Chemical Exposures, Medical History, and Risk of Squamous and Basal Cell Carcinoma of the Skin[1]

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Abstract

The role of non-sunlight-related risk factors for squamous cell carcinoma (SCC) and basal cell carcinoma (BCC) of the skin was investigated in a population-based, case-control study conducted among males in Alberta, Canada. In total, 180 SCC and 226 BCC cases and 406 randomly selected male controls, frequency matched by 5-year age groups to the cases, were interviewed by trained personnel using a standardized etiological questionnaire. Data were analyzed using conditional logistic regression techniques. After adjustment for age, skin and hair color, mother's ethnic origin, and sunlight exposure, elevated risks for SCC were seen in subjects exposed to insecticides [odds ratio (OR), highest tertile, 2.8; 95% confidence interval (CI), 1.4–5.6], herbicides (OR, highest tertile, 3.9; 95% CI, 2.2–6.9), and fungicides and seed treatments (OR, highest tertile, 2.4; 95% CI, 1.4–4.0), as well petroleum products, grease, and several other exposures. Elevated risks of BCC were seen in subjects exposed to fiberglass dust (OR, 2.0; 95% CI, 1.0–3.9) and dry cleaning agents (OR, 4.6; 95% CI, 1.1–19.7). Prior nondiagnostic X-ray treatment for skin conditions increased risk of both cancers. Although solar UV radiation is known to be the major environmental exposure causing nonmelanocytic skin cancer, results of this study suggest that nonsolar factors may also be important.

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Introduction

BCC[3] and SCC together constitute the most common type of cancer seen in white populations (1–3). Much of the incidence of these diseases is known to be due to exposure to solar UV radiation (4), but little information has been collected in a systematic manner that would permit the examination of other, nonsolar risk factors for these common cancers.

To identify both solar and nonsolar risk factors, a case-control study of nonmelanocytic skin cancer was conducted in the province of Alberta, Canada. This communication reports results for nonsolar risk factors.

Materials and Methods

Pathological reports on all male BCCs and SCCs diagnosed in the province of Alberta from January 1, 1983, through December 31, 1984, were obtained from the Alberta Cancer Registry, a population-based registry covering the entire province. Male cases were selected because their more diverse occupational histories were thought to be more useful in investigating both solar and nonsolar risk factors for these diseases. A selected group of Alberta male cases with first primary BCC (every fourth male registered with a BCC on the head or neck and every male with BCC at any other anatomical site) as well as all male cases with first primary SCC were invited to participate in the study, provided they were age 20–79 years at the time of diagnosis.

The age-eligible sample consisted of 314 BCC and 225 SCC patients. Informed consent was obtained, and successful etiological interviews were conducted with 72% (226) of the BCC and 80% (180) of the SCC patients.

A common male control series was randomly chosen from the Alberta Health Care Insurance Plan subscriber files. This prepaid, government-sponsored insurance plan covered virtually all residents of the province, with the exception of those who resided in Alberta for less than 3 months. The controls had no prior BCC or SCC and were age matched to the cases within 5-year age groups. In total, 573 age-eligible controls were invited to participate in the study, and full etiological interviews were completed on 71% (406) of these.

Each case and control was interviewed in his home by a trained interviewer using a standardized questionnaire similar to that used in the Western Canada Melanoma Study conducted from 1979 to 1981 in the western provinces of Canada (5–7). Interviewers were not informed of study hypotheses and were blinded to the disease status of subjects that they were sent to interview. All cases and controls were interviewed by the end of 1985.

Phenotype and pigmentation factors evaluated in the course of the interview included non-sun-exposed skin color

[3] The abbreviations used are: BCC, basal cell carcinoma; SCC, squamous cell carcinoma; OR, odds ratio; CI, confidence interval.

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Table 1 Medical history and risk of BCC and SCC: 226 BCC, 180 SCC, and 406 controls[a]

Factor	No. of controls	No. of BCC cases	No. of SCC cases	Adjusted OR, BCC[b]	95% CI	Adjusted OR, SCC[b]	95% CI
Skin cancer in mother or father							
Absent	397	211	167	1.0		1.0	
Present	9	15	13	2.2	0.9–5.4	4.5	1.6–12.4
				$P = 0.09$		$P = 0.004$	
Rheumatoid arthritis							
Absent	392	217	165	1.0		1.0	
Present	14	9	15	1.5	0.6–3.9	3.3	1.4–7.5
				P = ns		$P = 0.006$	
Nondiagnostic X-ray							
Never	398	214	166	1.0		1.0	
Ever	5	12	12	5.7	1.7–19.1	4.8	1.5–16.0
				$P = 0.005$		$P = 0.01$	
Ultraviolet lamp treatments							
Never	386	219	172	1.0		1.0	
Ever	13	7	7	1.0	0.3–2.7	0.7	0.2–2.3
				P = ns		P = ns	
Acne requiring treatment							
Never	373	206	168	1.0		1.0	
Ever	33	20	12	0.8	0.4–1.6	1.0	0.5–2.1
				P = ns		P = ns	
Psoriasis requiring treatment							
Never	392	218	172	1.0		1.0	
Ever	14	8	8	1.0	0.4–2.5	1.3	0.5–3.4
				P = ns		P = ns	
Prior mumps							
No	66	50	45	1.0		1.0	
Yes	292	157	115	0.7	0.5–1.2	0.6	0.4–1.0
				P = ns		P = ns	
Immunized as child?							
No	162	80	99	1.0		1.0	
Yes	244	146	81	0.9	0.6–1.3	0.6	0.4–0.9
				P = ns		$P = 0.02$	

[a] Numbers of cases and controls may be reduced in specific analyses due to missing data on medical history.
[b] ORs adjusted for age, skin color, hair color, and mother's ethnic origin; ns, not significant.

assessed using a five-step skin color panel, hair color using a wig maker's samples, and eye color assessed by direct observation. In addition, information on a propensity to burn rather than tan in the sun, sunburn history, degree of freckling in childhood, and tanning history was collected. Solar UV exposure was assessed for recreational and occupational pursuits in childhood, adolescence, and each decade of adult life. Results for phenotype and solar UV relationships have been published (8, 9). Information collected on other factors included: prior chronic diseases; viral and skin diseases; family history of cancer; exposure to a list of specific substances (*e.g.*, pesticides and tar products), whether in the workplace, home, or hobby setting; and history of smoking. A complete occupational history was taken, with information on job title, industry, and dates started and stopped for each occupation held 6 months or longer.

Because age is related to the risk of skin cancer and to the duration of exposure to both solar UV radiation and other environmental risk factors, all analyses were stratified by single year of age to control potential age confounding. Initial crude univariate ORs were calculated for each variable of interest, adjusting only for age. Conditional logistic regression techniques were then used to analyze significant variables, adjusting for skin and hair color, mother's ethnic origin, and sunlight exposure, because previous analyses have demonstrated that these are important in accounting for BCC and SCC risk (8, 9).

Results

Histories of previous skin cancer in parents of subjects conferred an elevated risk of both BCC and SCC (Table 1). Subjects with rheumatoid arthritis appeared to be at higher risk of SCC than those without, although no similar association was seen for BCC. Nondiagnostic X-rays, mostly used to treat dermatitis and other nonspecific skin disorders, conferred an elevated risk of both nonmelanocytic skin cancers. No elevated risk was seen for either tumor in subjects with prior acne or psoriasis serious enough to require medical treatment. Two unexpected findings appeared. First, a suggestion of decreased risk of both SCC and BCC in subjects who had mumps in childhood was found, although these associations were not statistically significant. In addition, subjects who were immunized against polio, diphtheria, or tetanus in childhood appeared to be at lower risk of SCC, although a similar reduced risk was not seen for BCC. No association with cigarette smoking was seen for SCC or BCC.

The occupational history delineating each job held for 6 months or more was analyzed to evaluate whether work in specific occupations increased the risk of either type of nonmelanocytic skin cancer. The results were largely unremarkable (Table 2), with an excess of BCC seen in social services and allied professions, although there were relatively small numbers of subjects in these occupations. A decreased risk of SCC was seen in clerical workers, and a similar suggestion of decreased overall risk was also observed in nongovernmental managers

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Table 2 Occupation and risk of BCC and SCC: 226 BCC, 180 SCC, and 406 controls

Occupation	No. of controls	No. of BCC cases	No. of SCC cases	Adjusted OR, BCC[a]	95% CI	Adjusted OR, SCC[a]	95% CI
Social sciences, social work							
Never	397	218	174	1.0		1.0	
Ever	9	8	6	3.4	1.1–9.9	1.6	0.5–5.2
				$P = 0.03$		P = ns	
Clerical worker							
Never	319	189	154	1.0		1.0	
Ever	87	37	26	0.8	0.5–1.2	0.5	0.3–0.8
				P = ns		$P = 0.01$	
Nongovernment managers and administrators							
Never	348	187	162	1.0		1.0	
Ever	58	39	18	1.1	0.7–1.8	0.6	0.3–1.1
				P = ns		$P = 0.09$	

[a] ORs adjusted for age, skin color, hair color, and mother's ethnic origin; ns, not significant.

and administrators. Interestingly, no elevated risk was seen for outdoor jobs such as farming for either BCC or SCC.

Evaluation of a list of specific substances to which subjects might potentially have been exposed suggested that a number of agents may be important in nonmelanocytic skin cancer (Table 3). Prior exposure to insecticides, herbicides, and fungicides or seed treatments appeared to be associated with an increased incidence of SCC but not BCC. Exposure to grain or coal dust was associated with an increased risk of SCC, whereas the risk of BCC was unchanged. Similarly, exposure to petroleum products (gasoline and oil), grease, and diesel fumes also appeared to increase the risk of SCC but not BCC, although some of these potential associations were not statistically significant.

Exposure to asbestos or fiberglass dust was associated with an increase in the risk of BCC but not SCC, and exposure to luminous paint was strongly related to BCC risk, although the risk estimate is based on a relatively small number of subjects. Occupational exposure to dry cleaning agents appears to confer a nonsignificant excess risk of BCC, with a suggestion of a similar effect also seen for SCC. Numbers of subjects exposed to these agents were small.

When possible, we attempted to test for the presence of a dose-response relationship between physical or chemical agents and cancer risk using an index of exposure calculated from data collected. The total duration of exposure to the time of diagnosis (time of interview in controls) in months was weighted by the source of exposure (direct job, workplace environment, hobby, or home) and intensity (<1 h/week, 1–4 h/week, 5–19 h/week, or ≥20 h/week). This was converted to lifetime h. Exposed subjects were divided into low- and high-exposure categories by dichotomizing exposure h among the controls, and these groups were then compared with subjects with no exposure.

The chemical and physical agents that demonstrated dose-response relationships with one of the non-melanocytic skin cancers did so exclusively with SCC. Because many of these agents were noted to be farm related, and because farmers may be exposed to more solar UV radiation than individuals in other occupations, it was thought that the associations detected might be due to confounding by occupational sun exposure, which was previously noted to be a risk factor for SCC (9). Therefore, a new model was fitted, adjusting for occupational sunlight exposure in addition to mother's ethnic origin and skin and hair color. Table 4 shows the associations between SCC and insecticides, herbicides, and fungicides and seed treatments. These remained statistically significant, and in most cases the dose-response gradients were strengthened by adjustment for sun exposure. The strength of the association with grain dust, however, decreased somewhat and is not statistically significant, although the dose-response gradient remains intact. Exposure to petroleum products, grease, and diesel fume also demonstrated dose-response relationships with SCC, as did exposure to coal dust.

Discussion

The results of this study indicate that factors other than solar UV radiation exposure may be important in accounting for the risk of BCC and SCC. However, because of difficulties inherent in retrospective studies, the results should be treated with caution until they are independently confirmed.

In this study, older men were asked to recall events that occurred many years in the past, thereby generating a substantial potential for misclassification in the data. However, providing that such misclassification occurs with approximately equal frequency among cases and controls, the effect should be to render the study findings conservative, because nondifferential misclassification makes it more difficult to detect a real association. There are several good reasons for believing that any misclassification present is roughly equally distributed between cases and controls. First, most of the associations with non-UV factors are unique to either BCC or SCC. It seems most improbable that for each of the significant associations between SCC and chemical exposure, for instance, that there was differential misclassification between SCC cases and the control subjects but not between BCC cases and the same control subjects. Also, because neither SCC or BCC is life threatening, rumination on the part of cases, which might lead to bias, is likely to be less problematic than would be the case with other cancers. Furthermore, because the study subjects and the interviewers were blinded to study hypotheses, and a standardized interview format was used, bias would have been unlikely to have significantly influenced subject case responses compared with control responses. Interviewers were also blinded to the disease status of study subjects at interviews.

A final concern affecting potential for bias in response is the number of cases with diagnoses of BCC or SCC of the skin occurring prior to the index cancer. A high prevalence of earlier primaries might conceivably have affected the way in which the case group answered questions on sun exposure. To evaluate the potential for such bias, all SCC were retrospectively linked in 1995 to the Alberta Cancer Registry, and a search was done for diagnoses occurring prior to January 1983. Three of 180

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Table 3 Chemical and physical exposures and risk of BCC and SCC: 226 BCC, 180 SCC, and 406 controls

Exposure	No. of controls	No. of BCC cases	No. of SCC cases	Adjusted OR, BCC[a]	95% CI	Adjusted OR, SCC[a]	95% CI
Insecticides							
Never	332	176	123	1.0		1.0	
Ever	74	50	57	1.3	0.9–2.1	1.7	1.1–2.7
				P = ns		P = 0.02	
Herbicides							
Never	277	156	101	1.0			1.0
Ever	129	70	79	1.1	0.8–1.7	1.5	1.0–2.3
				P = ns		P = 0.03	
Fungicides and seed treatments							
Never	237	150	84	1.0		1.0	
Ever	169	76	96	0.9	0.6–1.3	1.4	0.9–2.10
				P = ns		P = 0.09	
Grain dust							
Never	206	104	68	1.0		1.0	
Ever	200	102	112	1.1	0.8–1.6	1.5	1.0–2.3
				P = ns		P = ns	
Coal dust							
Never	298	159	111	1.0		1.0	
Ever	108	67	69	1.4	0.9–2.1	1.6	1.0–2.4
				P = 0.08		P = 0.03	
Petroleum products							
Never	230	138	89	1.0		1.0	
Ever	176	88	91	0.9	0.6–1.3	1.3	1.0–2.0
				P = ns		P = 0.08	
Grease							
Never	239	142	86	1.0		1.0	
Ever	167	84	94	0.9	0.6–1.3	1.4	0.9–2.1
				P = ns		P = 0.09	
Diesel fumes							
Never	266	141	97	1.0		1.0	
Ever	140	85	83	1.1	0.8–1.6	1.7	1.1–2.5
				P = ns		P = 0.01	
Pitch tar and tar products							
Never	354	194	153	1.0		1.0	
Ever	52	32	27	1.2	0.7–2.1	0.9	0.5–1.7
				P = ns		P = ns	
Dry cleaning agents							
Never	402	218	175	1.0		1.0	
Ever	4	8	5	4.6	1.1–19.7	2.9	0.5–15.7
				P = 0.04		P = ns	
Fiberglass dust							
Never	380	202	172	1.0		1.0	
Ever	26	24	8	2.0	1.1–3.9	0.8	0.3–1.9
				P = 0.03		P = ns	
Luminous paint							
Never	404	219	180	1.0		1.0	
Ever	2	7		6.7	1.2–38.0		
				P = 0.03			
Asbestos dust							
Never	380	197	170	1.0		1.0	
Ever	26	29	10	1.9	1.0–3.5	0.9	0.4–2.1
				P = 0.05		P = ns	

[a] ORs adjusted for age, skin color, hair color, and mother's ethnic origin; ns, not significant.

SCC cases had diagnoses of either type of skin cancer prior to the index tumors diagnosed in 1983 or 1984. A further three SCC cases had BCC diagnosed simultaneously with their SCC. Thus, it is unlikely that response bias due to prior diagnosis of skin cancer materially affected case responses to questions on either sunlight or other exposures.

Relatively little systematic study has been directed toward nonsolar risk factors for basal and SCC of the skin. The significantly elevated risk of SCC and the suggestive results for BCC with prior histories of skin cancer in parents are in basic accord with previous data from Australia (10). As well, an increased risk of nonmelanocytic skin cancers in subjects exposed to X-rays has been reported previously (11, 12). The association of SCC with prior rheumatoid arthritis has not been reported previously to our knowledge. Immunization in childhood against polio, diphtheria, and tetanus appeared to be protective against SCC, suggesting that the immune status of individuals might be important in SCC risk.

Analysis of occupational history from job title information was relatively uninformative. The elevated risk for subjects in the social sciences and social work suggests a higher risk of BCC in indoor rather than outdoor workers, as has been seen in

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Table 4 Risk of SCC for chemical exposures after adjustment for solar UV exposure: 180 SCC, and 406 controls

Type and level of exposure	No. of controls	No. of SCC cases	Adjusted OR, SCC[a]	95% CI
Insecticides				
Never	332	123	1.0	
Low	38	21	0.7	0.3–1.4
High	36	36	2.8	1.4–5.6
			P(trend) = 0.02	
Herbicides				
Never	277	101	1.0	
Low	65	33	1.9	1.0–3.6
High	64	46	3.9	2.2–6.9
			P(trend) < 0.001	
Fungicides and seed treatments				
Never	237	84	1.0	
Low	85	40	0.8	0.4–1.4
High	84	56	2.4	1.4–4.0
			P(trend) = 0.003	
Grain dust				
Never	206	68	1.0	
Low	100	45	1.3	0.8–2.2
High	100	67	1.6	1.0–2.6
			P(trend) = 0.06	
Petroleum products				
Never	231	89	1.0	
Low	87	42	0.9	0.5–1.7
High	88	49	2.5	1.5–4.4
			P(trend) = 0.002	
Grease				
Never	239	86	1.0	
Low	85	51	1.9	1.1–3.3
High	82	43	2.6	1.5–4.4
			P(trend) < 0.001	
Diesel fumes				
Never	268	97	1.0	
Low	68	28	1.0	0.6–1.8
High	70	55	2.3	1.4–3.7
			P(trend) < 0.001	
Coal dust				
Never	298	111	1.0	
Low	54	35	1.4	0.9–2.5
High	54	34	1.7	1.0–3.0
			P(trend) = 0.03	

[a] ORs adjusted for age, skin color, hair color, mother's ethnic origin, and occupational sunlight exposure in the 10 years prior to diagnosis.

studies of malignant melanoma (13). It should be noted that this is based on very small numbers of cases. The finding of a decreased risk of SCC in clerical workers, managers, and administrators suggests a protective effect of occupations with low levels of chronic occupational sun exposure. This finding is in accord with our earlier analyses showing an elevated risk of SCC in subjects with high levels of occupational sun exposure in the decade prior to diagnosis (9). No elevated risks of SCC were seen in the occupational analyses for jobs traditionally considered outdoor in nature, such as farming, construction, laboring, and forestry.

The elevated risks seen for SCC in subjects reporting the use of insecticides, herbicides, fumigants, and seed treatment agents has not been seen previously. Classic texts have described the occurrence of SCC after exposure to arsenic in medicinal agents (14) and in contaminated well water (15). An association between arsenic and nonmelanocytic skin cancer was also reported in a recent Egyptian study (16). It is possible that some of the association seen in the present study is due to the use of arsenicals as pesticides earlier in this century. However, the excess risk seems to be present also for exposure in the 1960s and 1970s, when organochlorine pesticides had displaced arsenic. That the associations with pesticides persist after control for skin and hair color, mother's ethnic origin, and sunlight exposure argues that they are not due to confounding. Furthermore, the associations were seen only for SCC and not for BCC, and a dose-response relationship was seen for each of the agents. However, the fact that associations were seen with insecticides, herbicides, and fungicides is curious, because they have different modes of action. It is, of course, possible that host and pigmentary factors and sun exposure are not adequately controlled with the relatively simple methods available, and that residual confounding exists in these analyses. Confirmation of these findings in other studies would help allay these concerns.

An association was seen with grain dust exposure and SCC, although the addition of sun exposure to the multivariate model reduces the statistical significance level of the observation. Grain dust is known to stimulate airway, skin, eye, and nose irritation (17). Although the mechanisms for such disturbances are not clear, it is known that there are both allergic and nonallergic airway responses to dust. The immediate pulmonary response in those who are sensitive to grain dust and the sensitivity of grain handlers to histamine and methacholine (18,

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19) suggest an altered immune response to the dust. Together with the earlier findings on childhood immunization and rheumatoid arthritis, the association with grain dust suggest that exploration of the immune status of SCC patients and perhaps of individuals with solar keratoses, which are known precursors of SCC (20), might be fruitful.

Coal dust and grease exposure also appeared to have dose-response relationships with SCC. The lack of association with coal tar products is surprising in view of previous studies showing an elevated risk with these substances (21).

For the most part, associations between BCC and chemical and physical agents involved relative few exposed subjects. Only exposures to dry cleaning fluids, fiberglass dust, and luminous paint were statistically significant. No previous studies have reported these associations, and the small numbers of subjects exposed to luminous paint and dry cleaning fluids did not permit exploration of a dose-response gradient. No dose-response gradient was seen with fiberglass dust exposure.

A number of associations with nonsolar factors have been identified in this study. It should be noted that the study explored an excess of 50 different potential exposures, and in such a study, a number of these might be expected to be statistically significant by chance alone. However, in several cases, dose-response gradients were detected, lending credibility to the findings and suggesting causality. Because most of these findings were detected for SCC only and not also for BCC, it seems unlikely that they would be due solely to exposure misclassification or bias.

Further etiological studies of nonsolar risk factors particularly for SCC are needed to confirm the associations seen in this investigation.

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References

1. Marks, R., Staples, M., and Giles, G. G. Trends in non-melanocytic skin cancer treated in Australia: the second national survey. Int. J. Cancer, *53:* 585–590, 1993.

2. Gallagher, R. P., Ma, B., McLean, D. I., Yang, C. P., Ho, V., and Warshawski, L. M. Trends in basal cell carcinoma, squamous cell carcinoma, and melanoma of the skin from 1973 through 1987. J. Am. Acad. Dermatol., *23:* 413–421, 1990.

3. Glass, A. G., and Hoover, R. N. The emerging epidemic of melanoma and squamous cell skin cancer. JAMA, *262:* 2097–2100, 1989.

4. IARC. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans, Vol. 55, Solar and Ultraviolet Radiation. Lyon, France: IARC, 1992.

5. Elwood, J. M., Gallagher, R. P., Hill, G. B., Spinelli, J. J., Pearson, J. C. G., and Threlfall, W. Pigmentation and skin reaction to sun as risk factors for cutaneous melanoma; the Western Canada Melanoma Study. Br. Med. J., *288:* 99–102, 1984.

6. Gallagher, R. P., Elwood, J. M., and Hill, G. B. Risk factors for cutaneous malignant melanoma: the Western Canada Melanoma Study. Rec. Res. Cancer Res., *102:* 38–55, 1986.

7. Elwood, J. M., Gallagher, R. P., Hill, G. B., and Pearson J. C. Cutaneous melanoma in relation to intermittent and constant sun exposure: the Western Canada Melanoma Study. Int. J. Cancer, *35:* 427–433, 1985.

8. Gallagher, R. P., Hill, G. B., Bajdik, C. D., Fincham, S., Coldman, A. J., McLean, D. I., and Threlfall, W. J. Sunlight exposure, pigmentation factors and risk of non-melanocytic skin cancer: I—basal cell carcinoma. Arch. Dermatol., *131:* 157–163, 1995.

9. Gallagher, R. P., Hill, G. B., Bajdik, C. D., Coldman, A. J., Fincham, S., McLean, D. I., and Threlfall, W. J. Sunlight exposure, pigmentation factors and risk of non-melanocytic skin cancer: II—squamous cell carcinoma. Arch. Dermatol., *131:* 164–169, 1995.

10. Green, A., Beardmore, G., Hart, V., Leslie, D., Marks, R., and Staines, D. Skin cancer in a Queensland population. J. Am. Acad. Dermatol., *19:* 1045–1052, 1988.

11. Ron, E., Modan, B., Preston, D., Alfandary, E., Stovall, M., and Boice, J. D. Radiation induced skin carcinomas of the head and neck. Radiat. Res., *125:* 318–325, 1991.

12. Shore, R., Albert, R., Reed, M., Harley, N., and Pasternack, B. Skin cancer incidence among children irradiated for ringworm of the scalp. Radiat. Res., *100:* 192–204, 1984.

13. Lee, J. A. H. Melanoma and exposure to sunlight. Epidemiol. Rev., *4:* 110–135, 1982.

14. Neubauer, O. Arsenical cancer: a review. Br. J. Cancer, *1:* 192–251, 1947.

15. Tseng, W. P. Effects and dose-response relationships of skin cancer and blackfoot disease with arsenic. Environ. Health Perspect., *19:* 109–119, 1977.

16. El Khwsky, F., Bedwani, R., D'Avanzo, B., Assaad, S., Ali, A. E. S., Mokhtar, S., and La Vecchia, C. Risk factors for non-melanomatous skin cancer in Alexandra Egypt. Int. J. Cancer, *56:* 375–378, 1994.

17. Broder, I. Overview of adverse pulmonary effects of grain dust. *In:* J. Dosman and D. Cockcroft (eds.), Principles of Health and Safety in Agriculture, pp. 97–103. Boca Raton, FL: CRC Press, Inc., 1989.

18. DoPico, G. A., Jacobs, S., Flaherty, D., and Rankin, J. Pulmonary reaction to durum wheat, a constituent of grain dust. Chest, *81:* 55–61, 1982.

19. Tabona, M., Chan-Yeung, M., Enarson, D., MacLean, L., Dorken, E., and Schulzer, M. Host factors affecting longitudinal decline in lung spirometry among grain elevator workers. Chest, *85:* 782–786, 1984.

20. Marks, R., Rennie, G., and Selwood, T. Malignant transformation of solar keratoses to squamous cell carcinoma. Lancet, *1:* 795–797, 1988.

21. Hueper, W. C. Chemically induced skin cancer in man. Natl. Cancer Inst. Monogr., *10:* 377–391, 1963.

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