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Skin Damage Prevention: (Artificial) Sunscreen vs (Natural) Facultative Pigmentation

By Donald L. Smith / Director of Research

Several organizations [1,2,3] claim that studies [4-9] show that the skin damage protection afforded by our (natural) facultative pigmentation (a "tan") resulting from sunbed exposure is only equivalent to a SPF 2 or SPF 3 (artificial) sunscreen even though they know, or ought to know, (a) that the dose (initial, maximum and cumulative) of facultative pigmentation-effective photons administered was too low in these studies (shown below) to stimulate a more protective level; and, (b) that the (natural) facultative pigmentation resulting from sunbed exposure is directly related to the dose (initial, maximum and cumulative) administered (see pages 2 and 3).

It was also stated that, in order for a sunbed tan to be considered worthwhile, protection equal to a SPF 15 would be required but they failed to mention that the dose (thickness) of (artificial) sunscreen applied affects the actual (real world) effectiveness of the protection and that (ironically) a SPF 15 only provides a SPF 2 to SPF 3 protection in the real world (see page 4). Moreover, since (artificial) sunscreens wash and sweat off, degrade when exposed to sunlight and contain potentially harmful chemicals and additives, they are a poor (secondary) substitute for the (primary) protection afforded by our (natural) constitutive and facultative pigmentation.

A low dose results in a minimally protective tan. The dose (initial, maximum and cumulative) of facultative pigmentation-effective photons administered in the studies shown below [4,5,6,7,8,9] that are the genesis for the claim that the protection resulting from sunlight or sunbed exposure is about the same as using an SPF 2 or SPF 3 (artificial) sunscreen was too low to stimulate a more protective level of (natural) facultative pigmentation (a ñtanò). Because the dose of facultative pigmentation-effective photons used in the studies was insufficient, the average level (SPF 2.28 equivalent) of (natural) facultative pigmentation was only marginally above the protection (skin type II = SPF 1.3 / skin type III = SPF 1.7) afforded the participants by their (natural) constitutive pigmentation. ***Thus, although these studies showed that a tan only provides the protection equivalent of a SPF 2 to SPF 3, the dose (initial, maximum and cumulative) of facultative pigmentation-effective photons administered was too low to stimulate a more protective TUVR (Tolerance to UVR) level.***

Study	TUVR (SPF Equivalent)	SUVR (Sensitivity)	Risk Reduction (%)	Maximum Dose (J/m^2)	Sessions (Number)
Cripps/Simulator /ST 2 [4]	2.4	0.42	58	280	12 sessions (1)
Cripps/Simulator /ST 3 [4]	2.45	0.41	59	280	12 sessions (1)
Cripps/Sunlight [4]	2.33	0.43	57	230	3 ½ Months (2)
Sheehan/Skin Type II/0.5 [5]	1.1	0.91	9	100	10 sessions (1)
Sheehan/Skin Type II/0.75 [5]	3.1	0.32	68	150	10 sessions (1)
Sheehan/Skin Type III/0.5 [5]	1.4	0.71	39	100	10 sessions (1)
Sheehan/Skin Type III/0.75 [5]	2.8	0.36	64	150	10 sessions (1)
Sheehan/Skin Type II/0.65 [6]	2.0	0.50	50	130	10 sessions (1)
Sheehan/Skin Type IV/0.65 [6]	2.0	0.50	50	130	10 sessions (1)
Pathak [7]	2.1	0.48	52	220	1,2,3,6,9 SBU (3)
Rivers [8]	2.7	0.37	63	340	12 sessions (1)
Devgun [9]	3.0	0.33	67	400	10 sessions (1)
AVERAGE	**2.28**	**0.48**	**52**	**210**	

(1) Number of sessions over two (2) weeks / (2) Madison, Wisconsin summer sun / (3) One SBU = 30 mJ/cm^2

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A moderate dose results in a moderatately protective tan. The Miller [10,11] and Caswell [12] studies show that an increase in TUVR (SPF equivalent) after a series of tanning sessions is proportional to the dose (initial, maximum and cumulative) regimen. A SPF 2 tan only requires 3-4 tanning low dose sessions and a SPF 3 only requires 4-6 low dose tanning sessions.

Miller Studies. Two studies by Miller, et al, [10,11] showed that the increase in TUVR (SPF ñequivalentò) achieved by a series of graduated exposures is directly proportional to the dose (initial, maximum and cumulative) administered. *[The increase in TUVR (SPF equivalent) was confirmed by the more reliable (than visual grading)* AE *(ITA) spectrophotometer measurement.]*

Week	Day	Session	Dose/A (J/m^s)	MED	TUVR (SPF)	AE	Dose /B (J/m^2)	MED	TUVR (SPF)	AE	Dose/C (J/m^2)	MED	TUVR (SPF)	AE
1	1	1	100	0.5	1.0		100	0.5	1.0		100	0.5	1.0	
1	2	2	130	0.65	1.3		140	0.7	1.4		150	0.75	1.5	
1	5	3	160	0.8	1.6	**2.5**	**200**	**1.0**	**2.0**	**2.7**	**230**	**1.15**	**2.3**	**3.0**
2	**8**	**4**	**200**	**1.0**	**2.0**		**280**	**1.4**	**2.8**		**340**	**1.7**	**3.4**	
2	11	5	250	1.25	2.5	3.7	400	2.0	4.0	4.9	500	2.5	5.0	6.1
3	**16**	**6**	**300**	**1.5**	**3.0**		560	2.8	5.6		600	3.0	6.0	
3	19	7	380	1.9	3.8	5.9	600	3.0	6.0	10.0	600	3.0	6.0	11.5
4	22	8	380	1.9	3.8		600	3.0	6.0		600	3.0	6.0	
4	25	9				**8.0**				**12.9**	600	3.0	6.0	14.1
5	29	10				7.3				11.9	600	3.0	6.0	**15.9**
			1900				2880				4320			
AVERAGE			**238**	**1.5**	**2.4**	**5.5**	**360**	**1.8**	**3.6**	**8.5**	**432**	**2.2**	**4.3**	**10.1**

Caswell Study: A study [12] by Michael Caswell confirmed the increase in TUVR (SPF equivalent) shown by the Miller study and documented that the increase is directly proportional to the dose (initial, maximum and cumulative) that is administered. *[Once again, the increase in TUVR (SPF equivalent) was confirmed by the more reliable* AE *(ITA) spectrophotometer measurement.]*

Day	Session	Skin Type 3 (J/m^2)	MED	AE	TUVR (SPF)	Skin Type 4 (J/m^2)	MED	AE	TUVR (SPF)
1	1	66	0.42		1.0.	66	0.42		1.0
3	2	66	0.42		1.0.	66	0.42		1.0
5	3	66	0.42	**2.0**	1.0.	66	0.42		1.0
8	**4**	**154**	**0.99**		**2.33**	**220**	**1.6**		**3.33**
10	**5**	**154**	**0.99**		**2.33**	220	1.6		3.33
12	6	154	0.99	**2.7**	2.33	220	1.6		3.33
15	7	330	2.1		5.0	330	2.1		5.0
17	8	330	2.1		5.0	330	2.1		5.0
19	9	330	2.1	4.0	5.0	330	2.1		5.0
22	10	440	2.8		6.67	440	2.8		6.67
24	11	440	2.8		6.67	440	2.8		6.67
26	12	440	2.8	6.8	6.67	440	2.8		6.67
29	13	440	2.8		6.67	440	2.8		6.67
31	14	440	2.8		6.67	440	2.8		6.67
33	15	440	2.8	8.5	6.67	440	2.8		6.67
36	16	550	3.5		**8.33**	550	3.5		**8.33**
38	17	550	3.5		8.33	550	3.5		8.33
40	18	550	3.5	9.0	8.33	550	3.5		8.33
43	19	550	3.5		8.33	550	3.5		8.33
45	20	550	3.5		8.33	550	3.5		8.33
47	21	550	3.5	10.7	8.33	550	3.5		8.33
50	22	550	3.5		8.33	550	3.5		8.33
52	23	550	3.5		8.33	550	3.5		8.33
54	24	550	3.5	**11.5**	8.33	550	3.5		8.33
		9,240				9,438			
AVERAGE		**385**	**2.45**	**6.9**	**5.8**	**393**	**2.51**		**6.0**

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Moderate dose summary. The exposure schedule regulations/guidelines promulgated by national and international regulatory agencies dictate the safe and gradual progression from the IST (Initial Session Time) to the MST (Maximum Session Time) for a new client of indoor tanning salons/solaria. As the table below shows, the ñaverageò TUVR (SPF equivalent) for the FDA [13], Health Canada [14] and CIE [15] exposure schedule regulations/guidelines is 6.5 (range: 5.33 to 8.0). The average decrease in SUVR (Sensitivity to Ultraviolet Radiation) was from 1.0 to 0.15, which means there is a ñrisk reductionò of 85% when the maximum allowable dose was reached.

Regulation/ Guideline	Dose/Start (J/m^2)	TUVR/Start (SPF Equivalent)	Dose/End (J/m^2)	TUVR /End (SPF Equivalent)	SUVR (Start)	SUVR (End)	Risk Reduction (%)
FDA	117	1.0	624	**5.33**	1.0	**0.19**	**81**
Health Canada	100	1.0	625	**6.25**	1.0	**0.16**	**84**
CIE	100	1.0	800	**8.0**	1.0	**0.13**	**87**
AVERAGE	**106**	**1.0**	**683**	**6.5**	**1.0**	**0.15**	**85**

A higher dose results in a more protective tan. The Cripps [4] study revealed that a higher dose does result in a more protective tan. When a higher dose (initial, maximum, average and cumulative), was administered in 12 sessions (average dose = 693 J/m^2 / starting dose <600 J/m^2 / ending dose >800 J/m^2) over a two week period, the resultant TUVR (SPF equivalent) was a robust 8.01. The TUVR (SPF equivalent) of 8.01 was 3.3 times higher than the 2.45 TUVR (SPF equivalent) that Cripps, et al, found when a maximum dose of 280 J/m^2 was administered. Significantly, the higher dose resulted in 87% risk reduction versus 59% risk reduction with the lower dose.

Study	TUVR (SPF)	SUVR (Sensitivity)	Risk Reduction (%)	Maximum Dose (J/m^2)	Average Dose (J/m^2)	Sessions (Number)
Cripps/Simulator /ST 3 [4]	**8.01**	0.13	**87**	>**800**	693	12 sessions (1)
Cripps/Simulator /ST 3 [4]	**2.45**	0.41	**59**	**280**		12 sessions (2)

(1) Number of sessions over two (2) weeks / (2) Number of sessions over two (2) weeks / 5 individuals received high dose

And a very low dose results (as you would expect) in very minimal protection. Compare this to the TUVR (SPF equivalent) resulting from use of the AU/NZ Sunlamp Standards [16] which limits the initial session dose to 0.5 MED; the maximum session dose to 0.9 MED; and limits the maximum weekly dose to 3.0 MED. As you can see from the table below, the average TUVR (SPF equivalent) is only 1.6, a level too low to provide adequate protection and reduce the risk of skin damage.

Regulation	Dose/Start (J/m^2)	TUVR/Start (SPF Equivalent)	Dose/End (J/m^2)	TUVR /End (SPF Equivalent)	SUVR (Start)	SUVR (End)	Risk Reduction (%)
AU/NZ-Type 2	125	1.0	225	**1.6**	1.0	**0.625**	**37.5**
AU/NZ-Type 3	175	1.0	315	**1.6**	1.0	**0.625**	**37.5**
AU/NZ-Type 4	225	1.0	405	**1.6**	1.0	**0.625**	**37.5**
AVERAGE	**175**	**1.0**	**315**	**1.6**	**1.0**	**0.625**	**37.5**

[Note: If the regulatory goal is to reduce the risk of skin damage, it is important to note that there is only a 37.5% risk reduction when the AU/NZ exposure schedule regulation/guideline is used as compared to the average of 85% risk reduction when the FDA, Health Canada and CIE exposure schedule regulations/guidelines are used. Increasing the dose (initial, maximum and cumulative) results in a darker level of (natural) facultative pigmentation; an increase in TUVR (Tolerance to UVR) and a decrease in SUVR (Sensitivity to UVR).]

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How well does (artificial) sunscreen protect the skin? The recommendation that an (artificial) sunscreen with at least an SPF of 15 be slathered on every day of the year no matter the season fails to disclose the fact that the dose (thickness) of sunscreen applied affects the actual (real world) effectiveness of the protection. As shown below, a SPF 15 only provides a SPF 2 to SPF 3 as typically applied in the real world. In order for the label SPF level to match the ñactualò SPF level a dose (thickness) of 2.0 mg/cm^$_2$ (100%) must be evenly applied. Several studies [17 through 26] show that the actual dose (thickness) applied is between 0.5 mg/cm^$_2$ (25%) and 0.76 mg/cm^$_2$ (39%).

SPF on label at (2.0 mg/cm^2)/100%	SPF of users at (1.0 mg/cm^2)/50%	SPF of users at **(0.76 mg/cm^2)/39%**	SPF of users at **(0.5 mg/cm^2)/25%**	SPF of users at (0.25 mg/cm^2)/12.5%
15	4.0	3.0	2.0	1.0
30	4.6	3.6	2.3	1.15
50	5.2	4.0	2.6	1.3
100	6.4	4.9	3.2	1.6

Real world protection summary. The table below compares the time that the skin of a skin type II individual (with a sunburning time of 10 minutes) will be protected from skin damage by a SPF 15 (artificial) sunscreen in the real world when the ñusual and customaryò dose (thickness) of 25% (0.5 mg/cm^2) or 39% (0.76 mg/cm^2) is applied with the time the skin will be protected by facultative pigmentation (a ñtanò). As shown below, a person applying a SPF 15 sunscreen ñexpectsò to have 150 minutes of protection but what they actually get in the ñreal worldò is only 20 minutes (25% dose) or 30 minutes (39% dose) and it is this ñfalse sense of securityò that is one of the main reasons people sunburn outdoors even though they used a sunscreen. However (natural) facultative pigmentation provides protection of 65 minutes (3.4 MED) to 76 minutes (4.0 MED), a very significant increase.

The table below also shows the protection time when a SPF 100 (artificial) sunscreen is applied, the ñexpectedò protection time is 1,000 minutes (16.7 hours) but the ñreal worldò protection time is only 32 minutes (25% dose); or, 49 minutes (39% dose).

SPF 15 Example (Skin type II individual)

Sun Burning Time = 10 minutes X 15 (100%/2.0 mg/cm^2)	**= 150 Minutes Protection**
Sun Burning Time = 10 Minutes X 2.0 (25%/0.5 mg/cm^2)	= 20 Minutes Protection
Sun Burning Time = 10 Minutes X 3.0 (39%/0.76 mg/cm^2)	**= 30 Minutes Protection**

SPF 100 Example: (Skin type II individual)

Sun Burning Time = 10 minutes X 100 (100%/2.0 mg/cm^2)	**= 1000 Minutes Protection**
Sun Burning Time = 10 Minutes X 3.2 (25%/0.5 mg/cm^2)	= 32 Minutes Protection
Sun Burning Time = 10 Minutes X 4.9 (39%/0.76 mg/cm^2)	**= 49 Minutes Protection**

Facultative Pigmentation Example: (Skin type II individual)

Sun Burning Time = 10 Minutes X 1.6 (AU/NZ Standard/0.85 MED)	**= 16 Minutes Protection**
Sun Burning Time = 10 Minutes X 2.9 (Light Tan/1.0 MED)	**= 29 Minutes Protection**
Sun Burning Time = 10 Minutes X 3.8 (Light/Medium Tan/2.0 MED)	**= 38 Minutes Protection**
Sun Burning Time = 10 Minutes X 5.7 (Medium Tan/3.0 MED)	**= 57 Minutes Protection**
Sun Burning Time = 10 Minutes X 6.5 (Regulatory Average/3.4 MED)	**= 65 Minutes Protection**
Sun Burning Time = 10 Minutes X 7.6 (Medium/DarkTan/4.0 MED)	**= 76 Minutes Protection**
Sun Burning Time = 10 Minutes X 6.5 (Regulatory High/4.4 MED)	**= 83 Minutes Protection**

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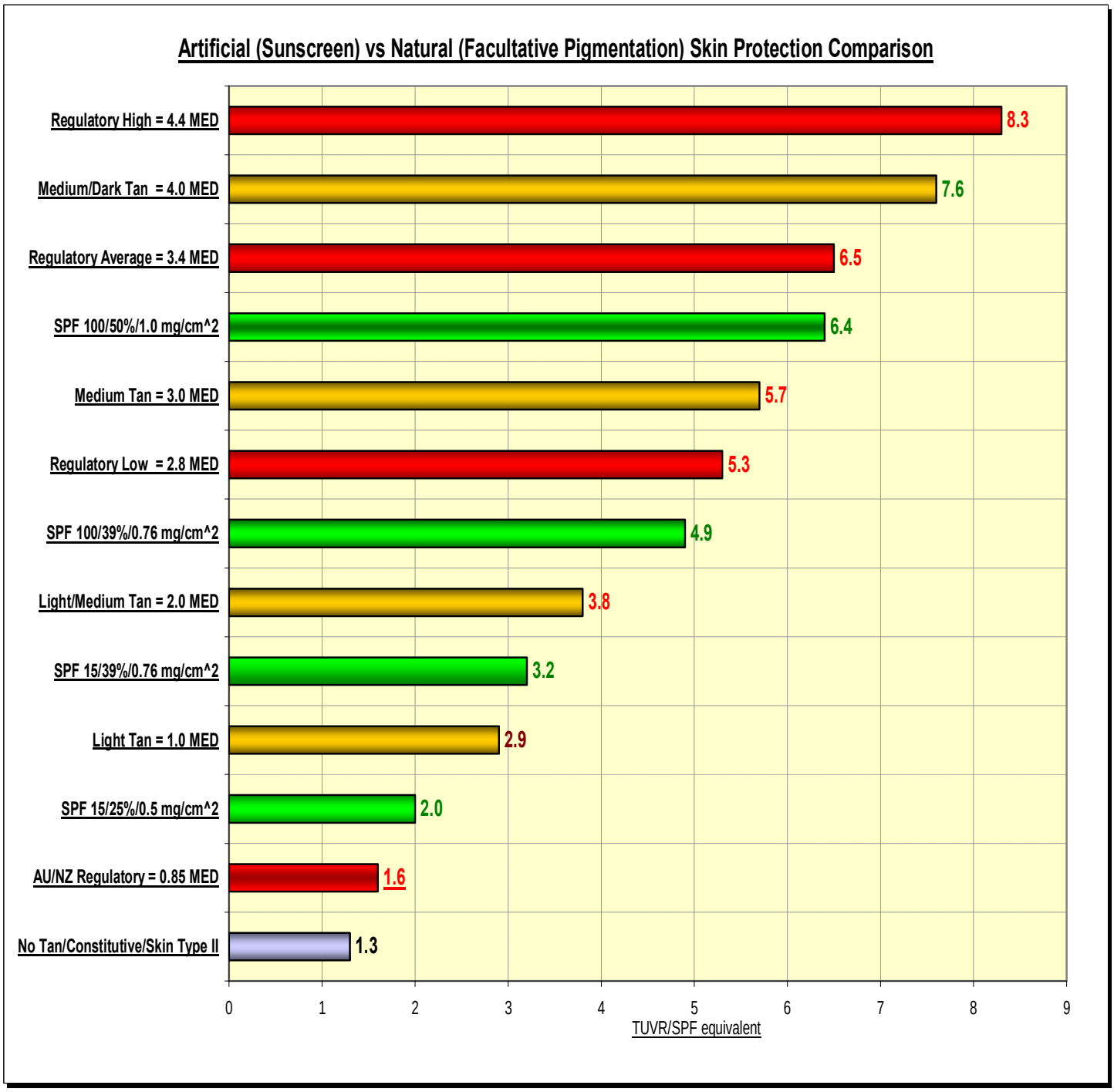
SUMMARY GRAPH: The graph below compares the skin protection afforded by an (artificial) sunscreen with the development of (natural) facultative pigmentation (a ñtanò) after controlled exposure to the facultative pigmentation-effective photons emitted by a sunbed.



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A new study shows that our (natural) constitutive and facultative pigmentation, but not the use of (artificial) sunscreen, was correlated with reduction of urinary 8-OHdG level in humans.

An article [27] titled ñSunlight exposure-mediated DNA damage in young adultsò by Masashi Kato, Machiko Iida, Yuji Goto, et al., was published (online) in *Cancer Epidemiology Biomarkers and Prevention* on June 15, 2011 and it contains very important new information. The authors found that ñincreased skin pigmentation levelò i.e., an increase in the level of (natural) facultative pigmentation but ñnot the use of sunscreenò was correlated with the reduction of urinary 8-OHdG level in humans. This important new information serves to confirm the premise that Mother Natureôs ñnormal and naturalò way of protecting the skin from ultraviolet radiation damage ╖our (natural) constitutive and facultative pigmentation ╖ is superior to the use of an ñartificialò sunscreen.

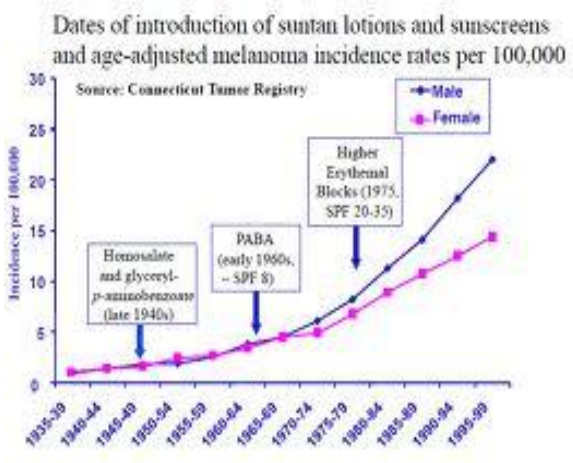
Key points from Kato, et al, article:

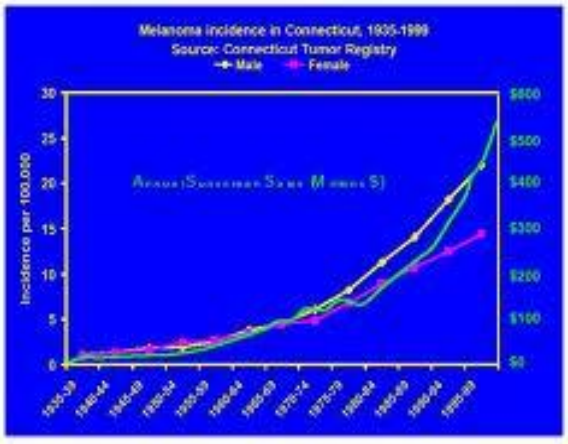
- **Urinary 8-OHdG (8-hydroxy-2'-deoxyguanosine) is thought to be a critical biomarker of carcinogenesis as well as oxidative DNA damage.**
- **There are two possible mechanisms for the regulation of urinary 8-OHdG levels by cutaneous melanin. One is via the antioxidative effect and the other is via physical block of sunlight.**
- **Our results suggest that cutaneous melanin-mediated physical block of sunlight rather than a melanin-mediated antioxidative effect plays an important role in the regulation of urinary 8-OHdG levels.**
- **Although we anticipated that urinary 8-OHdG levels would be decreased in regular sunscreen users, there was no difference between urinary 8-OHdG levels in sunscreen users and non-users in our study.**

The Kato, et al, study mandates that the following questions be addressed:

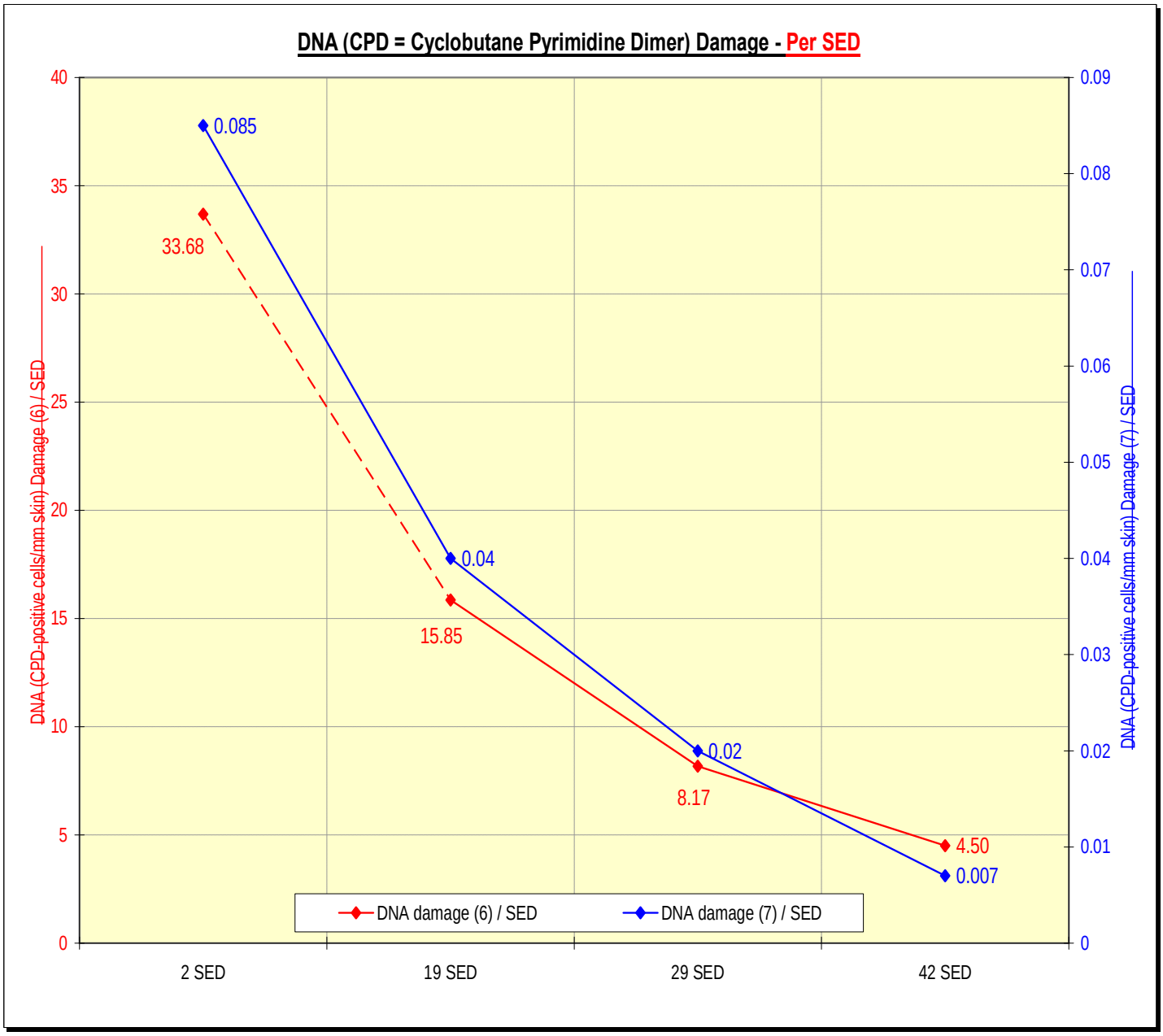
- Has the increased use of sunscreen over the past five decades been counterproductive?
- Is it possible that sunscreen use has increased the incidence of skin cancer?
- Has sunscreen use been part of the problem instead of being part of the solution?

The graph on the left (below) compares the introduction date of various sunscreen innovations with the increase in age-adjusted melanoma rates per 100,000 individuals and the graph on the right (below) compares the rate of melanoma increase with sunscreen sales. Together, these two graphs confirm the fact that the increase in sunscreen use has ñmirroredò the increase in the incidence of cutaneous malignant melanoma (CMM). The question that must now be addressed is whether the relationship between sunscreen use and CMM (shown on these graphs) is causal or coincidental.

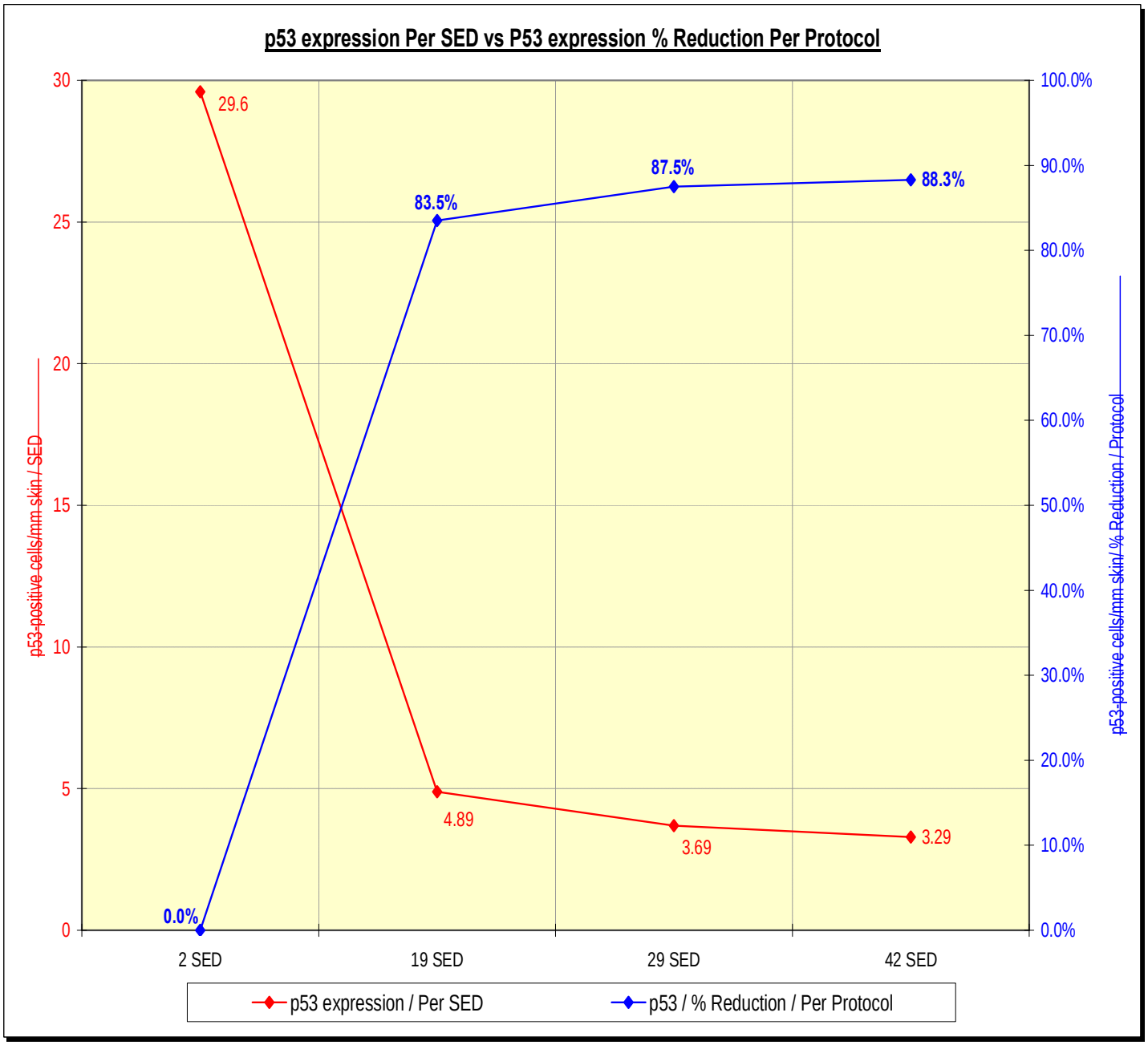




The Yamaguchi study. An important study by Yamaguchi, et al, [28] showed that the development of (natural) facultative pigmentation provides protection from skin damage beyond just the prevention of sunburning. This study looked at the ñmolecular markersò of skin damage in the subjects in the Miller studies [10,11] that received 19, 29 and 42 SED. The first graph shown below shows the DNA damage, i.e., CPD (cyclobutane pyrimidine dimer) formation (CPD-positive cells/mm skin) damage (6) per SED and (CPD-positive cells/mm skin) damage (7) per SED in the skin after exposure to 2, 19, 29 and 42 SED (1 SED = 100 J/m^2 / 2 SED = 1.0 MED). As you can see, there was an almost straight line decrease in the amount of CPD (6 & 7) formed as the cumulative dose increased. Moreover, this increase ñmirroredò the development of (natural) facultative pigmentation (see page 2 of this report) as the cumulative dose went from 2SED to 18 SED; 29 SED; and 42 SED.



The second graph (below) shows p53 expression per SED vs p53 expression (% reduction) per protocol in the skin after exposure to 2, 19, 29 and 42 SED (1 SED = 100 J/m^2) protocols. As you can see, there was a dramatic decrease in the amount of p53 expression per SED from 2 SED (2 SED = 1.0 MED) to 19 SED and a lesser decrease from 19 SED to 29 and 42 SED. Thus, it can be seen that the amount of p53 expressed is highest after the initial exposure of 2 SED/1.0 MED and then decreases dramatically as the dose reaches 19 SED (an 83.5% reduction); 29 SED (87.5% reduction); and 42 SED (88.3% reduction) showing the protection afforded by (natural) facultative pigmentation.



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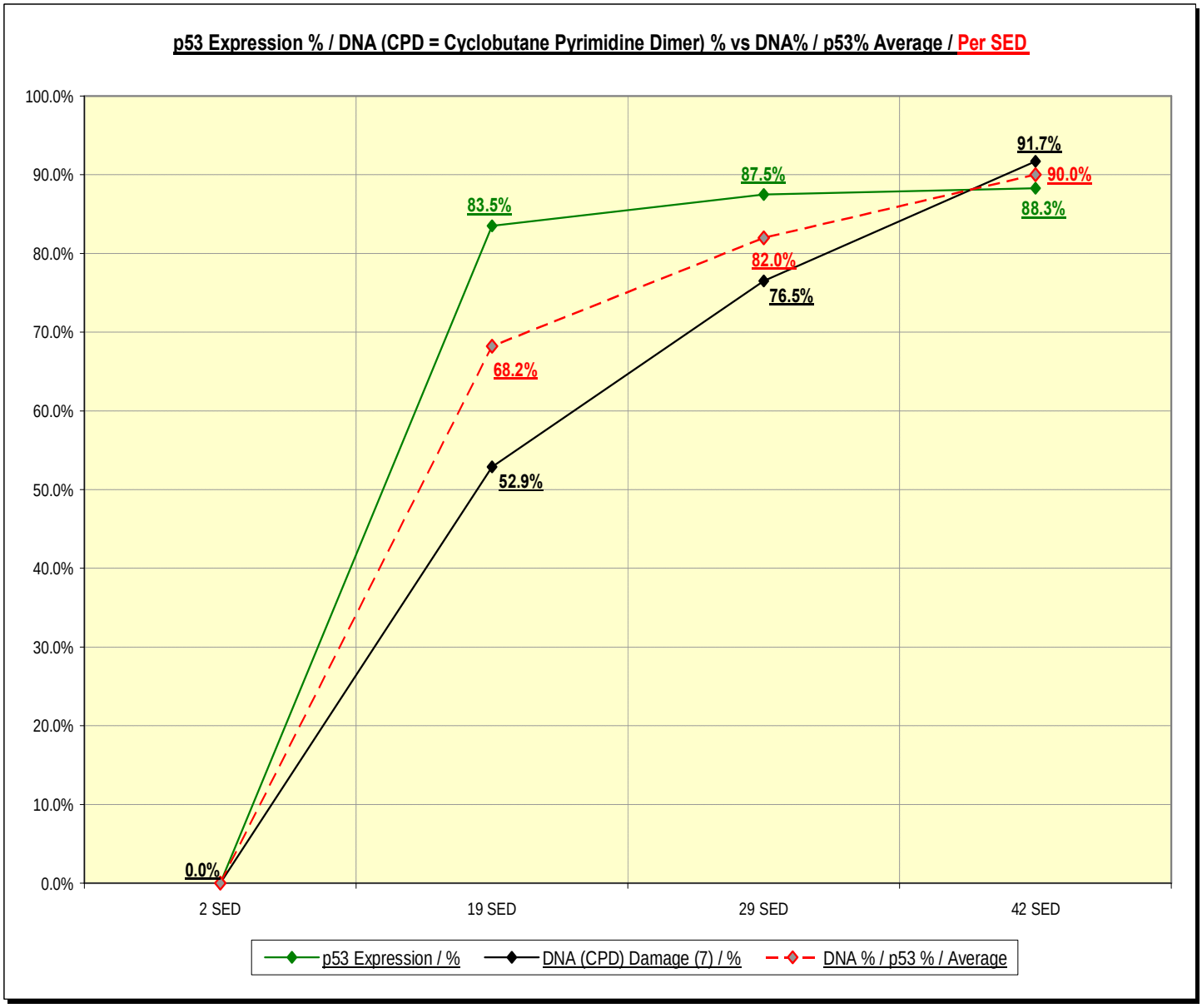
The last graph (below) shows both (a) p53 expression per SED vs p53 expression (% reduction) per protocol in the skin after exposure to 2, 19, 29 and 42 SED (1 SED = 100 J/m^2) protocols; (b) the DNA damage, i.e., CPD (cyclobutane pyrimidine dimer) formation (CPD-positive cells/mm) skin damage (7) / SED) in the skin; and, (c) the ñaverageò of these two parameters. As you can see, there was a dramatic decrease from 2 SED (2 SED = 1.0 MED) to 19 SED and a steady but lesser decrease from 19 SED to 29 and 42 SED. The graph shows that the amount of p53 expressed and DNA (CPD) formation decreases dramatically as the dose increased from 2 SED to 19 SED (an average 68.2 % reduction); from 19 SED to 29 SED (82.0% reduction); and from 29 SED to 42 SED (90.0% reduction) which means that the damage to the skin decreases in direct proportion to the development of (natural) facultative pigmentation.



The truth about sunscreens containing UVA filters. The ñinconvenient truthò about sunscreens containing UVA filters is that their primary (and perhaps only) purpose is to interrupt the bodyôs normal, natural and contravolitional (taking place without conscious thought or action) way to protect the skin from damage, i.e., the development of (natural) facultative pigmentation. In other words, a sunscreen containing UVA filters prevents the body from tanning and no study (that I am aware of) has looked at the ñunintended consequencesò of preventing the development of (natural) facultative pigmentation. Indeed, given the information disclosed by Kato, et al, (page 6), an ñincreased skin pigmentation levelò i.e., an increase in the level of (natural) facultative pigmentation but ñnot the use of sunscreenò was correlated with the reduction of urinary 8-OHdG level in humans and, therefore, the use of an (artificial) sunscreen to prevent the development of (natural) facultative pigmentation may result in adverse (and unintended) health consequences.

The original purpose of a sunscreen was to protect the skin of skin type 1 individuals (who are genetically incapable of tanning) from sunburning and for use by other skin types to prevent them from sunburning while they built up a protective level of (natural) facultative pigmentation (a ñtanò). Then, over time, the message changed to recommending that everyone (no matter their skin type) slather on a sunscreen every day of the year (no matter the time of day or season of the year).

In 2004 an article [29] by Dominique Moyal titled ñPrevention of ultraviolet-induced skin pigmentationò opened the door to dramatically expand the sales of sunscreen products (by adding UVA filters) and forcing clients to be totally reliant (because the use of a sunscreen incorporating UVA filters prevents the development of ñnaturalò facultative pigmentation) on the use of an (artificial) sunscreen for skin protection.

[Note: Even though several responders, including myself, requested that FDA "warn" the public that use of an (artificial) sunscreen containing UVA filters would prevent the development of (natural) facultative pigmentation but FDA declined to do so. Ironically, the primary reason FDA gave for declining to issue this warning was that (natural) facultative pigmentation (a "tan") only results in protection equivalent to a SPF 2 or SPF 3 (artificial) sunscreen.]

***Cui bono fuerit*? Who profits from this?** [Cicero: *Pro Milone* XII.xxxii] Cui bono fuerit? was the principle on which the Roman Judge Lucius Cassius Longinus (2nd Century B.C) decided his cases. He was convinced that the crime was usually committed by the person (or persons) who profited, or would have profited, by the crime and it is hoped that regulatory agencies, legislative bodies and the public will consider this important question: Who will profit from the claim that the skin damage protection afforded by our (natural) facultative pigmentation (a ñtanò) is only equivalent to a SPF 2 or SPF 3 (artificial) sunscreen? **I suggest that the any organization recommending that everyone, no matter their skin type, slather on a sunscreen every day of the year, no matter the time of day or season of the year, are the ones profiting from this claim.**

Sunbeds are the ideal year-round source of vitamin D-effective photons. It is an accepted fact that exposure to the vitamin D-effective wavelengths of ultraviolet radiation stimulate the cutaneous production of vitamin D. A recent article [30] reported that a series of graduated exposure in a sunbed ñé results in higher 25(OH)D concentrations and prevents increased seasonal prevalence of vitamin D deficiency during the winterò and several other studies [31,32,33,34] confirm that sunbeds emit vitamin D-effective photons. It is also an accepted fact that sunlight is an unreliable source of vitamin D-effective photons [35], especially during the fall and winter months in northern latitudes. **Thus, when compared to (uncontrolled exposure) sunlight, (controlled exposure) sunbeds are the ideal year-round source of vitamin D-effective photons.**

Summary and Conclusions.

1. The reason the studies [4,5,6,7,8,9] showed that a sunbed tan only resulted in the protection afforded by a SPF 2 to SPF 3 (artificial) sunscreen was because the dose (initial, maximum and cumulative) of facultative pigmentation-effective photons administered was too low to stimulate a higher level of protection.

    a. **Conclusion: A low dose of facultative pigmentation-effective photons will inevitably result in a low level of TUVR (SPF equivalent).**

2. The level of (natural) facultative pigmentation that develops after exposure to facultative pigmentation-effective photons is directly proportional [10,11,12] to the dose (initial, maximum and cumulative) of facultative pigmentation-effective photons.

    a. **Conclusion: In order to develop a higher level of (natural) facultative pigmentation, there must be exposure to a higher dose (initial, maximum and cumulative) of facultative pigmentation-effective photons.**

3. Therefore, if the studies [4,5,6,7,8,9] had used a higher dose (initial, maximum and cumulative) of facultative pigmentation-effective photons, there would have been a higher level of (natural) facultative pigmentation (a ñtanò).

    a. **Conclusion. This statement is confirmed by the fact that when Cripps [4] utilized a higher dose (initial, maximum, average and cumulative), the TUVR (SPF equivalent) was 8.01 but only 2.45 when a lower dose was administered.**

4. When the three most common (FDA, Health Canada and CIE) regulatory schemes [13,14,15] are used to control the dose (initial, maximum and cumulative) of facultative pigmentation-effective photons the average TUVR (SPF equivalent) is 6.5.

    a. **Conclusion. A skin type 2 individual who has a "sunburning time" of 10 minutes with the protection afforded by their (natural) constitutive pigmentation can increase their sunburning time to 65 minutes with a 3.4 MED dose or 76 minutes with a 4.0 MED dose.**

5. When the regulatory scheme adopted by Australia and New Zealand, i.e. the AU/NZ Sunlamp Standards, [16] is used to control the dose (initial, maximum and cumulative) of facultative pigmentation-effective photons, the TUVR (SPF equivalent) is only 1.6.

    a. **Conclusion. Thus, a skin type 2 individual who has a "sunburning time" of 10 minutes when they have not developed (natural) facultative pigmentation above the protection afforded by their (natural) constitutive pigmentation will only be able to increase their sunburning time to 16 minutes.**

6. A skin type 2 individual with a ñsunburning timeò of 10 minutes before applying a SPF 15 (artificial) sunscreen expects to have 150 minutes of protection but what they actually get in the ñreal worldò is only 20 minutes (25% dose / 0.5 mg/cm^2) or 30 minutes (39% dose / (0.76 mg/cm^2) of protection.

    a. **Conclusion. The "false sense of security" that people have because of the wide difference between the "expected" coverage and the "actual" coverage is one of the main reasons they sunburn outdoors even though they used a sunscreen.**

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7. When a skin type 2 individual who has a ñsunburning timeò of 10 minues before applying a SPF 100 (artificial) sunscreen expects to have 1,000 minutes (16.7 hours) of protection but the ñreal worldò protection time is only 32 minutes (25% dose / 0.5 mg/cm^2); or, 49 minutes (39% dose / (0.76 mg/cm^2).

    a. **Conclusion. The "false sense of security" that people have because of the wide difference between the "expected" coverage and the "actual" coverage is one of the main reasons they sunburn outdoors even though they used a sunscreen.**

8. However, the development of (natural) facultative pigmentation in the carefully controlled environment found in a sunbed results in protection of 65 minutes (3.4 MED dose) to 76 minutes (4.0 MED dose).

    a. **Conclusion. The level of (natural) facultative pigmentation is directly proportional to the dose (initial, maximum and cumulative) administered.**

9. The Kato, et al, study [x] showed that our (natural) constitutive and facultative pigmentation ╖but not the use of a sunscreen ╖prevented the formation of urinary 8-OHdG (8-hydroxy-2ô-deoxyguanosine), a substance known to be a critical biomarker of carcinogenesis as well as oxidative DNA damage**.**

    a. **Conclusion. This is the first study showing that (artificial) sunscreens do not have the same ability to protect the skin from UVR damage that our (natural) constitutive and facultative pigmentation has.**

10. The Miller [8,9] and Yamaguchi [x] studies showed that there was a ñdose dependentò reduction in molecular markers (p53 expression and CPD / cyclobutane pyrimidine dimer formation) as the dose (initial, maximum and cumulative) increased to 19, 29 and 42 SED.

    a. **Conclusion. Controlled exposure to facultative pigmentation-effective photons increases an individual's TUVR (SPF equivalent) in a "dose dependent" manner and also protects against p53 expression and DNA (CPD) damage formation in a "dose dependent" manner. Thus, the protection afforded by (natural) facultative pigmentation goes beyond the prevention of sunburning.**

11. The primary (and perhaps only) purpose of the UVA filters in broad-spectrum (artificial) sunscreen is to interrupt the bodyôs normal, natural and contravolitional (taking place without conscious thought or action) mechanism to protect the skin from damage, i.e., the development of (natural) facultative pigmentation.

    a. **Conclusion. A sunscreen containing UVA filters prevents the body from tanning and no study has yet looked at the "unintended consequences" of preventing the development of (natural) facultative pigmentation**.

12. Several scientific studies [30,31,32,33,34] have shown that sunbeds emit a (controlled) high level of vitamin D-effective photons.

    a. **Conclusion. When compared to (uncontrolled exposure) sunlight, (controlled exposure) sunbeds are the ideal (and most convenient) year-round source of vitamin D-effective photons.**

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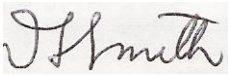

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