vs Canada, plus exposure time was 30 minutes which was higher than in the use of Canadian equipment today
- Westerdahl did two studies from the same group of cases and controls, one on sunbed risk and one for sunscreen risk
- Westerdahl's Sunbed study found OR of 1.2 for "ever" use and 1.8 OR for regular sunbed use. In Westerdahl's Sunscreen study, with the same patients he found 1.3 OR for ever use and 1.8 OR for always use sunscreens.

Sunbeds and sunscreens had the same Odds Ratio – 1.8 for regular users. The use of sunbeds (home or commercial) and chemical sunscreens have the same increase risk of melanoma.

Westerdahl et al,. Risk of cutaneous malignant melanoma in relation to use of sunbeds: further evidence for UV-A carcinogenicity. British Journal of Cancer (2000) 82(9), 1593-1599

Definition of Photoprotection by Wilkipedia:

Photoprotection is a group of mechanisms that nature has developed to minimize the damage that the human body suffers when exposed to UV radiation. This damage mostly occurs in the skin, but the rest of the body can be affected by the oxidative stress caused by UV light.

Photoprotection of the human skin is achieved by extremely efficient internal conversion of DNA, proteins and melanin. Internal conversion is a photochemical process that converts the energy of the UV photon into small, harmless amounts of heat. If the energy of the UV photon were not transformed into heat, then it would lead to the generation of free radicals or other harmful reactive chemical species (e.g. singlet oxygen, or hydroxyl radical).

In DNA this photoprotective mechanism evolved four billion years ago at the dawn of life.[1] The purpose of this extremely efficient photoprotective mechanism is to prevent direct DNA damage and indirect DNA damage. The ultrafast internal conversion of DNA reduces the excited state lifetime of DNA to only a few femtoseconds (10−15s)—this way the excited DNA has not enough time to react with other molecules.

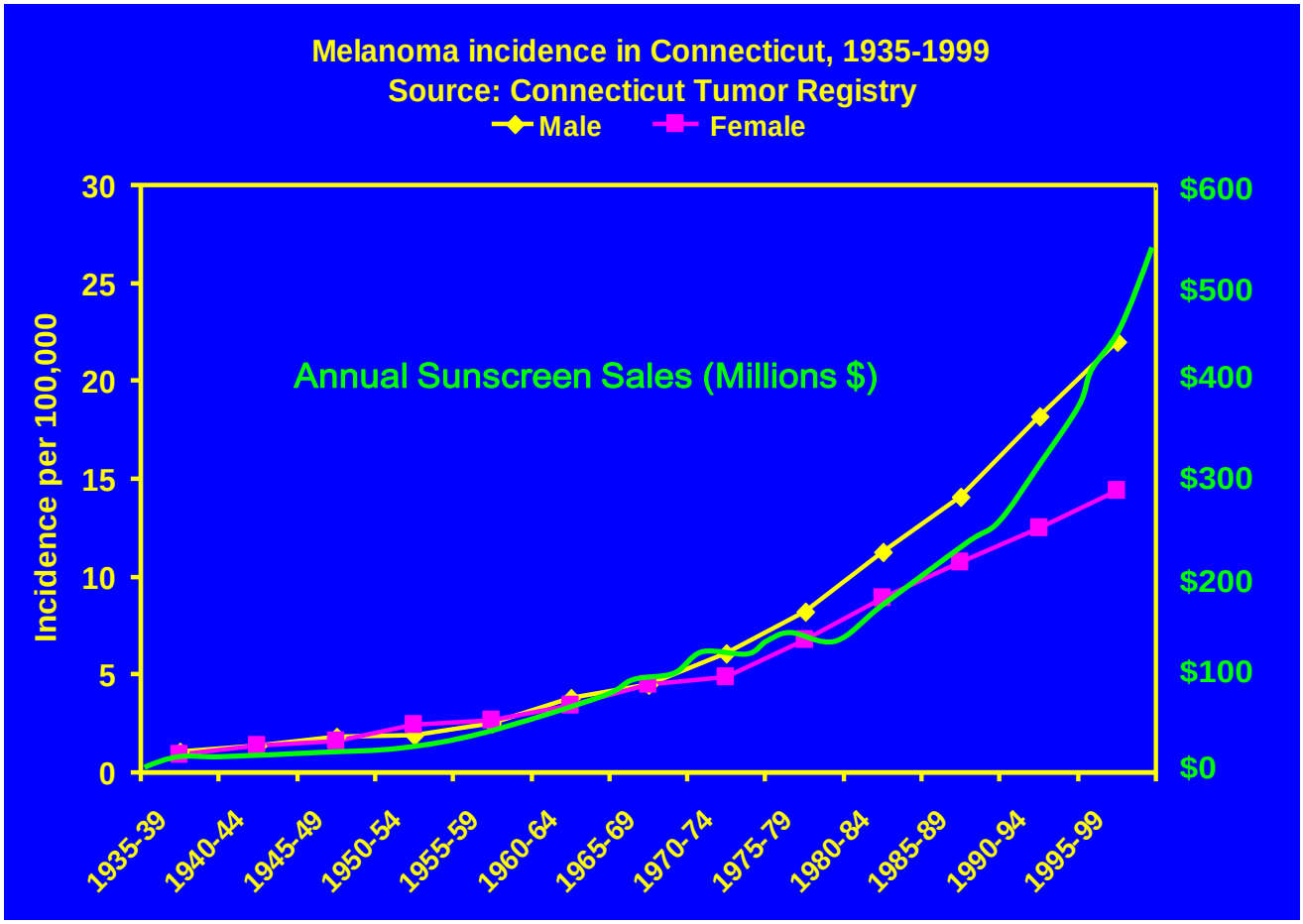
For melanin this mechanism has developed later in the course of evolution. Melanin is such an efficient photoprotective substance that it dissipates more than 99.9% of the absorbed UV radiation as heat. [2] This means that less than 0.1% of the excited melanin molecules will undergo harmful chemical reactions or produce free radicals

The cosmetic industry claims that the UV filter acts as an "artificial melanin". But those artificial substances used in sunscreens do not efficiently dissipate the energy of the UV photon as heat. Instead these substances have a very long excited state lifetime. [3]
In fact, the substances used in sunscreens are often uses as photosensitizer in chemical reactions. (see Benzophenone).

This discrepancy between melanin and sunscreen ingredients is one of the reasons for the increased melanoma risk that can be found in sunscreen users compared to non-users. (see sunscreen)

1. ^ "ultrafast internal conversion of DNA". Retrieved 2008-02-13.
2. ^ Meredith, Paul; Riesz, Jennifer (2004). "Radiative Relaxation Quantum Yields for Synthetic Eumelanin". *Photochemistry and photobiology* **79** (2): 211–216. doi:10.1562/0031-8655(2004)079<0211:RCRQYF>2.0.CO;2. ISSN 0031-8655. PMID 15068035.
3. ^ [a] [b] Cantrell, Ann; McGarvey, David J; (2001). "3(Sun Protection in Man)". *Comprehensive Series in Photosciences* **495**: 497–519. CAN 137:43484.

SUNSCREEN VS MELANOMA RATE IN THE USA



Edward D. Gorham, Ph.D., University of California San Diego

A study reviewing the results of sunscreen use reported that sunscreen use has been shown to prevent SCC but not BCC or melanoma. The study concluded "*Blanket advice to the public to wear sunscreens at any time outdoors is not at this time warranted. Instead, advice should focus on individual risk characteristics that are unequivocal, such as pigmentary phenotype, family history, and nevus type and number, and recommend*

avoidance of sun exposure by those who are clearly at high risk and reasonable enjoyment of outdoor activities with less anxiety by those who are clearly at reduced risk."

Berwick M. Counterpoint: Sunscreen Use Is a Safe and Effective Approach to Skin Cancer Prevention. Cancer Epidemiol Biomarkers Prev 2007;16(10):1923-4 doi:10.1158/1055-9965.EPI-07-0391

Damage to DNA and a Tan's Photoprotection

Much has also been made of the potential damage to DNA that can be caused by UV radiation, with suggestions that an existing tan gives the wearer a false sense of protection against the harmful effects of the sun.

On page 222 of the IARC (International Agency for Research on Cancer) Monographs Volume 55, section 5.4.2 it states *"In humans, pigmentation protects against erythema and histopathological changes. People with a poor ability to tan, who burn easily and have light eye and hair colour are at a higher risk of developing melanoma, basal-cell and squamous-cell carcinomas."* A tan is increased pigmentation. Therefore a tan protects against DNA damage.

http://monographs.iarc.fr/ENG/Monographs/vol55/index.php

On page 223 of the IARC Monographs Volume 55, section 5.4.5 it states *"Most of the DNA damage after a single exposure is repaired within 24 h."* If the damage is repaired naturally by the body in 24 h, it's hard to call that damage or a cancer risk.

http://monographs.iarc.fr/ENG/Monographs/vol55/index.php

Dr. Sam Shuster, Emeritus Professor of Dermatology, Newcastle University UK said this about a tan *"A suntan is an evolutionary device, it protects against burning. A suntan is just a sign of increased pigment, melanin, in the skin and is a natural biological response to the sun, not a sign of skin damage."*

http://www.dailymail.co.uk/health/article-1301722/The-melanoma-epidemic-Dont-panic--terrible-mistake.html

Yuji Yamaguchi in 2008 initiated a study to assess whether facultative pigmentation (tanning) induced by repeated UV irradiation is photoprotective. The study concluded *"These results suggest that pigmentation induced in skin by repeated UV irradiation protects against subsequent UV-induced DNA damage."* The report went on to say *"it may reflect development of a mature, efficient defense system."*

Yamaguchi Y. et al., Cyclobutane pyrimidine dimmer formation and p53 production in human skin after repeated UV irradiation Experimental Dermatology 2008; 17: 916-924

Professor Julia Newton-Bishop, an epidemiologist who led the research at Leeds University, UK, said *"it seems regular exposure helps the skin adapt and protect itself against the harmful affects of sunshine". "Increased levels of vitamin D made in the skin while exposed to sunlight may also be protective."* They found that those who spent between four to five hours in the sun each day over the weekend were less likely to develop tumours.

Newton-Bishop JA et al., Relationship between sun exposure and melanoma risk for tumours in different body sites in a large case-control study in a temperate climate, Eur J Cancer (2010), doi:10.1016/j.ejca.2010.10.008

"These results suggest that pigmentation induced in skin by repeated UV irradiation protects against subsequent UV-induced DNA damage."

Yamaguchi Y. et al., Cyclobutane pyrimidine dimmer formation and p53 production in human skin after repeated UV irradiation Experimental Dermatology 2008; 17: 916-924

This was confirmed by Rebecca Mason, University of Sydney, Australia. Their research in 2010 showed that *"like increased cornification and increased pigmentation, increased concentrations of Vitamin D compounds in skin act to protect against the next, rather than the initial UV Exposure."*

Mason, R et al, Photoprotection by 1,25-dihydroxyvitamin D and analogs: Further studies on mechanisms and implications for UV-damage Journal of Steroid Biochemistry & Molecular Biology 121 (2010) 164-168

In an interview Dr. Rebecca Mason from Australia stated Australian's are not getting enough sunlight exposure and this is increasing their risk of skin damage.
Professor Rebecca Mason at Sydney University's Bosch Institute for Medical Research says studies have found the Vitamin D-like compound can *"reduce DNA skin damage by 50 per cent and probably by more than 60-80 per cent".* And went on to say *"Many Australians do not realise that they don't have adequate vitamin D levels. A lot of people simply don't get outside much, or are not out at times of the day when sunlight can help make vitamin D, or don't have much skin exposed,"*
Dr Mason has been researching vitamin D for many years and in a 2010 research paper showed that vitamin D is the 3rd part of photoprotection from the sun.

http://www.adelaidenow.com.au/vitamin-d-like-compound-can-reduce-skin-damage-by-50-per-cent/story-e6frea6u-1226542342093
Mason RS, Sequeira VB, Dixon KM, Gordon-Thomson C, Pobre K, Dilley A, Mizwicki MT, Norman AW, Feldman D, Halliday GM, Reeve VE. Photoprotection by 1,25-dihydroxyvitamin D and analogs: Further studies on mechanisms and implications for UV-damage. Journal of Steroid & Molecular Biology 121 (2010) 164-168

Benefits of UV Exposure - Sunlight or UV Emitting Devices

To fully appreciate the possible risk of UV exposure one must also consider the enormous benefits of sunshine including vitamin D production.

In this national, population-based study of all incident lymphoid neoplasms diagnosed in Australia between 2002 and 2006; increasing incidence with increasing distance from the equator was observed for several types of non-Hodgkin and Hodgkin lymphoma. The study concluded "*Our findings support a possible protective effect of UVR exposure on the risk of several neoplasms, possibly through vitamin d-related immune modulation critical in lymphomagenesis*."

Van Leeuwen et al,. Latitude Gradients for Lymphoid Neoplasm Subtypes in Australia Support an Association with Ultraviolet Radiation Exposure. International Journal of Cacncer doi: 10.1002/ijc.28081

A study by Marianne Berwick in 2005 found that sunburn, high intermittent sun exposure, skin awareness histories and solar elastics were statistically inversely associated with death from melanoma. Patients with high solar elastosis had 60% better survival (HR of 0.40) than those without for melanoma deaths. This study highlights a dilemma. It is possible that cases of CM induced by intense UVR have a more benign course than cases of CM not induced by UVR. The study concluded that *"sun exposure is associated with increased survival from melanoma".*

Berwick et al. Sun exposure and mortality from melanoma. Journal of the National Cancer Institute, vol. 97, No. 3, February 2, 2005

A study evaluating the associations between sun exposure to MRI measures of brain injury in MS found that increased sun exposure was associated with increased whole brain volume and grey matter volume after adjusting for disability and vitamin D levels. The study concluded "*Sun exposure may have direct effects on MRI measures of neurodegeneration in MS, independently of vitamin D.*"

Zivadinov et al,. Interdependence and contributions of sun exposure and vitamin D to MRI measures in multiple sclerosis. J Neurol Neurosurg Psychiatry doi: 10.1136/jnnp-2012-304661

A letter published in 2008, supported that the beneficial effects of UV irradiance outweigh the risks. Solar UVB has always been the primary source for vitamin D for life on earth. It concluded: "*Solar UVB is the natural way to obtain vitamin D but, of course, care should be taken to limit irradiance in order to reduce the risk of adverse effects*."

Grant WB. Scientific and social controversies regarding UV and pigmentation: the beneficial effects of UV irradiance outweigh the risks. Pigment Cell Melanoma Res. 22; 137-138 doi: 10.1111/j.1755-148X2008.00523.x

Notably, UVR is one of few environmental exposures that may both cause and protect against disease. A study funded by the World Health Organization (WHO) set out to assess the overall disease burden attributable to ultraviolet radiation (UVR) at global and regional levels. The disease burden would be measured in DALYs – disability-adjusted life years consistent with WHO's global burden of disease studies. The burden of disease that might result from reduction of global UVR exposure to very low levels was estimated for the three vitamin D-deficiency bone diseases – rickets, osteomalacia and osteoporosis. The positive role vitamin D could play in reducing cancers, autoimmune diseases, cardiovascular diseases and infectious diseases was not considered due to insufficient evidence. The study reported "*UVR exposure is a minor contributor to the world's disease burden, causing an estimated annual lose of 1.6 million DALYs; i.e. 0.1% of the total global disease burden. A markedly larger annual disease burden, 3.3 billion DALYs, might result from reduction in global UVR exposure to very low levels.*" So if UVR exposure was reduced to very low levels the disease burden due to vitamin D insufficiency would be over 2000 times (1.6m to 3.3b) the current disease burden for UVR diseases such as skin cancers.

Lucas RM, McMichael AJ, Armstrong BK, Smith WT. Estimating the global disease burden due to ultraviolet radiation exposure. International Journal of Epidemiology 2008;1-14 doi:10.1093/ije/dyn017

A study published in Anti-Cancer Agents in Medical Chemistry in 2012 reviewed how much sunlight is appropriate to balance between the positive and negative effects of solar UV-exposure. Statements made in the research paper include:

- **Pale skin increases the risk of all types of skin cancer, while ability to tan lessens the risk, with decreases greatest in risk of squamous cell carcinoma followed by basal cell carcinoma and then melanoma**
- An increasing body of evidence now indicates that the vitamin D endocrine system is of relevance for carcinogenesis and progression of non-melanoma skin cancer and that vitamin D compounds may hold promise as effective agents for the prevention and treatment of these malignancies.
- It has been speculated that the beneficial (protective) effect of less intense solar radiation outweighs its negative (mutagenic) effect. In agreement with this assumption, some authors concluded that many lives could be prolonged through careful exposure to sunlight or possibly more safely, vitamin D supplementation, especially in non-summer months.
- **To summarize, it is important that recommendations of health campaigns on sun protection represent a balanced view of positive and negative effects of solar UV-exposure.**
- The important take home message for dermatologists and other clinicians is that health campaigns promoting strict sun protection procedures to prevent skin cancer may increase the substantial health risk of vitamin D-deficiency.

The study concluded "*If we follow the recommendations discussed above carefully, they will help to ensure an adequate vitamin D-status, thereby protecting us against adverse effects of strict solar UV-protection. Most importantly, these measures will protect us sufficiently against the multiple negative effects of vitamin D-deficiency on health without greatly increasing our risk of developing UV-radiation-induced skin cancer. To reach this goal it is important that health campaigns transfer this information to the general population, and to every clinician, especially dermatologists*."

Mason RS, Reichrath J. Sunlight Vitamin D and Skin Cancer. Anti-Cancer Agents in Medicinal Chemistry, 2013 Jan. 1;13(1):83-97

A study from Germany looked at vitamin D levels in patients with melanoma. We know that increased UVB exposure will increase your vitamin D level. Therefore you would expect that if increased UV exposure caused melanoma than these patients would have higher vitamin D levels. The study reported the opposite – "*we observed considerably decreased median 25(OH)D serum levels in patients with MM.*" In addition the study found "*that decreased 25(OH)D serum levels are associated with increased tumour thickness and advanced tumour stage*." The study concluded "*there is increasing evidence that patients with MM who strictly avoid sun exposure might benefit from 25(OH)D supplements that are sufficient to maintain serum levels above 30 ng/ml*."

Gambichler T, Bindsteiner M, Hoxtermann S, Kreuter A. Serum 25-hydroxyvitamin D serum levels in a large German cohort of patients with melanoma. Br J Dermatol 2013 Mar;168(3):625-8 doi: 10.1111/j.1365-2133.2012.11212.x

In a Harvard Health Family Health Guide publication, Dr. Robert Stern, chair of the Department of Dermatology at Harvard-affiliated Beth Israel Deaconess Medical Center reported on the benefits of moderate sun exposure. "*The relationship between sun exposure and skin cancer risk isn't as straightforward as you might think. Genes are a factor, of course: Some protect, some promote. So is skin type: People with pale skin who sunburn easily and don't tan are more likely to get sun-related skin cancer. As for exposure, the "dose" and its timing are crucial. Several studies have suggested that suddenly getting a lot of sun is more dangerous then steady exposure over time*." Dr. Stern went on to explain how UVB exposure to your skin does you some good. "*They kick off the chemical and metabolic chain reaction that produces vitamin D. Research shows that many people have low vitamin D levels.*"

Stern RS. Benefits of Moderate Sun Exposure. The Harvard Medical School Family Health Guide June 2004.
http://www.health.harvard.edu/fhg/updates/update0604d.shtml

"People with pale skin who sunburn easily and don't tan are more likely to get sun-related skin cancer."

Stern RS. Benefits of Moderate Sun Exposure. The Harvard Medical School Family Health Guide June 2004.
http://www.health.harvard.edu/fhg/updates/update0604d.shtml

In a 2 part study published in the Journal of the American Academy of Dermatology, researchers reviewed the possible role of vitamin D in the prevention of skin cancer. They report "*Mice lacking the vitamin D receptor develop increased numbers of nonmelanoma skin cancers, and the addition of vitamin D decreases the growth of nonmelanoma skin cancer and melanoma cells in vitro and in mouse models*." Potential mechanisms of action include inhibiting the hedgehog signaling pathway, the key pathway underlying development of basal

cell carcinoma (BCC) and upregulating DNA nucleotide excision repair enzymes. For SCC and melanoma the study reports "*the topical application of vitamin D appears to accelerate the clearance of cyclobutane dimmers that are characteristic of UV radiation-induced DNA damage. The antiproliferative and prodifferentiative effects of vitamin D and its metabolites have been shown in some, but not all, melanoma cell lines.*"

Tang JY, Fu T, Lau C, Oh DH, Bikle DD, Asgari MM. Vitamin D in cutaneous carcinogenesis Part I and II. J Am Acad Dermatol. 2012 Nov;67(5):817.e1-11; quiz 827-8. doi: 0.1016/j.jaad.2012.07.022.

In a prospective study, melanoma patients with high serum 25(OH)D levels at the time of melanoma diagnosis had thinner tumors, a lower risk of relapse, and a higher overall survival rate compared to those with low serum 25(OH)D levels. The study concluded "*Patients with melanoma, and those at high risk of melanoma, should seek to ensure vitamin D sufficiency*."

Newton-Bishop JA, et al,. Serum 25-Hydroxyvitamin D3 Levels Are Associated With Breslow Thickness at Presentation and Survival From Melanoma. J Clin Oncol. 2009 Nov 10;27(32):5439-44. doi: 10.1200/JCO.2009.22.1135. Epub 2009 Sep 21.

"Patients with melanoma, and those at high risk of melanoma, should seek to ensure vitamin d sufficiency."

Newton-Bishop JA, et al,. Serum 25-Hydroxyvitamin D3 Levels Are Associated With Breslow Thickness at Presentation and Survival From Melanoma. J Clin Oncol. 2009 Nov 10;27(32):5439-44. doi: 10.1200/JCO.2009.22.1135. Epub 2009 Sep 21

A recent review looked at the curious relationship between melanoma risk and sun exposure where sunburn is causal but occupational sun exposure is not. They found that vitamin D might also have a role in susceptibility to melanoma. This was due to the evidence that higher serum vitamin D levels may influence tumor cell proliferation. The study concluded "*The possible results of high vitamin D levels on the immune system remain unclear however and a source of some concern, but the data support the view that serum levels in the range 70-100 nmol/L might be a reasonable target for melanoma patients as much as for other members of the population*."

Field S, Newton-Bishop JA. Melanoma and vitamin D. Molecular Oncology (2011). doi:10.1016/j.molonc.2011.01.007

In a systematic review, researchers investigated evidence that the possible preventive effect of sunlight on cancer might be mediated not only by vitamin D but also other pathways. Thy found that for sustained vitamin D production chronic (continuous) sun exposure is probably more effective than intermittent bouts of intense exposure, particularly when it is considered that vitamin D production is self-limiting. The study concluded "*The evidence that chronic (not intermittent) sun exposure decreases the risk of colorectal, breast, prostate cancer and NHL is accumulating and gradually getting stronger. We therefore think that, particularly in countries with a moderate climate, intermittent sun exposure (and sunburn) should on the one hand be discouraged, because of skin cancer prevention, while on the other hand (moderate) chronic exposure possibly should be advised*."

Van der Rhee H, Coebergh JW, de Vries E. Is prevention of cancer by sun exposure more than just the effect of vitamin D ? A systematic review of epidemiological studies.Eur J Cancer. 2013 Apr;49(6):1422-36. doi: 10.1016/j.ejca.2012.11.001. Epub 2012 Dec 10

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In an early study of mice, UV dose was analyzed on tumor growth. Researchers found that that the average growth rate of the tumors is not dependent on the daily dose and that more UV energy has to be delivered to a mouse for the formation of tumors if a high daily dose is used. Stronger UV doses did not create additional tumors. They found that this was a protective reaction to the UV radiation. Thus, the animal becomes partially adapted to higher levels of UV exposure. The study concluded "*It seems as though an animal becomes more resistant to the UV-stimulus as the rate at which the stimulus is presented is increased: an adaptive phenomenon.*"

Gruijl FR, VanDer Meer JB, Van Der Leun JC. Dose-time Dependency of Tumor Formation by Chronic UV Exposure. Photochemistry and Photobiology Vol. 37, No. 1. pp.53-62, 1983

A study investigated the effect of whole body UVA exposure on the systemic blood circulation in humans. Many studies have suggested a linear rise in blood pressure at increasing distances from the equator. Similarly, blood pressure is higher in winter than summer. The study reported "*Immediately after UVA irradiation, as well as up to 60 minutes after the light stimulus, the values of systolic as well as diastolic blood pressure were reduced in all subjects as compared to control values determined before the irradiation procedure*." The study concluded "*UVA irradiation of human skin caused a significant drop in blood pressure even at moderate UVA does. The effects were attributed to UVA induced release of nitric oxide (NO) from cutaneous photolabile NO derivates*."

Oplander C et al,. Whole Body UVA Irradiation Lowers Systemic Blood Pressure by Release of Nitric Oxide From Intracutaneous Photolabile Nitric Oxide Derivates. Circ Res. 2009;105:1031-1040. doi: 10.1161/CIRCRESAHA.109.207019

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Humans evolved being exposed for about half of the day to the light of the sun. This study proposed that sunlight, independent of the benefits of melatonin, and vitamin D, acting on the skin, mobilizes nitric oxide (NO) stores which provide cardio-protective and anti-hypertensive effects. The study concluded "*Irrespective of the precise mechanism(s) of action, a modulation (e.g. by dietary measures) of the NO-related store in the skin and cautious bodily exposure to sunlight would seem to provide cardiovascular benefits. The future is bright – let a little sunshine into your heart*."

Feelisch M, Kolb-Bachofen V, Liu D, Lundberg JO, Revelo LP, Suschek CV, Weller RB. Is sunlight good for our heart? European Heart Journal (2010) 31, 1041-1045 doi:10.1093/eurheartj/ehq069

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Statements made on Cancer Research's UK website about exposure to UV light shows that not all UV exposure should be considered carcinogenic to humans. Climate in the UK is similar to Canada and this type of health message should be used in Canada.

"Most people in the UK only need to spend a short amount of time in the sun to make enough vitamin D. This is typically less than the time taken to lead to sunburn and a higher risk of skin cancer. It's possible to find a balance between enjoying the beneficial effects of the sun while not increasing the risk of skin cancer."

http://www.sunsmart.org.uk/skin-cancer-facts/howdoweknow/vitamind/

The proceedings from the 2nd CIE Expert Symposium on Lighting and Health reviewed the action spectrum for vitamin D synthesis and erythema. They found that vitamin D synthesis is initiated almost entirely by UVB wavelengths, while UVA contributes significantly to the erythemal effect. Short regular sub-erythemal exposures in the middle of the day would be best practice. Such advice is contrary to public health policies which recommend staying out of the sun in the middle of the day, and is thus a matter of some controversy. One of the conclusions drawn by the CIE "*There is a clear need to revisit public health policies on both sun exposure and vitamin D supplementation*."

Webb AR. Ultraviolet Benefits and Risks – The Evolving Debate. 2nd CIE Expert Symposium om "Lighting and Health". CIE x031:2006 UDC: 612.014.481-06 ISBN 3 901 906 55 X

CIE "*There is a clear need to revisit public health policies on both sun exposure and vitamin D supplementation*."

Webb AR. Ultraviolet Benefits and Risks – The Evolving Debate. 2nd CIE Expert Symposium om "Lighting and Health". CIE x031:2006 UDC: 612.014.481-06 ISBN 3 901 906 55 X

A study in Copenhagen, Denmark compared doses of natural UVR with artificial UVB irradiation and measured vitamin D serum levels. The study found that an artificial UVB exposure of 6 SEDs of hands and face induces significant 25(OH)D synthesis whereas an equivalent dose of solar UVR of hands and face does not. It concluded "*Artificial UVB was thus at least 8 times more efficient in increasing 25(OH)D than solar UVR at a UV-exposed area consisting of approximately hands and face*."

Datta P, Bogh MK, Olsen P, Eriksen P, Schmedes AV, Grage MML, Philipsen PA, Wulf HC. Increase in serum 25-hydroxyvitamin-D3 in humans after solare exposure under natural conditions compared to artificial UVB exposure of hands and face. Photochem. Photobiol. Sci., 2012, 11, 1817 doi: 10.1039/c2pp25093d

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A systematic review and meta-analysis of 151,978 MS patients compared MS risk to birth month. The study concluded "*Month of birth has a significant effect on subsequent MS risk. This is likely to be due to ultraviolet light exposure and maternal vitamin D levels, as demonstrated by the relationship between risk and latitude*."

Dobson R, Giovannoni G, Ramagopalan S. The month of birth effect in multiple sclerosis : systematic review, meta-analysis and effect of latitude. J Neurol Neurosurg Psychiatry. 2013 Apr;84(4):427-32. doi: 10.1136/jnnp-2012-303934. Epub 2012 Nov 14.

A European study investigating the effects of sunbed sessions and vitamin D supplements on colds found that three non-burning sunbed sessions weekly was more effective at raising vitamin D blood levels than 1,000 IU of daily vitamin D supplement. The study involved 105 young adults (18-30 years old) most of whom were female. The sunbed users rose from an insufficient level of 62 nmol/L to 109 nmol/L. The group taking 1000 IU of vitamin D supplement had levels 15% less than the sunbed users at 93 nmol/L. The control group dropped 7 nmol/L to 55 nmol/L over the course of the study in the winter. The study concluded "*The sub-sunburn sunbed treatment was effective in tanning and increasing the 25(OH)D serum level,..*."

Gruijl FR, Pavel S. The effects of a mid-winter 8-week course of sub-sunburn sunbed exposures on tanning, vitamin D status and colds. Photochem Photobiol Sci. 2012 Dec;11(12):1848-54. doi: 10.1039/c2pp25179e

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A study completed in 2011 in Sweden on MS cases found that subjects with low UVR exposure had a significantly increased risk of MS (+120%) compared with those who reported the highest exposure (OR 2.2). Similarly, subjects who had vitamin D levels less than 50 nmol/L had a 40% increased risk (OR 1.4). The study concluded "*UVR exposure may also exert a protective effect against developing MS via other pathways than those involving vitamin D*."

Baarnhielm M, Hedstrom AK, Kockum I, Sundqvist E, Gustafsson SA, Hillert J, Olsson T, Alfredsson L. Sunlight is associated with decreased multiple sclerosis risk: no interaction with human leukocyte antigen-DRB1*15. European Journal of Neurology 2012 doi: 10.1111/j.1468-1331.2011.03650.x

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UV Light and Vitamin D

It has also long been understood that our bodies produces vitamin D naturally through exposure of the skin to Ultraviolet B. The two sources of UVB available to Canadians are sunlight and sunbeds. Because of our Northern geography, sunlight does not produce vitamin D in humans from approximately October - March. The other way to get vitamin D is through diet or supplements. A typical Canadian diet provides very little vitamin D.

Tanners have been scientifically shown to have 90% higher vitamin D levels than non-tanners. A study in Alberta found that regular indoor tanners had the highest vitamin D levels compared to supplement users and people who got lots of sun exposure.

Tangpricha V et al. Tanning is associated with optimal vitamin D status (serum 25-hydroxyvitamin D concentration) and higher bone mineral density. Am J Clin Nutr 2004;80:1645–9

Schwalfenberg et al,. Addressing vitamin D deficiency in Canada: A public health innovation whose time has come. Public Health (2010), doi:10.1016/j.puhe.2010.03.003

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Sunbeds are a great source of Vitamin D. In a Court Affidavit, Dr. Reinhold Vieth, Mount Sinai Hospital, Toronto, foremost researcher on Vitamin D in Canada stated the following:

". . . sunbeds and summer sunshine are effective means by which to increase our serum 25(OH)D levels. The advantage of a tanning bed is that exposure to UV light can be controlled more precisely than casual sun exposure and thus can be safer than advising the public to guess at their own sun exposure from sunlight,"

The UVB in a tanning bed makes vitamin D through your skin the same as sunshine. Dr. M Holick demonstrated that the skin has a large capacity to produce cholecalciferol (vitamin D) and that whole-body exposure to one minimal erythermal dose of simulated solar ultraviolet radiation is comparable with taking an oral dose of between 10,000 and 25,000 IU of Vitamin D without the chance of toxicity.

Holick. Environmental factors that influence the cutaneous production of vitamin D. Am J Clin Nutr 1995;61(suppl):638S-45S

A tanning bed exposes both sides of your body at the same time making it highly efficient in producing vitamin D.

Vitamin D obtained through UV exposure to the skin poses no risk of toxicity as the body has a natural shut-off system for its production of Vitamin D. Health organizations sometimes state that taking a vitamin D supplement is "safer" than obtaining vitamin D from UV exposure. There is no published scientific evidence or references to support this statement.

Consider that the 2010 UK Vitamin D Consensus from 7 health organizations recommended:

"There is not enough evidence to support a recommendation for food fortification or widespread vitamin D supplementation for the general population. Unlike vitamin D produced in the skin, there is the potential that vitamin D from supplements and fortificants could build up to toxic levels and there is not enough evidence about the possible risks of raised vitamin D blood levels in the general population over a long period of time."

The 7 organizations were; the British Association of Dermatologists, Cancer Research UK, Diabetes UK, the Multiple Sclerosis Society, the National Heart Forum, the National Osteoporosis Society, and the Primary Care Dermatology Society.

They urged people to enjoy the sun safely and take care not to burn, helping to ensure the benefits of Vitamin D can be enjoyed without the risk of skin cancer being raised unnecessarily. They recommended 10-15 minutes exposure to the sun at midday during summer months without sunscreens.

http://info.cancerresearchuk.org/prod_consump/groups/cr_common/@nre/@sun/documents/generalcontent/cr_052628.pdf

A tanning bed on the other hand will generate over 10,000 IU of vitamin D per session. (Holick). Given that this is natural production, there is no risk of overdose or toxicity. According to research published by Edmonton's Dr. Gerry Schwalfenberg from subjects in Edmonton, Alberta, in the medical journal "Public Health", regular sunbed users had optimum vitamin D levels, even higher than those who take high-dosage vitamin D supplements.

Holick. Environmental factors that influence the cutaneous production of vitamin D. Am J Clin Nutr 1995;61(suppl):638S-45S
Schwalfenberg et al,. Addressing vitamin D deficiency in Canada: A public health innovation whose time has come. Public Health (2010), doi:10.1016/j.puhe.2010.03.003

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When reviewing the consequences of a particular action such as UV exposure, one must also look at the total results of that action. UV exposure produces Vitamin D in the skin. Vitamin D is heavily involved in repair mechanisms, fixing potential cancer causes before they evolve into a tumor.

For example:

Animal studies have shown that mice lacking the vitamin D receptor (VDR) are predisposed to developing skin tumors either from chemical carcinogens or from chronic UVR exposure. Studies suggest that vitamin D production and subsequent signaling through the VDR in the skin may have evolved in part as a protective mechanism against UVR induced epidermal cancer formation.

A study published in 2012 reviewed vitamin D signaling protection against UVB induced tumor formation. Researchers reviewed vitamin D's effect on keratinocyte proliferation and differentiation, the promotion of innate immunity and DNA damage repair. The study concluded "*thus, by these mechanisms one can conclude that the skin has evolved protective mechanisms against UVR induced carcinogenesis, and that these mechanisms involve vitamin D.*"

Bikle DD. Protective actions of vitamin D in UVB induced skin cancer. Photochem Photobiol Sci. 2012 Nov 19;11(12):1808-16. doi: 10.1039/c2pp25251a

A study published in 2012 found evidence indicating "*that vitamin D signaling protects the skin from cancer formation by controlling keratinocyte proliferation and differentiation, facilitating DNA repair, and suppressing activation of the hedgehog (Hh) pathway following UVR exposure*". Chemical sunscreens over an SPF of 8 block vitamin D production from UVB light. This would stop this protective measure initiated from vitamin D.

Bikle et al,. Protective role of vitamin D signaling in skin cancer formation. J Steroid Biochem Mol Biol. 2012 Oct 8. pii: S0960-0760(12)00187-2. doi: 10.1016/j.jsbmb.2012.09.021

A 2013 study from Australia examined the various signaling pathways involved in the vitamin D-induced protection of skin cells from UV. Past studies have shown that vitamin D receptor knock-out mice show increased susceptibility to photocarcinogenesis. Vitamin D protects skin and reduces DNA damage and skin carcinogenesis through increased p53 which facilitates DNA repair, a reduction in CPDs, a reduction in nitric oxide products, and the inhibiting of UV-induced immunosuppresion. The study concluded "*Taken together, the studies reviewed here indicate that the inhibition of UV-induced cell death by vitamin D compounds is indeed a protective effect.*"

Dixon et al,. Vitamin D and death by Sunshine. Int. J. Mol. Sci. 2013, 14, 1964-1977; doi:10.3390/ijms14011964

Most humans depend on sun exposure to satisfy their requirements for vitamin D. A study reviewing sunlight benefits stated "*Although chronic excessive exposure to sunlight increases the risk of nonmelanoma skin cancer, the avoidance of all direct sun exposure increases the risk of vitamin D deficiency, which can have serious consequences*."

Holick MF. Sunlight and vitamin D for bone health and prevention of autoimmune diseases, cancers, and cardiovascular disease. Am J Clin Nutr 2004;80(suppl):1678S-88S.

Sunlight may be related to cognitive function through vitamin D metabolism or circadian rhythm regulation. A study investigated whether ground and satellite measures of solar radiation are associated with cognitive decline. The study reported that after adjustment for covariates, the odds ratio of cognitive decline for solar

radiation exposure below the median vs above the median was 1.88 or an 88% increased risk. The study concluded "*We found that lower levels of solar radiation were associated with increased odds of incident cognitive impairment*."

Kent ST, Kabagambe EK, Wadley VG, Howard VJ, Crosson WL, Al-Hamdan MZ, Judd SE, Peace F, McClure LA. The relationship between long-term sunlight radiation and cognitive decline in the REGARDS cohort study. Int J Biometeorol. 2013 Jan 24 DOI: 10.1007/s00484-013-0631-5

Are Youth more vulnerable to UV Light

As to suggestions that individuals are more vulnerable to UV radiation before the age of 18, this is usually backed up by a reference study by Phillipe Autier in the European Journal of Cancer which states: "*Childhood and adolescence are periods of greater biological vulnerability to UV radiations, and thus prohibition of the use of tanning devices before 18 years old seems wise"* This study is not based on any hard scientific evidence. There is no data analyzing skin damage from people receiving enough UV to tan versus people receiving no UV exposure.

Autier also states: *"Sunburn experience during childhood or during adulthood is a risk factor for melanoma, and the risk increases with increasing numbers of sunburns"* but concedes that *"at present, there are no scientific data indicating that intentional exposure to UV radiations emitted by sunbeds is less harmful than intentional exposure to sunlight."* Essentially, Autier is arguing that sunlight and sunbeds are the same with presumably corresponding benefits and risks. Managed properly, sunbeds do not pose an inherently higher risk than natural sunlight.

Philippe Autier. Perspectives in melanoma prevention: the case of sunbeds. European Journal of Cancer 40 (2004) 2367-2376

In fact, two studies from Australia confirm the fact that melanoma for childhood and adolescents comes from genetic factors, not cumulative UV exposure.

Queensland, Australia has the highest incidence rates of childhood melanoma in the world. A study of risk factors for childhood melanoma (children less than 15 years) found that the strongest determinants were constitutional factors including the presence of more than 10 naevi greater than 5 mm RR 9.9, heavy facial freckling RR 6.4, an inability to tan on exposure to the sun RR 8.8, and a family history of melanoma RR 4.2. The study found: *"No measures of acute or chronic exposure to solar UV radiation were associated with childhood melanoma in our study."*

"No measures of acute or chronic exposure to solar UV radiation were associated with childhood melanoma in our study."

Whiteman et al,. Risk factors for childhood melanoma in Queensland, Australia. Int. J. Cancer: 70, 26-31 (1997)

Whiteman et al,. Risk factors for childhood melanoma in Queensland, Australia. Int. J. Cancer: 70, 26-31 (1997)

A study from Australia looking at melanoma risk in adolescents (15-19 years) confirmed the Whiteman study results and found that *"the strongest risk factor associated with melanoma in adolescents in a multivariate model was the presence of more than 100 nevi, 2 mm or more in diameter – Odds Ratio = 46.5." "Other risk*

factors were red hair OR 5.4, blue eyes OR 4.5, inability to tan after prolonged sun exposure OR 4.7, heavy facial freckling OR 3.2 and family history of melanoma OR 4.0." There was no association with sunscreen use overall and no difference between cases and controls in cumulative sun exposure in Australia's high exposure environment. The study *concluded "Lack of association with reported sun exposure is consistent with the high genetic susceptibility in this group".*

"the strongest risk factor associated with melanoma in adolescents in a multivariate model was the presence of more than 100 nevi, 2 mm or more in diameter – Odds Ratio = 46.5."

Youl et al,. Melanoma in adolescents: A case-control study of risk factors in Queensland, Australia. Int. J. Cancer: 98, 92-98 (2002) DOI 10.1002/ijc.10117

Youl et al,. Melanoma in adolescents: A case-control study of risk factors in Queensland, Australia. Int. J. Cancer: 98, 92-98 (2002) DOI 10.1002/ijc.10117

A recent study in the US reports that sunbed use actually lowers the melanoma risk. A large case-control study conducted in the US and completed in 2011 looked at sunbeds and sunlamps and their risk of melanoma. They found for females, use before age 20 yr, current use and years of use were not significant after adjustments. The estimated relative odds of melanoma was 0.8 for occasional users (<10 sessions) and 1.1 for more frequent users (10+ sessions). For males, the melanoma risk from sunbeds was 0.90 with no significant difference between occasional and frequent users.

Fears et al,. Sunbeds and sunlamps: who used them and their risk for melanoma. Pigment Cell Melanoma RES. doi: 10,1111/j.1755-148X.2011.00842.x

A large-scale case-control study in Western Canada found *"in terms of sunburn history in childhood, this suggests that rather than the occurrence of sunburn itself increasing the risk of melanoma, the risk is due to the characteristics of pigmentation associated with poor sun tolerance."* According to Elwood et al., sunburn is not a likely independent risk factor for melanoma, but more likely an associated event that seems to be determined by some constitutional factors, such as degree of pigmentation, capability for tanning, inclination to burning (the less epidermal melanin, the less ability to tan), and greater propensity for burning, with all of these being factors which seem to put a person at increased risk for developing melanoma.

Elwood et al., Pigmentation and skin reaction to sun as risk factors for cutaneous melanoma: western Canada Melanoma Study. Br Med J (Clin Res Ed). 1984 Jan 14;288(6411):99-102

"The lowest risk was in subjects with a moderate or deep tan in both summer and winter".

Elwood et al,. Sunburn, suntan and the risk of cutaneous malignant melanoma – The Western Canada Melanoma Study. Br. J. Cancer (1985), 51, 543-549

It has also been asserted that most people get more than half of their lifetime sun exposure before the age of 18.

Dadlani, C., & Orlow, S. J. (2008). Planning for a brighter future: A review of sun protection and barriers to behavioral change in children and adolescents. Dermatology Online Journal, 14(9), 1.

A number of studies suggest otherwise. The Dadlini study actually said 25-50 percent of a person's lifetime sun exposure is said to occur before 18-21 years of age. It was referenced to two studies by Godar. Most health organizations take the most damaging quote and published the highest number available.

In fact, Godar reported that only about 23 percent of lifetime exposure occurs by age 18. This research was also the reason why Health Canada removed their statement from its website with respect to UV exposure before the age of 18, as did medical associations around the world

Godar DE, Urbach F, Gasparro FP, Van der Leun JC. UV doses of young adults. Photochem Photobiol 2003; 77(4):453-457

A study of childhood and adolescent melanoma in the United States from 1973-2009 had some baffling results. Overall, pediatric melanoma increased by an average of 2% per year. But this increase was solely from the low UVB exposure area or the North part of the USA. The study reported "*there was an unexpected increased trend in low UV-B exposure registries, with a negative trend in high UV-B registries for 15-19 year olds starting in 1985*." The study went on to say "*recent data has indicated stable UV measurements since the 1990's. This is consistent with our results suggesting UV-B exposure is not the primary factor in increased melanoma incidence.*"

Wong JR, Harris JK, Rodriguez-Galindo C, Johnson KJ. Incidence of Childhood and Adolescent Melanoma in the United States: 1973-2009. Pediatrics. 2013 Apr 15. DOI: 10.1542/peds.2012-2520

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Wong JR, Harris JK, Rodriguez-Galindo C, Johnson KJ. Incidence of Childhood and Adolescent Melanoma in the United States: 1973-2009. Pediatrics. 2013 Apr 15. DOI: 10.1542/peds.2012-2520

A study from the National Cancer Institute, U.S. Department of Health and Human Services published in 2007 studied cancer in young adults age 15-29 and found that melanoma was the 2nd most common type of cancer in this age group. It went on to say *"the etiology of melanoma in 15-29 year old individuals is not known. Solar/ultraviolet irradiation does not appear to be as important a causative factor in this age group as it is in older individuals"*. It concluded *"most of the melanomas that occur in young persons arise in dysplastic nevi or in parts of the body that are likely to have been protected from ultraviolet light exposure"*.

Bleyer A, O'Leary M, Barr R, Ries LAG (eds): Cancer Epidemiology in Older Adolescents and Young Adults 15 to 29 Years of Age, Including SEER Incidence and Survival: 1975-2000. Chapter 5. National Cancer Institute, NIH Pub. No. 06-5767. Bethesda, MD 2006.

Melanoma Risk in Young

A Canadian study that looked at melanoma incidence from 1956 to 2005 concluded "*The rates of CMM are slowing; however, this change is confined to younger individuals".* The report went on to say that *"Familial melanoma, which accounts for 10% of all melanoma, is characterized by early onset (typically <40 years). It is likely that the CMMs observed for those younger than 40 years are largely accounted for by this group of people."*

Pruthi et al,. Incidence and anatomic presentation of cutaneous malignant melanoma in central Canada during a 50-year period: 1956 to 2005. J Am. Acad. Dermatol., 2009, 61, 44-50

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A study from Australia looking at melanoma risk in adolescents (15-19 years) found that *"the strongest risk factor associated with melanoma in adolescents in a multivariate model was the presence of more than 100 nevi, 2 mm or more in diameter – Odds Ratio = 46.5." "Other risk factors were red hair OR 5.4, blue eyes OR 4.5, inability to tan after prolonged sun exposure OR 4.7, heavy facial freckling OR 3.2 and family history of melanoma OR 4.0."* There was no association with sunscreen use overall and no difference between cases and controls in cumulative sun exposure in Australia's high exposure environment. The study *concluded "Lack of association with reported sun exposure is consistent with the high genetic susceptibility in this group".*

Youl et al,. Melanoma in adolescents: A case-control study of risk factors in Queensland, Australia. Int. J. Cancer: 98, 92-98 (2002) DOI 10.1002/ijc.10117

An outdoor work and skin cancer study from Bavaria concluded "*CMM risk was not significantly associated with outdoor work.*"

Radespiel-Troger M, Meyer M, Pfahlberg A, Lausen B, Uter W, Gefeller O. Outdoor work and skin cancer incidence: a registry-based study in Bavaria. Int Arch Occup environ Health (2009) 82:357-363 doi: 10.1007/s00420-008-0342-0

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Whiteman et al,. Risk factors for childhood melanoma in Queensland, Australia. Int. J. Cancer: 70, 26-31 (1997)

This evidence was further supported in a 2011 study by Faye Elliott, University of Leeds, UK. Elliott studied the relationship between sunbed use and melanoma risk in a large case-control study in the United Kingdom. They found no evidence for sunbed use as a risk factor for melanoma in the UK – OR 1.06. They also stated *"Age at first use of sunbeds showed a small non-significant increased risk for use < 25 years – OR 1.16"* [14].

[14] Elliott F, et al, Relationship between sunbed use and melanoma risk in a large case-control study in the United Kingdom. Int. J. Cancer: 000, 000-000 (2011)

If melanoma and skin cancer is actually increasing it would be hard to blame UV exposure since we all know that public UV exposure overall is reducing. People and children are spending more time indoors than ever before. We work indoors and play indoors. And when people are outside they are covered in chemical sunscreen.

Does using a sunbed actually increase the melanoma risk of younger users? A recent study in the US reports that it actually lowers the melanoma risk. A large case-control study completed in 2011 in the US looked at sunbeds and sunlamps and their risk of melanoma. They found for females, use before age 20 yr, current use

and years of use were not significant after adjustments. The estimated relative odds of melanoma was 0.8 for occasional users (<10 sessions) and 1.1 for more frequent users (10+ sessions). For males the melanoma risk from sunbeds was 0.90 with no significant difference between occasional and frequent users.

Fears et al,. Sunbeds and sunlamps: who used them and their risk for melanoma. Pigment Cell Melanoma RES. doi: 10,1111/j.1755-148X.2011.00842.x

Younger sunbed users need the guidance of a trained operator.

A study of 1252 students aged 14-19 in Stockholm revealed that of all sunbed users, 44% reported erythema.

Boldeman et al,. Sunbed use in relation to phenotype, erythema, sunscreen use and skin diseases. A questionnaire survey among Swedish adolescents. Br J Dermatol 1996 Nov;135(5):712-6

Almost all equipment is self-serve in Sweden, to reduce this type of risk, all is required is someone, trained and industry certified to control the equipment. The highest risk factor for skin cancer is the number of sunburns over a lifetime, with intermittent exposure which normally leads to overexposure in the sun being next highest risk.

The Canadian Dermatology Association (CDA) Risk on UV exposure and Melanoma

The Canadian Dermatology Association (CDA) on their website agrees that excessive UV exposure, sunburns and genetic factors are the primary risk factors for melanoma. They fail to mention UV exposure at a young age as a risk factor for melanoma. Here is what the CDA report:

Excessive exposure to ultraviolet (UV) from the sun and sunbeds plays a leading role in the development of melanoma and is the most preventable cause of this disease. Experts estimate about 90% of melanomas are associated with severe UV exposure and sunburns over a lifetime.

The risk factors for melanoma according to the CDA:

- Fair, sun-sensitive skin that burns rather than tans; freckles; red or blonde hair.
- Many moles – more than 50.
- Moles which are large or unusual in colour or shape.
- A close family history of melanoma or a personal history of melanoma.
- Had excessive exposure to UV from the sun or sunbeds.
- A history of severe sunburns

Therefore the risk of the use of tanning equipment starts with genetics, skin type and number of moles. The CDA quote that 90% of melanoma is associated with UV exposure is incorrect given that BRAF mutations, which are not induced by UV, are found in 50+% of all melanoma. This is why any warning about the use of tanning equipment should identify genetic/skin type risk.
http://www.dermatology.ca/skin-hair-nails/skin/skin-cancer/malignant-melanoma/

The National Cancer Institute

Intermittent sun exposure seems to be the most important risk factor for melanoma. Intermittent sun exposure is, by definition, sporadic, and is commonly associated with recreational activities, particularly among indoor workers who use weekend or vacation time to be outdoors and whose skin has not adapted to the sun. Chronic

sun exposure is incurred by consistent, repetitive sun exposure, usually during outdoor work or more extensive recreational activities. Chronic sun exposure, as observed in those occupationally exposed to sunlight, is either protective or without increased risk for the development of melanoma.

http://www.cancer.gov/cancertopics/pdq/genetics/skin/HealthProfessional/page4

Go to this link. There is more for other causes of melanoma.

Canadian Cancer Society (CCS) Survey

A study commissioned by the Canadian Cancer Society (CCS) in Ontario showed that 60% of tanning facilities didn't ask the age of tanners or assessed their type of skin for the possibility of burning, and 99% didn't advise **those with a probability of burning** not to tan.

First, there is no law that requires a tanning facility to ask the age of tanners or to skin type clients. It's a recommendation by Health Canada and also a JCTA guideline. Our organization supports the implementation of professional standards which would require such disclosure

According to the Youthography survey, the researchers visited the salons but never went through and actually signed up for a tanning session. Salon's normally discuss pricing first, then there is a purchase at which time a client card is filled out, which includes skin typing and age. The results would have been very different if the researchers had actually paid for a tan and gone into the tanning room.

Cancer Risk Factors in Ontario – 2013 Report

A new report issued in March 2013 by Cancer Care Ontario, called Cancer Risk Factors in Ontario, provided a summary of the epidemiologic evidence for a wider range of cancer risk factors. The majority of these risk factors have a higher risk than sunbeds and yet there is no warning sign for the public.

Some of the key findings from cancer Risk Factors in Ontario:

- tobacco - 9 times (900% increase) risk for lung cancer
- alcohol - 75% increase in oral cavity cancers, 20 - 30% increased risk for colon, breast,
- red meat, processed meat 17 - 18% increase risk of colon cancer - same as tanning beds and eaten by all ages! NO WARNING LABEL REQUIRED for this type of product
- body fatness - 13-55% increased risk of various cancers
- Oral contraceptives - 57% increased risk liver cancer, 24% breast, 65% cervical
- Ultraviolet Radiation – Sunshine – Melanoma – intermittent exposure 61% risk, chronic exposure 5% reduced risk.
- Indoor Tanning Devices – Melanoma – intermittent exposure 15-22% . Sunbeds are three to four times LESS risk than outdoor intermittent sun exposure

- XRay - medical - brain cancer 4.6 times risk, thyroid 12 times risk
- Asbestos - 5 times risk for lung cancer
- wood dust 2-50 times risk for sinonasal cancer

- Arsenic - 2 times risk for skin cancer (SCC)
- Mineral Oils - lightly processed - 20% higher risk of melanoma skin cancer. Studies have seen up to a 2 times risk for mineral oil metal working fluids. NO WARNING FOR SKIN CANCER HERE
- Polycyclic aromatic hydrocarbons - motor vehicle exhaust, industrial emissions, roadway paving, roofing, chimney sweeps - 1.2 -2.3 increased risk of lung cancer for road pavers, roofers and chimney sweeps
- under medical conditions and treatments - methoxsalen + UVA (Phototherapy) – SCC skin cancer - 5-10 times greater risk than general population. OUT DATED WARING LABEL FROM 1989

			Magnitude of risk*			
Risk factor/exposure	Cancer	Direction of association	Total exposure	Intermittent exposure	Chronic exposure	Strength of evidence[a]
Solar ultraviolet (UV) radiation	Skin (melanoma)	↑	1.34[b]	1.61[b]	0.95[b]	Sufficient
	Skin (BCC)	↑	0.98[c]	1.38[c]	1.19[c]	
	Skin (SCC)	↑	1.53[c]	0.91[c]	1.64[c]	
	Lip	↑	…	…	…	Limited
	Eye	↑	…	…	…	
UV-emitting indoor tanning devices	Skin (melanoma)	↑		1.15–1.22[d]		Sufficient
	Eye	↑		1.30–3.40[a]		
	Skin (SCC)	↑		…		Limited

Abbreviations: UV= ultraviolet; BCC= basal cell carcinoma; SCC=squamous cell carcinoma

Sources: [a]IARC, 2012; [b]Gandini S, 2005; [c]Armstrong, 2001; [d]International Agency for Research on Cancer Working Group on artificial ultraviolet (UV) light and skin cancer, 2006; [e]Hirst, 2009

*Relative risk (RR) estimate for: highest exposure category to lowest for estimates of solar ultraviolet radiation; ever vs. never-use of UV-emitting tanning devices.
..Magnitude of risk not shown in table if strength of evidence is "probable" or "limited."

Cancer Care Ontario. Cancer Risk Factors in Ontario: Evidence Summary. Toronto, Canada, 2013. This report is available at www.cancercare.on.ca/riskfactor

Research on the Misinformation the Public receives on UV Light and Skin Cancer

In 2003 Dr. B. L. Diffey published a paper that concluded "*Sunbed use could be regarded as a relatively minor self-imposed detriment to public health compared with other voluntary pleasurable activities associated with significant mortality, such as smoking and drinking alcohol. While cosmetic tanning using sunbeds should be discouraged, prohibition is not warranted especially as exposure to the sun, which cannot be regulated, remains the major contributory factor to the risk of melanoma.*"
Diffey – A quantitative estimate of melanoma mortality from ultraviolet A sunbed use in the U.K. British Journal of Dermatology 2003; 149: 578-581

In a guest editorial in Skin & Allergy News, a professor of dermatology questioned the benefit of public education messages endorsed by dermatologists that sun exposure is the major cause of melanoma. Dr. Rhodes stated "*melanoma is a heterogeneous disease with multiple causes, arising from potential precursor moles that have little or nothing to do with sun exposure, including dysplastic nevi, congenital nevi, and abnormal moles on acral surfaces and mucous membranes*." "*We really do not know what portion of melanomas can be prevented by sun avoidance.*" Dr. Rhodes concluded "*The public deserves more focused, effective, and accurate messages about melanoma and melanoma risk factors*."

Rhodes, A. Melanoma's Public Message. Skin & Allergy News 2003;34 (4): 1-4

An early study (1987) of risk factors for cutaneous melanoma found that in 18% to 85% of cases, melanoma of the skin develops in a preexisting pigmented mole. The study reported "*The most important melanoma risk factors (in decreasing order of importance) for a given individual are as follows: a persistently changed or changing mole, adulthood, irregular varieties of pigmented lesions, a congenital mole, Caucasian race, a previous cutaneous melanoma, a family history of cutaneous melanoma, immunosuppresion, sun sensitivity and excessive sun exposure.*"

Rhodes et al,. Risk Factors or Cutaneous Melanoma. JAMA 1987 258:3146-3154

A study by professor of Dermatology Arthur Rhodes, published in 2006, discussed intervention strategies for cutaneous melanoma and the role of UV.

The magnitude of the role of UVR in the initiation and/or promotion of CM has been a contentious issue for at least 50 years. There are convincing arguments for a relationship between some cases of CM and UVR in humans, suggested by biologic experiments of nature and therapeutic misadventure. "*Not all recent epidemiologic studies support a convincing relationship between CM and UVR."*

Although UVR appears to have a role in the development of CM and even nevomelanocytic nevi, physicians and the lay public must understand that UVR is not the only pathway to the development of this tumor. Although CM may appear on chronically sun-exposed and intermittently sun-exposed sites, CM also develops in relatively sun-protected sites, i.e., acral surfaces, nail matrix, hairy scalp, and double-covered areas. Whereas at least some cases of CM appear to be directly related to UVR exposure, the strength of association of chronic and intermittent UVR exposure in the overall epidemiology of CM is complex, confusing, and often weak at best. Chronic UVR exposure and other measures of UVR exposure appear to be inversely associated with CM risk and even CM mortality in some studies.

"Public messages related to UVR avoidance and protection as an intervention strategy to prevent CM have been based on circumstantial data and inference rather than clinical trials and sound scientific data." the research want on to *say "What has not been emphasized to the public is the relative weakness of the association compared to other known risk factors."*

Other than lentigo maligna melanoma, which constitutes an average of 7% of CM cases and which usually occurs on sun-damaged skin of elderly White adults, we have little notion about the true proportion of CM cases directly attributable to UVR. CM has divergent pathways of origin. Some cases of CM are related to identifiable melanocytic precursors that have a heritable basis, on UVR-exposed and UVR-protected sites, whereas other cases of CM are probably directly related to UVR exposure in predisposed individuals.

"Unfortunately, as a result of public messages that emphasize the role of ultraviolet radiation (UVR) exposure in tumor development, most general physicians and lay people believe that most if not all cases of CM are the direct result of UVR exposure. In fact, we do not know the case fraction of CM directly attributable to UVR, and the unintended consequences of current messages directly linking UVR exposure and CM development may be thwarting the primary intervention goal of reducing tumor-related mortality."

Rhodes AR. Cutaneous melanoma and intervention strategies to reduce tumor-related mortality: what we know, what we don't know, and what we think we know that isn't so. Dermatologic Therapy, Vol. 19, 2006, 50-69

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Rhodes AR. Cutaneous melanoma and intervention strategies to reduce tumor-related mortality: what we know, what we don't know, and what we think we know that isn't so. Dermatologic Therapy, Vol. 19, 2006, 50-69

An early study reviewed the risk factors for melanoma and reported that cutaneous melanoma usually appears as a changing or unusual-appearing mole. In their study, 81% of cases had observed a significant and persistent change in the presenting lesion with respect to colour, size, borders or surface and/or reported that the lesion had a history of bleeding, itching or pain and this represented a relative risk greater than 400. The study recommended "*the general public must be made acutely aware of the need for immediate evaluation of a "persistently changed or changing" "ugly", or "unusual" mole.*"

Rhodes AR, Weinstock MA, Fitzpatrick TB, Mihm MC, Sober AJ. Risk Factors for Cutaneous Melanoma. JAMA 1987 258:3146-3154

Nelemans et al 1995 – "*If there is a relationship between sunlight exposure and melanoma risk, it is not a straightforward one. Melanoma risk does not simply increase with an increasing amount of accumulated exposure to ultraviolet radiation. This is illustrated by the fact that the incidence of melanoma is higher among indoor than among outdoor workers and that melanoma does not predominantly occur on body sites that are most frequently exposed to the sun*." The study found that the pooled odds ratio for intermittent sunlight exposure was 1.57 and for chronic exposure was a 27% decreased risk of melanoma (OR 0.73). This proves that chronic UV exposure protects against melanoma risk.

Nelemans PJ, Rampen FHJ, Ruiter DJ, Verbeek ALM. An addition to the controversy on sunlight exposure and melanoma risk: A meta-analytical approach. J Clin Epidemiol Vol. 48, No. 11, pp. 1331-1342, 1995

"*If there is a relationship between sunlight exposure and melanoma risk, it is not a straightforward one. Melanoma risk does not simply increase with an increasing amount of accumulated exposure to ultraviolet radiation."*

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An early case-control study analyzed susceptible subgroups and melanoma risk when exposed to sunshine. "*Subjects with little or no ability to tan exhibited substantially higher relative risks than good tanners for qualitative measures of occupational, recreational and overall sun exposure and for a history of severe sunburn with blistering. For good tanners, moderate exposure was protective against melanoma, whereas much exposure resulted in increased risk, but not to as high a level as for poor tanners. One can speculate that tanning may confer a shielding effect on the skin and that moderate sun exposure may actually protect against melanoma by promoting a tan in individuals who do so easily or in some other unknown way*." In this study, the odds ratio for recreational sun exposure in the subgroup of "no or light tanners" was higher (OR = 2.82) than that among "average or dark tanners" (OR = 1.13).

Dubin N, Mosseson M, Pasternack BS. Sun Exposure and Malignant Melanoma among Susceptible Individuals. Environmental Health Perspectives Vol. 81, pp. 139-151, 1989

"*For good tanners, moderate exposure was protective against melanoma,*"

Dubin N, Mosseson M, Pasternack BS. Sun Exposure and Malignant Melanoma among Susceptible Individuals. Environmental Health Perspectives Vol. 81, pp. 139-151, 1989

A study evaluating tanning beds and the incidence of skin cancer analyzed a large US cohort of nurses (NHSII). The study provided subset analysis for both high and low skin pigmentation. This was based on hair colour and reaction to sun exposure during childhood or adolescence. The study reported for women who used a sunbed > 3 times per year during their ages of 25 to 35 years, high pigmentation had a 12% reduced risk of melanoma (OR 0.88).

Zhang M, Qureshi AA, Geller AC, Frazier L, Hunter DJ, Han J. Use of Tanning Bed and Incidence of Skin Cancer. J Clin Oncol. 2012 May 10;30(14):1588-93. doi: 10.1200/JCO.2011.39.3652. Epub 2012 Feb 27

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The Western Canada Melanoma Study also detected a significant inverse association between melanoma and chronic or long-term occupational sun exposure in men, with the lowest risk (OR = 0.5) in those with maximum occupational exposure, suggesting that chronic exposure may be protective. The study went on to say "*It is known that chronic sun exposure tends to thicken the epidermis and lead to a year-round tan and both of these afford protection of the underlying melanocytes from the effects of the sun, thus perhaps providing a biological explanation for the lower incidence of cutaneous melanoma among outdoor workers*." The study concluded *"These observations suggest that part of the increase in the incidence of melanoma in low-sunlight areas may be due to a reduction over the past 40 years of the size of this group of individuals "protected" by their exposure to UV light*."

Gallagher RP, Elwood JM, Yang CP. Is Chronic Sunlight Exposure Important In Accounting For Increases In Melanoma Incidence? Int J Cancer. 1989 Nov 15;44(5):813-5.

An early attempt of a systematic review and meta-analysis of tanning beds, sunlamps and risk of cutaneous malignant melanoma uncovered a number of confounding factors. They reported "*In order to attempt to make the analysis reflect, as far as possible, recreational rather than medical use of sunlamps/sunbeds, we did not include studies of psoralen and UVA radiation therapy*." They went on to say "*The published articles covered a time period of nearly 20 years. During this time the UV emissions of artificial tanning devices changed in character. Sunlamps used up to the late 1970s were usually used in the home setting (except medical use) and emitted primarily UVB (sometimes with a small component of UVC)*." This raises the question as to whether results from the early studies can be legitimately combined with those of more recent studies. The study further cautioned "*Finally, although we have used risk estimates adjusted for phenotype factors and, when possible, for sun exposure, it is unlikely that the studies have achieved complete control for these potential confounders. If individuals who use artificial tanning devices are more likely to suntan, as many suspect, then some of the elevated risk seen might be due to recreational sun tanning*." Finally the study concluded "*If there is a causal relationship between sunlamp/sunbed exposure and melanoma, the public health question is asked: how important is the risk? It is not possible with the data available to answer this question with any certainty*."

Gallagher RP, Spinelli JJ, Lee TK. Tanning Beds, Sunlamps, and Risk of Cutaneous Malignant Melanoma.Cancer Epidemiol Biomarkers Prev. 2005 Mar;14(3):562-6.

Phototherapy and Risk of Skin Cancer

Dermatologists have been treating skin conditions with phototherapy equipment for over 75 years. Treatments used include broadband (BB) UVB, narrowband (NB) UVB and PUVA phototherapy. PUVA combines the use of a psoralen such as 8-methoxypsoralen and UVA exposure.

Consumers can also purchase phototherapy equipment directly in Canada from companies such as Solarc Phototherapy from Barrie ON which advertise "No prescription required, Health Canada compliant for Vitamin D Deficiency." This type equipment carries a 1989 warning label which was also used by UV tanning equipment up until 2005. These types of equipment are listed on long with UV light from sunlight and tanning equipment.

http://www.solarcsystems.com/

WHO IARC recognize UV Phototherapy treatments as a dangerous Group 1 carcinogen:

Methoxsalen (8-methoxypsoralen) plus ultraviolet A radiation - Group 1 - Volume 24, Sup 7, 100A

Research has shown that phototherapy equipment, especially PUVA treatments have significant skin cancer risks.

A prospective study of 1380 patients that received photochemotherapy using oral methoxsalen (8-methoxypsoralen or psoralen) and ultraviolet A radiation (PUVA) treatments in 1975 or 1976 reported an elevated relative risk for melanoma of RR 5.4. The study stated that "*In many patients who receive PUVA therapy irregular, pigmented macules develop and persist long after the therapy is stopped*." The study concluded "*About 15 years after the first treatment with PUVA, the risk of malignant melanoma increases, especially among patients who receive 250 treatments or more*."

Stern RS, Nichols KT, Vakeva LH. Malignant melanoma in patients treated for psoriasis with methoxsalen (psoralen) and ultraviolet A radiation (PUVA). N Engl J Med 1997;336:1041-5.

Stern updated this prospective study in 2012 and looked at SCC and BCC risk. They reported that of the 1380 people who had PUVA treatments in the study 25% developed SCC and 24% developed BCC. The reported IRR for SCC was 20.92 and for BCC was 3.09. The study concluded "*Exposure to more than 350 PUVA treatments greatly increase the risk of SCC*."

Stern RS. The risk of squamous cell and basal cell cancer associated with psoralen and ultraviolet A therapy: a 30-year prospective study. J Am Acad Dermatol. 2012 Apr;66(4):553-62. doi: 10.1016/j.jaad.2011.04.004

A systematic review of 49 published studies psoralen UV-A and narrowband UV-B therapy was published in 2012. The study concluded "*There is an increased risk of skin cancer following PUVA, shown by both US and European studies. The greater risk measured by the US studies may be at least partly explained by high UVA dose exposure and the lighter phototypes of the treated patients.*"

Archier et al,. Carcinogenic risks of psoralen UV-A therapy and narrowband UV-B therapy in chronic plaque psoriasis: a systematic literature review. J Eur Acad Dermatol Venereol 2012 May;26 Suppl 3:22-31 doi: 10.1111/j.1468-3083.2012.04520.x.

The Dermatology Phototherapy Contradiction

Dermatology groups still market “safe” UV sunbed sessions in their own offices to treat purely cosmetic skin conditions while simultaneously telling the world that any UV exposure not received in their offices is dangerous.

Dermatology lobbies that teenagers should not use sunbeds in salons at all, it also concludes that dermatology phototherapy can be delivered to children when a dermatologist decides such a treatment is necessary.

The AAD has publicly called its own usage of sunbeds safe as a treatment for psoriasis and many dermatology clinics use the word on web pages describing phototherapy procedures . Their argument is that a doctor monitors UV treatments in a phototherapy clinic, but so does a professional salon.

Dermatology phototherapy often involves intentional sunburn, where sunbed exposure times are designed to stay below a sunburning level. Phototherapy units are more intense than standard indoor tanning salon sunbeds. In lobbying for tighter regulations on indoor tanning sunbeds, senior dermatology leaders have denied any connection between phototherapy and skin cancer while saying that indoor sunbeds do cause skin cancer.

Many psoriasis patients visit tanning salons today as a more accessible, less expensive and less invasive way to self-treat cosmetic conditions. A significant percentage of indoor tanning clients are referred by doctors for therapeutic reasons and those doctors include dermatologists.

If UV exposure from UV-emitting tanning equipment were as dangerous as dermatologists claim, then dermatologist would be guilty of providing the same treatment in their medical practices.

Many patients are referred to tanning salons instead of dermatologists by physicians, as the cost of a tanning session is almost always less expensive than a dermatology-based phototherapy session. The AAD reported that in 1993 dermatologists administered 873,000 visits for phototherapy sessions. By 1998 that figure had dropped by 94% to 53,000 visits.

If it was purely a health care debate the dermatologists would stop using phototherapy equipment themselves.

Professional Commercial Tanning Facilities Reduce Risk

A professional commercial tanning salon following JCTA guidelines has a number of factors which control the use of UV producing equipment. The equipment itself is not the risk. Using the equipment improperly is where the problem lies. This is why JCTA guidelines stress the following:

Canadian tanning salons are run in a very controlled, professional manner. Over exposure to UV light is quite different than just plain exposure. Commercial sunbathing salons take particular care NOT to burn or over-expose anyone. Scientific studies show that intermittent exposure, sunburns and people with certain genetic factors (11 known) and skin types are the significant risk factors for skin cancer [1]. People who have regular exposure to UV, such as Outdoor workers, have a lower risk of melanoma [9, 10, 15].

Professional salons take the following steps to ensure that sunbathers are protected against over-exposure to UV:

- all new sunbathers are skin typed to determine first if they can sunbathe and secondly to develop proper exposure times.
- all sunbathers start on a graduated exposure schedule based on skin type to ensure that they gradually build up exposure in a non-burning fashion
- the JCTA recommends all salons get parental consent for any tanners under the age of 18
- all sunbathers must wear protective eyewear
- sunbathers must follow the manufacturer's recommended exposure schedule
- all salon staff are trained and certified. The trained staff controls the sunbed timers

These steps and controls are what are used in a professional salon to reduce risk of overexposure as compared to outdoor sun exposure or uncontrolled home unit.

Consider:

Salon Operator – an operator controls access to equipment and advises each client on every visit they tan. Computer software/Client card displays the last time a client tanned. The operator controls the time each client receives on the equipment to ensure an optimal exposure time with no burning. The operator does a skin type review of the client and ensure that they set the equipment to ensure the client is not burned or over-exposed.

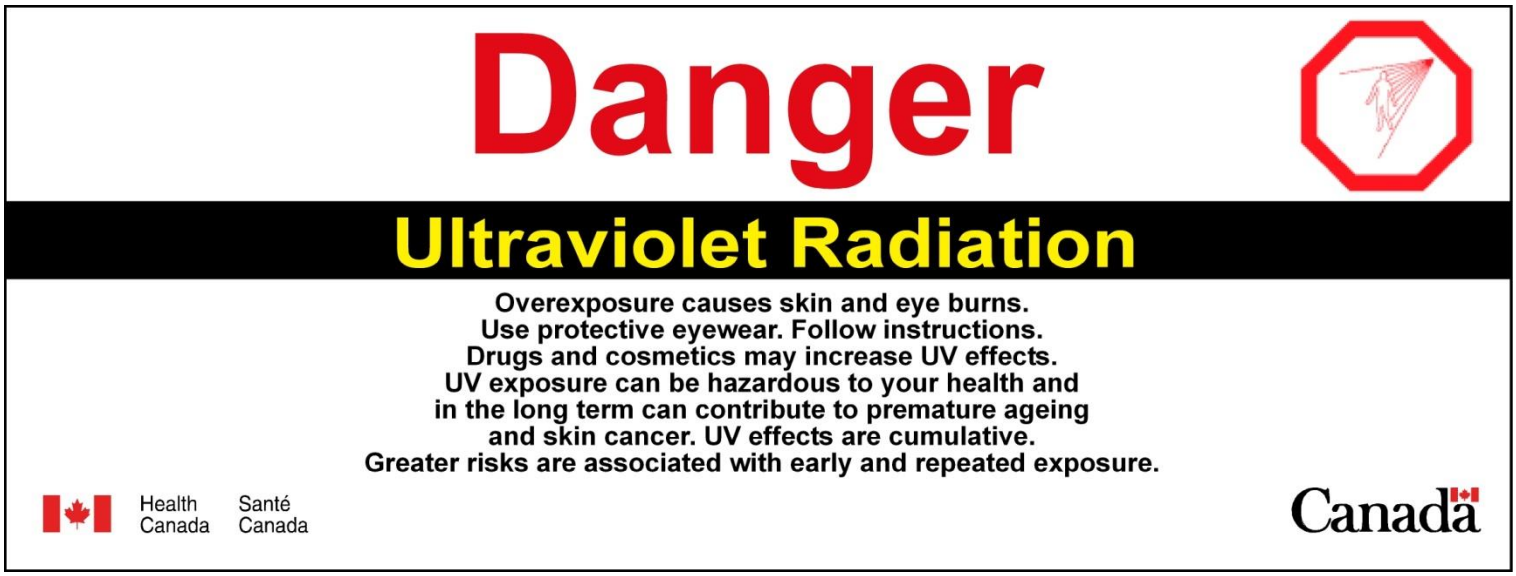
Tanning Bed Timer Control – all equipment is controlled by a timer which shuts the equipment off automatically after a session. This can be controlled by the operator at the main desk. The client can also manually switch the equipment off any time they require.

Protective Eyewear - salons either sell or provide protective eyewear to clients and advise them to be worn at all times when using the tanning equipment. Professional commercial tanning salons require that customers wear approved tanning goggles while tanning to prevent eye damage. The Health Canada Warning Label says the same.

Unfortunately there are no provincial regulations that customers must have approved eyewear on each time they tan. That is why the JCTA is asking provincial governments to develop Professional Standard for the tanning industry.

Minimizing risk is always about who controls the equipment, bans just have the public find equipment that is uncontrolled – self-serve, home units or outside.

Equipment Safety Labels - Health Canada requires all tanning equipment to have a "Danger Ultraviolet Radiation" sticker with all consumer warnings prominently displayed. In addition, manufacturers provide a recommended exposure schedule recommending controlled tanning exposure times by skin type and by session number or week.


Danger
Ultraviolet Radiation
Overexposure causes skin and eye burns.
Use protective eyewear. Follow instructions.
Drugs and cosmetics may increase UV effects.
UV exposure can be hazardous to your health and
in the long term can contribute to premature ageing
and skin cancer. UV effects are cumulative.
Greater risks are associated with early and repeated exposure.
Health Canada
Santé Canada
Canada


Danger
Rayonnements ultraviolets
La surexposition provoque des brûlures aux yeux et à la peau.
Porter le dispositif de protection des yeux. Suivre les instructions.
Médicaments et cosmétiques peuvent augmenter les effets des UV.
L'exposition aux UV peut avoir des effets nocifs sur la santé
et contribuer, à long terme, au vieillissement prématuré
et au cancer de la peau. Ces effets sont cumulatifs.
Plus l'exposition réguliére commence tôt,
plus les risques qui y sont associés sont élevés.
Santé Canada
Health Canada
Canada

ACTUAL SIZE
