Phototoxicity Occurring During the Manufacture of Ultraviolet-Cured Ink

Edward A. Emmett, MB, BS; Barbara R. Taphorn; John R. Kominsky, MS

• **Four workers employed in the manufacture of ultraviolet-cured inks complained of photosensitivity characterized by an intense burning sensation during sun exposure. Three of these workers developed dermatitis on exposed areas following sun exposure. Six compounds used as photoinitiators in the ink formulations were found to absorb solar ultraviolet radiation. Two preparations of mixed isomers (ortho and para) of amyl dimethylaminobenzoate were found to be phototoxic to Ehrlich ascites cells in vitro and to produce diphasic phototoxic reactions in vivo after topical application on symptomatic workers, asymptomatic workers, or previously unexposed subjects. These responses could be prevented in two subjects by the application of a 10% sulizobenzone sunscreen prior to sun exposure. Two other photoinitiators, Michler's ketone and thioxanthone were phototoxic in vitro but not after topical application in vivo.**

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Several men employed in the formulation of ultraviolet (UV)-cured inks complained of photosensitivity. In this article, we describe the investigation of these complaints and the finding that two preparations of mixed isomers of amyl dimethylaminobenzoate designated as absorber A and absorber B in the form in which they were used in this process were able to induce phototoxic responses both in vitro and after applications to human skin. Two other materials, thioxanthone and Michler's ketone were phototoxic in vitro, but a phototoxic response could not be demonstrated from topical application in vivo.

ULTRAVIOLET-CURED INKS

Ultraviolet cured inks are of relatively recent development but their use is becoming widespread. The ink composition varies but usually consists of one or more conventional pigments dispersed in a polymeric vehicle. The vehicle usually contains the following: (1) various polyfunctional acrylic monomers such as trimethylol propane triacrylate or pentaerythritol triacrylate, alone or in combination with monofunctional acrylic monomers; (2) an UV reactive unsaturated polymer such as acrylated urethane polyester oligomer, acrylated epoxy resin oligomer, and a variety of other types of acrylic base polymers; (3) one or more photoinitiators such as benzophenone, or isomers of amyl dimethylaminobenzoate; (4) diluents such as primary and aliphatic alcohols or phthalates; (5) hydrogen transfer agents such as triethanolamine; and (6) a variety of miscellaneous agents, including stablizers, surfactants, fillers, flattening agents, and polymerization inhibitors.

The first step in the curing process is the absorption of UV radiation by the photoinitiator (such as benzophenone). The absorption of UV results in the generation of free radicals or other photoexcited states, which in turn cause polymerization of the vehicle in which the pigments are incorporated and thus cure of the ink film. Medium pressure mercury arc lamps are generally used for the cure.

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From the Department of Environmental Health, University of Cincinnati, (Dr Emmett and Ms Taphorn), and the National Institute for Occupational Safety and Health (Mr Kominsky), Cincinnati.

Reprint requests to Department of Environmental Health, Room 5251, College of Medicine, 231 Bethesda Ave, Cincinnati, OH 45267 (Dr Emmett).

Table 1.—Clinical Features in Four Employees Complaining of Photosensitivity

Characteristic	Employees 1	2	3	4
Symptoms	Burning on exposed areas within 5-10 minutes' sun exposure; exposure and swelling (lasting 2-3 days) after sun exposure	Burning on exposed areas within 5-10 minutes' sun exposure; erythema and swelling occur within a few hours of exposure	Burning on exposed areas within a few minutes of sun exposure, including sunlight through car window	Burning on exposed areas within a few minutes of sun exposure
Observed morphology	Erythema with sharp margin on V of neck and dorsal aspects of hands; lichenification of forearm	Erythema with fairly sharp margin on dorsal surface of both forearms	Erythema with sharp margins on forearms, outer surface of ears, and V of neck, sparing submental area	None
History of preexisting dermatitis	History of recent vesicular eruption on arms; severe exacerbation following sun exposure	No preexisting dermatitis	Recent intermittent itching, erythema, swelling, and scaling of forearm	Dermatophyte infection of feet
Results of closed patch testing with ink resin components*	Pentaerythritol triacrylate 0.2% in petrolatum: + +* Trimethylpropane triacrylate 1% in petrolatum: + + + Hexanediol diacrylate 1% in petrolatum: + + epoxy acrylate 1% in petrolatum: + +	No positive reactions	Pentaerythritol triacrylate 0.2% in petrolatum: + + Trimethylpropane triacrylate 1% in petrolatum: + + Hexanediol diacrylate 1% in petrolatum: + + Epoxy acrylate, 1% in petrolatum: +	No positive reactions

*Grading of reactions performed, using the scale recommended by the International Contact Dermatitis Group (1): negative reaction, —; doubtful reaction, ?+; weak (nonvesicular) reaction, +; strong (edematous or vesicular) reaction, + +; and extreme reaction, + + +.

CLINICAL FEATURES

Four white men, aged from 26 to 52, employed weighing, mixing, or milling UV-cured inks, complained of sensitivity to sunlight. The clinical characteristics in these subjects are given in Table 1. Subjective complaints included burning (smarting) on exposed areas occurring within a few minutes of sun exposure, persisting until the exposure ceased and erythema and swelling occurring within minutes to hours of sun exposure and lasting for up to several days. In two of the men, there was evidence of eczematous dermatitis, in both cases apparently due to allergic contact sensitization to certain acrylates used in the UV-cured ink formulation. The results of patch testing with these materials are shown in Table 1. In both men, the occurrence of marked smarting on sun exposure suggested additional photosensitivity, in one subject additional severe exacerbations of his eczematous lesions were described following sun exposure while gardening.

The unprotected skin and clothes of these men were frequently occupationally contaminated with components of the UV-ink. In no case was there a history of taking any known photosensitizing drug within the last year, a family history of photosensitivity, or clinical evidence of any disease known to be associated with photosensitivity. In each case, the burning sensation on sun exposure had not been present prior to exposure to UV-cured ink components and appeared temporally related to the manufacture of ultraviolet-cured ink.

INVESTIGATION OF PHOTOSENSITIVITY METHODS

Absorption Spectra.—In order to determine which of the materials used in the UV-cured ink formulation might cause photosensitization, the UV absorption spectra of all ingredients were determined in ethanol using a double beam spectrophotometer (Coleman 124).

In Vitro Phototoxicity.—Each of the UV-cured ink ingredients that absorbed UV radiation above 290 nm was evaluated for phototoxicity in an Ehrlich ascites cell system.

Ehrlich ascites cells were harvested from a mouse eight to ten days after implantation. After an initial staining with trypan blue and counting to ensure that the cells were viable, the cells were washed twice in lactated Ringer's solution before being put in suspension at a concentration of 2×10^6 cells/ml. Four sets (A, B, C, and D), each of two cell suspensions, were then studied. Sets A and B were incubated with the test material in the dark for two hours at room temperature, sets C and D were similarly incubated with the Ringer solution vehicle. After incubation, the cells were washed twice before being resuspended in lactated Ringer's solution in small Petri dishes. Sets A and C were then irradiated at a room temperature of 24 C, while sets B and D remained in the dark. The percentage of dead cells in each sample was determined 24 hours later by staining with the nonvital dye trypan blue. Cells were stained with this dye in the dark for at least two minutes before readings were made. Duplicate readings of at least 200 cells each were performed on each cell suspension. The mean and variance was calculated for the four percentage counts obtained on each set of results and the results for the different sets compared by the analysis of variance of a two by two factorial. In this way, it could be determined whether an interaction between the irradiation and the incubated chemical had occurred. Significance was determined using a two tailed t-test. The phototoxicity assay was recorded as positive when a statistically significant ($P < .05$) interaction between irradiation and the incubated chemical was determined in the above manner. The cells were irradiated either for ten minutes at a distance of 30 cm from a bank of four sunlamps (predominantly 293 to 325 nm radiation) or for one hour at a distance of 30 cm from a bank of four BLB40 black lights (predominently 330 to 380 nm radiation). The radiant exposure from the sunlamps measured with an IL700 spectroradiometer detector and narrow-band filter with a maximum spectral response at 297 nm and half power points at 291 and 303 nm was 9.4×10^{-6} joules/sq m. The

radiant exposure from the black lights measured with a IL700 spectroradiometer and detector with a wide-band filter with maximum response at 365 nm and half power points 330 and 400 nm was 4.1×10^{-4} joules/sq m. This exposure from the bank of sunlamps was sufficient to produce a slight decrease in the viability of control cells, whereas no alteration in the control cells was seen following this exposure from the blacklights. Where the compounds were not readily soluble in water, 5% ethanol in lactated Ringer's solution was used in place of lactated Ringer's solution for the incubation step. For each test, preliminary incubations at varying concentrations of the test material with the Ehrlich ascites cells were made in order to determine nontoxic concentrations for incubation.

Photopatch Testing.—Photopatch testing was performed on three employees (subjects 1 through 3), who complained of photosensitivity and four employees free of photosensitivity using the following UV-cured ink components: benzophenone, 5% in petrolatum; diethoxyacetophenone, 5% in petrolatum; thioxanthone, 5% in petrolatum; Michler's ketone, as is; absorber A, 5% in petrolatum; absorber B, 5% in petrolatum; absorber A, as is; and absorber B, as is.

Ink components used for photopatch testing were applied evenly to the central gauze portion of adhesive plastic strips 3.8 sq cm in size before application to the skin. Two duplicate sets of patches were applied to the back and were covered, secured, and occluded with several layers of hypoallergenic surgical tape. One set of patches was removed 24 hours after application and the area gently cleansed with 70% ethanol in water. These sites were then exposed to sunlight. Four employees working a day shift were exposed to 25 minutes of clear noon, July, Cincinnati (39 degrees north) sunlight and three employees to 35 minutes of sunlight from 3:50 PM that same day. Reactions were observed during the irradiation. The second set of patches that functioned as a dark control for the development of allergic contact or irritant reactions was removed 48 hours after application and the site gently cleansed with 70% alcohol. Reactions at both sites were read and graded one hour later.

In two subjects complaining of photosensitivity, the minimal erythema dose (MED) was determined as previously described,[2] an area 5 × 10 cm was irradiated with four times the MED and photopatch testing performed with a routine tray of diphenhydramine hydrochloride (Benadryl) cream; bergamot oil, 10% in petrolatum; bithionol 1% in petrolatum; 4'5 dibromosalicylanilide, 1% in petrolatum; eosin, as is; hexachlorophene, 0.5% in petrolatum; promethazine hydrochloride (Phenergan Cream), as is; tripelennamine (Pyribenzamine), 1% in petrolatum; tribromosalicylanilide, 1% in petrolatum; trichlorocarbanalide, 0.5% in petrolatum; triflurocarbanalide, 0.5% in petrolatum; and ragweed oleoresin.

In Vivo Phototoxicity and Photoprotection.—Two patches each of purified amyl *p*-dimethylaminobenzoate—absorber A and absorber B were applied as described above to the backs of four white subjects, aged 25 to 35, who had no previous industrial exposure to amyl *p*-dimethylaminobenzoates. Purified amyl *p*-dimethylaminobenzoate was also examined as were the two absorbers represent mixed isomers (para and ortho) dimethylaminobenzoate. After 24 hours one set of patches was removed and the area lightly cleansed with 70% ethanol in water while the second set was covered with an additional layer of black plastic tape. Twenty minutes after removal of the first set of patches, subjects were exposed to clear, noonday, Cincinnati (39 degree north) sunlight on Aug 5, 1975, for 30 minutes. Immediately afterwards, the second, previously occluded set of patches was removed and the area similarly cleansed with 70% ethanol in water. On three subjects, a third and fourth set of patches were applied that were also removed and the areas cleansed 20 minutes before sun exposure. The third set was immediately evenly covered with 2.5 μl/sq cm of a sunscreen containing 5%-*p*-aminobenzoic acid in alcohol (Presun) and the fourth evenly covered with 2.5 μl/sq cm of a sunscreen containing 10% sulisobenzone (Uval) 15 minutes prior to sun exposure. The reactions were observed during sun exposure, hourly for 5 hours and again 48 and 72 hours after patch application. No subjects had further sun exposure over this observation period.

RESULTS

Six photoinitiators used in UV-cured inks were found to absorb UV radiation above 250 nm namely benzophenone, thioxanthone, 2,2-diethoxyacetophenone, 4,4'-bis (dimethylamino) benzophenone (Michler's ketone), and two different commercial preparations of industrial grade mixed isomers of amyl dimethylaminobenzoate designated as absorber A and absorber B. The absorption peaks of these substances in alcohol are shown in Table 2. Absorber A had an absorption peak at 310 nm (ϵ -15,000) with one half the peak absorption at 288 and 326 nm, absorber B an absorption peak at 310 nm (ϵ = 20,000) with one half the peak absorption at 289 and 325 nm. Pharmaceutical grade amyl *p*-dimethylaminobenzoic acid had an absorption peak at 306 nm (ϵ = 30,000) with one half the peak absorption at 287 and 322 nm. Amyl ortho dimethylaminobenzoic acid has an absorption peak at 344 nm.

The results obtained from the in vitro phototoxicity assay of these substances using Ehrlich ascites cells are shown in Table 3. It will be seen that benzophenone and diethoxyacetophenone were not demonstrably phototoxic whereas Michler's ketone, thioxanthone, and the two absorbers containing mixed isomers of amyl dimethylaminobenzoate were phototoxic in this system. Both light sources, sunlamps and black lights, produce comparable results. Purified amyl *p*-dimethylaminobenzoate and *p*-aminobenzoate were also examined for phototoxicity in the same manner. As seen from the table, the amyl *p*-dimethylaminobenzoate was phototoxic whereas *p*-aminobenzoic acid was not. No phototoxicity was seen whether *p*-aminobenzoic acid was examined dissolved either in lactated Ringer's solution or 5% alcohol in lactated Ringer's solution.

The results of phototesting observed on three employees complaining of photosensitivity (subjects 1, 2, and 3 of Table 1) and four other employees not complaining of photo-

Table 2.—Absorption Peaks Above 250 nm of Certain Photoinitiators Used in UV-Cured Ink Formulations

Benzophenone	252, 338 nm
Thioxanthone	257, 286, 299, 379 nm
Diethoxyacetophenone	250, 330 nm
Michler's ketone	370 nm
Absorber A	310 nm
Absorber B	310 nm

Table 3.—In Vitro Phototoxicity of Photoinitiators Used in UV-Cured Inks and of Certain Other Chemically Related Substances

Test Substance	Concentration	Black Light (330-380 nm)	Sunlamp (293-325 nm)
Benzophenone	5×10^{-5} M	Negative	Negative
Thioxanthone	5×10^{-6} M	Positive ($P < .001$)	Positive ($P < .005$)
Diethoxyacetophenone	5×10^{-5} M	Negative	Negative
4,4′-bis (dimethylamino) benzophenone (Michler's ketone)	5×10^{-5} M	Positive ($P < .001$)	Positive ($P < .001$)
Absorber A (mixed isomers of amyl	5×10^{-5} M	Positive ($P < .001$)	Positive ($P < .001$)
p-Dimethylaminobenzoate)	5×10^{-6} M	Positive ($P < .02$)	
Absorber B (mixed isomers of)	5×10^{-5} M	Positive ($P < .001$)	Positive ($P < .001$)
Amyl *p*-dimethylaminobenzoate	5×10^{-6} M	Positive ($P < .001$)	
p-Aminobenzoate	5×10^{-5}	Negative	Negative
Amyl-*p*-dimethylamino	5×10^{-5} M	Positive ($P < .001$)	Positive ($P < .001$)
benzoate	5×10^{-6} M	Positive ($P < .05$)	

Table 4.—Reactions at Sun Exposed Sites*

	Subjects						
	1	2	3	5	6	7	8
Absorber A during sun exposure	+S	+S	?+S	?+S	+S	+S	—
24 hr after sun exposure	++	—	+	++	+	+	+
Absorber B during sun exposure	+S	+S	?+S	?+S	+S	—	—
24 hr after sun exposure	—	—	+	+	—	—	—

*S indicates smarting reaction during sun exposure. Grading of visible reactions was performed, using the scale recommended by the International Contact Dermatitis Group[1]: negative, —; doubtful reaction, ?+; weak (nonvesicular) reaction +; strong (edematous or vesicular) reaction, ++; and extreme reaction, +++. Subjects 1, 6, 7, and 8 were exposed to noonday sun and subjects 2, 3, and 5 to afternoon sun.

Table 5.—Reactions Observed in Four Normal Volunteers at Completion of Sun Exposure and 24 Hours After Sun Exposure*

	At Completion of Sun Exposure Subjects				24 Hours After Sun Exposure Subjects			
Treatment	1	2	3	4	1	2	3	4
Kept in dark								
Purified amyl p-dimethylaminobenzoate	—	—	—	—	—	—	—	—
Absorber A	—	—	—	—	—	—	—	—
Absorber B	—	—	—	—	—	—	—	—
No treatment	—	—	—	—	—	—	—	—
30 Minute sun exposure								
Purified amyl p-dimethylaminobenzoate	2	—	—	—	1	—	—	—
Absorber A	3	2	—	2	2	2	—	2
Absorber B	3	2	—	2	2	2	—	2
No treatment	—	—	—	—	—	—	—	—
30 Minute sun exposure +5% *p*-aminobenzoic acid screen								
Purified amyl p-dimethylaminobenzoate	—	—	—	NT	—	—	—	NT
Absorber A	2	1	—	NT	2	1	—	NT
Absorber B	2	1	—	NT	2	1	—	NT
No treatment	—	—	—	NT	—	—	—	NT
30 Minute sun exposure +10% sulisobenzone screen								
Purified amyl p-dimethylaminobenzoate	—	—	—	NT	—	—	—	NT
Absorber A	—	—	—	NT	—	—	—	NT
Absorber B	—	—	—	NT	—	—	—	NT
No treatment	—	—	—	NT	—	—	—	NT

*Readings were recorded according to the criteria: No reaction, —; mild erythema with indistinct borders, 1; marked erythema with distinct borders, 2; erythema with edema, 3; erythema, edema and vesiculation, 4; and not tested, NT.

sensitivity are shown in Table 4. The majority of subjects complained of a sharp stinging or burning sensation within a few minutes of sun exposure localized to the exposed areas where absorber A as is, and absorber B, as is, had been applied. Four subjects developed sharply marginated erythema and two faint or questionable erythema responses on the absorber A site during sun exposure and three subjects sharply marginated erythema and two faint questionable erythema at the absorber B site during sun exposure. Twenty-four hours after sun exposure, six of the seven men (including subject 7, a deeply pigmented black) had a reaction consisting of uniform erythema and edema on the area exposed to undiluted absorber A and sunlight; two of the seven men also had reactions to undiluted absorber B under the same conditions. No reactions were seen to absorbers A or B on sites occluded from sunlight. No reactions were seen with the other UV ink components phototested either on exposed or occluded sites, apart from a single weak reaction to Michler's ketone on a patch test site occluded from sunlight that was interpreted as an irritant reaction. No reactions were seen on sun-exposed or control sites tested with either absorber A or absorber B diluted to 5% in petrolatum. Photosensitive subjects 2 and 3 had further testing. Both had a normal minimal erythema dose; negative photopatch testing with a routine tray, no demonstrable circulating antinuclear antibody, and no abnormal reaction at 24, 48, or 168 hours to irradiation with

four times the minimal erythema dose of UVB.

In order to examine the characteristics of the phototoxic responses to absorber A and absorber B more closely, further observations were made on the phototoxic responses in four individuals without previous known industrial exposure to these materials. Observations were also made on the photoprotective effect of sunscreens representative of two different classes of sunscreens in three of these individuals. Three of the four subjects noted a sharply localized intense burning sensation that commenced a few minutes after the areas patch tested with absorbers A and B were exposed to sunlight. No burning occurred on the covered areas. The burning sensation was accompanied by erythema, and in one case, prominent edema. One subject also developed erythema and a burning sensation at the site of application of the purified amyl *p*-dimethylaminobenzoate. In each case, the burning stopped within a few minutes of the cessation of sun exposure.

The reaction was observed to be diphasic, the erythema and swelling began to fade shortly after sun exposure ceased and had disappeared in two subjects within one hour and in the third, more severely affected subject, two hours after sun exposure ceased. Four to five hours after the sun exposure, erythema recurred and remained for two to five days. Mild discomfort and irritation accompanied the reappearance in each case, but the intense burning noted during sun exposure was not evident. The reactions noted in each subject immediately following and 24 hours after sun exposure are shown in Table 5.

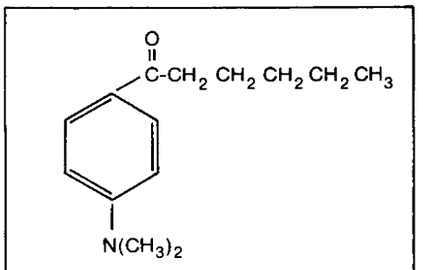
Two of the three subjects to whom sunscreens were applied developed a phototoxic response so that the degree of photoprotection could be estimated. As seen from Table 5, the prior application of a 5% *p*-aminobenzoic acid sunscreen provided partial protection against the reaction whereas the application of a 10% sulisobenzone sunscreen was able to completely suppress the development of the phototoxic response under the conditions of exposure.

COMMENT

The two industrial preparations of mixed isomers of amyl dimethylaminobenzoate designated as absorber A and absorber B were demonstrated to be phototoxic with either UVA (280 to 315 nm) or UVB (315 to 400 nm) to Ehrlich ascites cells in vitro and with sunlight to human skin in vivo. The phototoxic reaction could be reproduced even in deeply pigmented black skin. The phototoxic nature of the in vivo responses was indicated by the relative immediacy of the response to sunlight, the intense burning sensation on exposure to sunlight, the ability to reproduce the reaction in other employees not complaining clinically of photosensitivity, as well as in previously unexposed persons, and the failure of reactions to occur when these substances were diluted. The chemical structure of amyl *p*-dimethylaminobenzoate is shown in the Figure. Although Michler's ketone was noted to be phototoxic in vitro, a phototoxic response to this substance could not be reproduced by topical application to human skin and subsequent exposure to sunlight. Thioxanthone also gave a phototoxic response in vitro, but not at 5% concentration in vivo and was not studied further as it had rarely been used in the industrial process.

Two of the four employees complaining of photosensitivity also had allergic contact sensitization to acrylic monomers and epoxy acrylate oligomers to which they had been exposed. However, sun exposure of skin involved in allergic contact dermatitis does not usually provoke the very intense burning sensation and the exacerbations of dermatitis that these subjects described.[3,4] Their photosensitivity seems better explained as clinical phototoxic reactions to the undiluted absorbers A and B that they handled during their work.

It has been previously suggested[5] that the allergenicity of benzyl salicylate, a weak contact sensitizer, may have been enhanced by the phototoxic effect of simultaneously applied topical methoxsalen. The same effect may have enhanced the allergenicity of the acrylate monomers and epoxy acrylate oligomers in this instance. One



Amyl p-dimethylaminobenzoate

Chemical structure of amyl dimethylaminobenzoate.

could also speculate that the simultaneous allergic contact dermatitis in several of these employees with attendant damage to the epidermal barrier might have increased the penetration of the phototoxic materials and thus rendered the phototoxic response more easily elicitable.

The phototoxic reactions observed on human skin after experimental application of the two industrial absorbers were noted to be diphasic. The burning sensation (smarting reaction) described during sun exposure and the ensuing dermatitis observed by employees appear to parallel the two phases. The initial intense smarting reaction on sun exposure has been noted in other phototoxic reactions, such as those occurring in pitch workers[6] and is common to all crude coal tars. In addition, demeclocycline hydrochloride (Declomycin) phototoxicity is associated with paraesthesiae during sun exposure, and burning and itching during sun exposure are commonly observed in patients with erythrocytic protoporphyria. In the case of the mixed isomers of amyl dimethylaminobenzoate, the initial burning was also accompanied by erythema and sometimes transient wheal formation confined to the site of the reaction. This reaction disappeared completely before the recurrence of erythema and discomfort some hours later.

The phototoxic reactions observed with the two industrial preparations of undiluted mixed isomers of amyl dimethylaminobenzoate was somewhat surprising as purified amyl *p*-dimethylaminobenzoate is demonstrably effective at a 5% concentra-

tion as a sunscreen.[7] No phototoxicity was observed in these employees when a 5% concentration of amyl *p*-dimethylaminobenzoic acid was applied. A difference between protective and toxic responses depending on the circumstances of use would seem to illustrate the important toxicologic principle that the hazard from any material is highly dependent on the circumstances of its use. The phototoxic reaction on human skin with pure amyl *p*-dimethylaminobenzoate was less marked than with the mixed isomer preparations suggesting that other isomers may be the most important phototoxic agents. However, pure amyl *p*-dimethylaminobenzoate could be observed in vitro to be phototoxic, whereas neither benzophenone nor *p*-aminobenzoic acid were observed to be phototoxic in vitro.

The phototoxic effect observed on the skin of volunteers could be blocked by the prior topical application of a 10% sulisobenzone (2-hydroxy-4-benzophenone-5 sulphonic acid) preparation but only partially blocked by a similar application of 5% *p*-aminobenzoic acid in alcohol. This suggests that sulizobenzone preparations which are effective screens in the UVA as well as the UVB range[1] may prove useful sunscreening agents for use in accidental skin contamination by the amyl dimethylaminobenzoate esters. The relative ineffectiveness of *p*-aminobenzoic acid (absorption maximum in water 266 nm, absorption extending to 310 nm) presumably depends on its ineffectiveness against the wavelengths of 310 and greater. Although it has been held that only physical sunscreens such as zinc oxide, titanium dioxide, or red veterinary petrolatum are effective against industrial phototoxic agents,[8] there seems to be no good reason why a sunscreen eliminating the requisite wavelengths cannot satisfactorily block phototoxic responses as has been recently demonstrated in the case of vinblastine.[9]

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Nonproprietary Names and Trademarks of Drugs

Tribromsalan—*Cuticura Soap, Lifebuoy, Praise, Temasept IV, Tuasal.*

Triclocarban—*Dial Soap, Creed Bar Soap, Jergens Deodorant Soap, Tackle Medicated Soap, TCC.*

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