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Workmen's Compensation Problems 1954

BULLETIN 180

UNITED STATES DEPARTMENT OF LABOR

James P. Mitchell, Secretary

BUREAU OF LABOR STANDARDS

Paul E. Gorske, Director

Foreword

This Bulletin No. 180, contains the proceedings of the 40th annual convention of the International Association of Industrial Accident Boards and Commissions, which was held in Quebec, Canada, October 3–7, 1954.

Proceedings of the convention have been prepared for publication by the staff of the Bureau of Labor Standards.

Occupational Cancer Hazards

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A. Scope

Occupational cancer hazards have plagued mankind for thousands of years. Undue exposure to agents of our natural environment such as solar radiation or infections with the worm, Schistosoma haematobium, have doubtlessly been responsible for the occurrence of cancers of the exposed skin among outdoor workers and of cancer of the bladder among farmers of the Nile valley even during prehistoric times. However, it was not until the advent of modern industrialism some 150 years ago that occupational and industry-related agents have attained a preeminent position in the human cancer problem. Not only are occupational carcinogens the only well defined known causes of human cancers, but their number and variety have steadily increased since Pott in 1775 first related the occurrence of scrotal cancer among chimney sweeps to their exposure to soot.

Our real knowledge and appreciation of occupational cancer producing agents as phenomena of modern industrialism dates back some

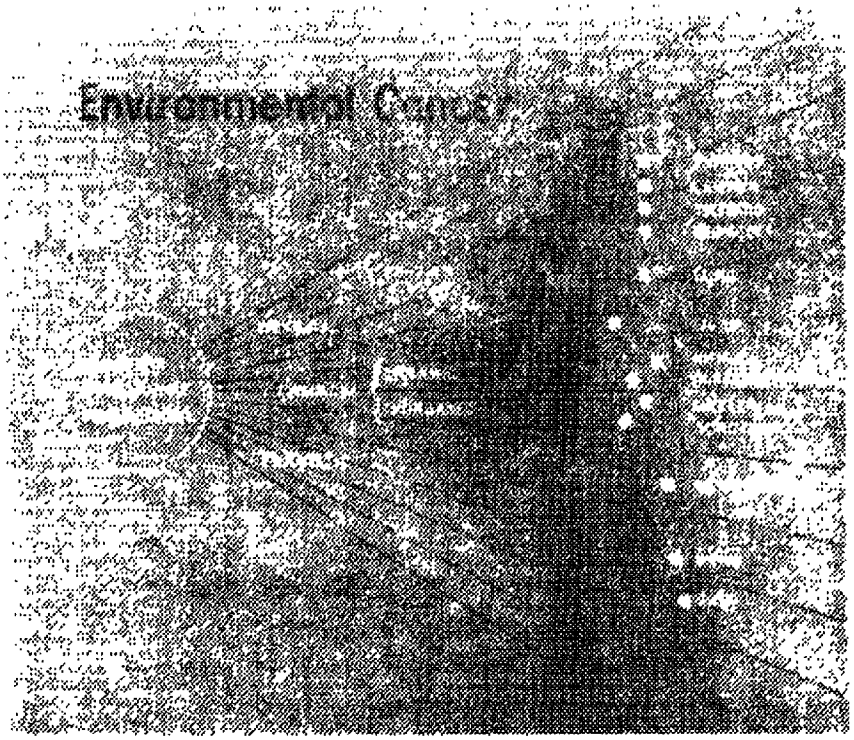


Figure 1.

75 years with the discovery of the relation between the occurrence of skin cancer and an occupational exposure to coal tar (fig. 1). The data given in the following tables (1 and 1a) briefly summarize the salient additions to the information on occupational cancer hazards made since that time. They list the various chemical and physical agents so far recognized or strongly suspected as causes of cancer in man. They show, moreover, the different organs and tissues representing the target organs of these agents and the number of occupational cancer cases placed on record or known to have been recognized as caused by them.

TABLE 1.—*Occupational Cancers Recorded in the United States and Foreign Countries up to 1952*

Agent	Sites of cancer	Number of cancer cases in U.S.A.	Number of cancer cases in Europe	Total
Arsenic	Skin	7	25	32
Coal tar, pitch	do	110	3,150	3,260
Petroleum	do	70	29	99
Shale oils	do	7	1,900	1,907
Lignite tar and paraffin	do		14	14
Creosote oil	do	1	40	41
Anthracene oil	do		20	20
Soot-carbon black	do	1	190	191
Aromatic amines	Bladder	350–400	1,200	1,600
Roentgen- and radium-radiation	Skin	(?)	(?)	(?)
	Leukemia	16	13	29
	Lung		650	650
Arsenic	Lung	7	16	23
Asbestos	do	32	53	85
Chromates	do	75	65	140
Nickel	do	0	84	84
	Nasal cavity	0	51	51
	Nasal sinus			
Coal tar, pitch	Lung	0	53	53
Petroleum	do	7	33	40
Benzol	Leukemia	11	31	42
Isopropyl	Lung	1	0	1
	Larynx	4	0	4
	Nasal sinus	6	0	6
Radioactive chemicals	Bones	9	0	9
	Nasal sinus	3	0	3
Total		[1] 750	7,650	8,400

[1] Approximate.

TABLE 1A.—*Time Table of Discoveries of Occupational Cancers*

Name of agent	Date	Discoverer	Organs affected	Nature of exposure
Chemicals—Organic:				
Soot	1775	Pott	Skin (scrotum)	Occupational.
Coal tar (lignite)	1876	Volkmann	Skin	Do.
Coal tar (bituminous)	1892	Butlin	do	Do.
Coal tar (anthracite)	1910	Zweig	do	Do.
Coal tar (fumes)	1933	Kawabata	Lung	Do.
Creosote oil	1920	O'Donovan	Skin	Do.
Anthracene oil	1903	Oliver	do	Do.
Paraffin oil (shale oil)	1876	Bell	do	Do.
Petroleum still coke	1890	Derville and Guermonprez.	do	Do.
Petroleum paraffin oil	1915	Schamberg	do	Do.
Petroleum lubricating oil	1838	Heller	do	Do.
Shale lubricating oil	1910	Wilson	do	Do.
Benzol	1925	Delore & Borgomano	Blood-forming tissues	Do.
Aromatic amines (dye intermediates).	1895	Rehn	Bladder, ureter, kidney.	Do.
Isopropyl oil	1952	Weil, Smyth & Nale.	Nasal sinuses, larynx, lung.	Do.
Chemicals—Inorganic:				
Arsenic	1822	Paris	Skin	Do.
	1887	Hutchinson	do	Medicinal.
	1930	Snape	Lung	Occupational.
Asbestos	1933	Lynch & Smith	do	Do.
Chromates	1935	Pfeil	do	Do.
Nickel	1932	Grenkil	Nasal cavity and sinuses, lung.	Do.
Physical agents:				
Ultraviolet radiation	1894	Unna	Skin	Do.
				Environmental.
Roentgen radiation	1902	Frieben	do	Occupational.
	1903	Wyss	do	Medicinal.
	1911	Von Jagic, Schwartz & von Stirkenrock.	Blood-forming tissues.	Occupational.
	1922	Bock	Bone	Medicinal.
Radioactive substances	1879	Hesting & Hesse	Lung	Occupational.
	1922	McNeal, Ward and Wilts.	Skin	Do.
	1928	Lowy	Blood-forming tissues	Do.
	1931	Martland	Bone	Do.

However, an indiscriminate acceptance of these figures as reflecting the actual scope of the occupational cancer hazards would seriously be misleading for the following reasons. The presence of occupational cancer hazards has so far been established for only a few worker groups having contact with one or several of the carcinogenic agents listed. While, for instance, it is generally known that workers employed in gas works and coal tar distilleries or engaged in the cleaning of petroleum stills and having skin and respiratory contact with coal and petroleum pitch and tar show an excessive liability to cancer of the skin, and apparently also, of the lung, there does not exist any official record of similar cancerous complications among electrode workers in aluminum production plants where an exposure to fumes

from the electrodes consisting of a mixture of coal tar, pitch, and petroleum coke exists. Reliable information, however, is on hand that these workers suffer from cancers of the skin and, possibly, also the respiratory tract. The extent of this exposure hazard may be estimated by the fact that approximately two-thirds of a pound of electrode carbon is consumed for each pound of metallic aluminum produced, of which 800,000 short tons were consumed in the United States in 1945.

It is equally significant that agents which have been reported to have caused occupational cancers among workers of an industry of one country, do not seem to have the same effect upon the workers of an identical industry of another country, if the available data would be accepted at their face value. Since carcinogens behave in their fundamental disease-producing properties like pathogenic microorganisms, there is no sound reason to assume that carcinogens make any distinctions in their activity or effectiveness for any national reasons. In fact, it can be shown that such scientifically incomprehensible discrepancies are mainly due to local differences of reporting of occupational cancer cases.

The existence of an excessive liability of gas house retort and coke oven workers to cancer of the lung has been reported from Japan, Canada, and England, but not from any other country despite the fact that the exposure conditions for workers employed in such operations are identical. The occurrence of an appreciable number of arsenic cancers of the skin among German users of arsenical insecticides was placed on record during recent years. If full credence would be given to reports on arsenic cancers observed in the United States, one would be led to believe that arsenicals in our part of the world are carcinogenic for almost exclusively medicinal reasons. Again, it is hard to assume that arsenicals would make such distinctions. A similar situation seems to exist concerning the carcinogenicity of lubricating oils obtained from petroleum in different parts of the world. Although there were placed on record by English investigators during recent years, several hundred cases of skin cancers due to occupational contact with lubricating oils, practically nothing is officially known as to the existence of such industrial diseases from the countries on this side of the Atlantic. One seems to be content with attributing here the English observations to the carcinogenic action of shale oils, the large-scale industrial use of which was discontinued during the 1920's, and therefore cannot account for the more recent English cancer cases observed among spinners, mechanics, engineers and other workers having had exclusive contact with lubricating and cooling oils of petroleum derivation.

It is worth noting in this connection that during the last 20 years, there has been an almost complete lack of published additions to the list of occupational coal tar, pitch and creosote oil cancers of the skin from the United States, although such cancers have been observed and are recorded as such in industrial records, and despite the fact that the annual reports of the Chief Inspector of Factories of England and Wales have regularly listed notifications of such lesions to the extent of from 100 to 250 cases per year.

One may also reflect on the fact that the last published figures on occupational cancers in American dye workers appeared in 1938 and listed less than 100 cases, all occurring among employees of one company. During the intervening 15 years, the total number of such cases has risen several fold and no official data have become available from any of the other companies producing and handling carcinogenic aromatic amines used in the production of dyes and rubber antioxidants. In contrast, recent reports by English investigators supported by the English chemical industries have supplied adequate figures on the number, incidence rates, and attack risks of bladder cancer among English dye and rubber workers who had been exposed to the same carcinogenic aromatic amines with which the corresponding worker groups in this country have occupational contact.

Finally, some consideration must be given to the fact that the general population has varying degrees of contact with some of the carcinogenic agents through their presence in industrial wastes released into the environmental air and bodies of water, and entering subsequently the soil and the fauna and flora living in such a polluted environment and used in part for nutritional purposes. In the past, claims as to the occurrence of so-called "neighborhood" cases of cancer have rarely been made. The well established former occurrence of arsenic cancers among inhabitants of a German region in which the drinking water supply was polluted with arsenic from mine slag heaps represents the first occurrence of this type.

Following the discovery of bladder cancer hazards among German dye workers, it was asserted that an environmental plant contamination with carcinogenic aromatic amines was responsible for the production of bladder cancers among persons living in the vicinity of this plant. While the actual existence of such conditions was denied at that time by other investigators, the recent demonstration of "neighborhood cases" of berylliosis makes such claims appear as not unreasonable possibilities since a highly potent carcinogen, such as beta-naphthylamine, is involved which might have produced such results if the waste disposal and housekeeping methods of the plant were inadequate. It is probable that the future occurrence of cancers of the lung and bone among individuals who lived near beryllium-producing or processing plants might furnish additional evidence of

cancer hazards related to environmental plant pollution if beryllium and its compounds are not only carcinogenic for rats and rabbits but also for man. Similar conditions of industrial atmospheric pollution exist around arsenic smelters, chromate producing plants, carbon black factories and oil refineries, as well as uranium ore mills.

The recent compensation claims advanced on the basis of an alleged existence between cigarette smoking and lung cancer also point to the possibility of a greater future importance of the compensation aspects connected with the occurrence of cancers from industry-related sources, such as exposure to carcinogens contained in the exhaust fumes from diesel and gasoline engines, or present as additives and contaminants in foodstuffs.

It appears from these considerations that there can be no doubt that the actual number of occupational cancers is considerably higher than the number of cases placed on record. It is of some interest, moreover, that the number of cases recorded in medical journals is appreciably higher than the number of occupational cancers which during the last 20 years have come for decision before compensation boards because compensation claims in many such cases have been settled out of court. With a few exceptions, such as the incidence of lung cancer among American chromate manufacturers and among German and Czechoslovakian radioactive ore miners, of nasal sinus, larynx and lung tumors among American isopropyl alcohol manufacturers, and of skin cancer among English coal tar manufacturers and processers, no reliable information is available on the incidence rate of occupational cancers of the various types among different occupational population groups.

B. Special Aspects of Occupational Cancers

A fair and competent adjudication of compensation claims for cancers allegedly caused by occupational agents or conditions requires an adequate knowledge of the special aspects related to the production and development of these tumors and of the criteria by which they may be distinguished from non-occupational environmental (medicinal, dietary, cosmetic, habitual) as well as "spontaneous" cancers.

1. Definition of the Term "Occupational Cancer".—The term "occupational cancer" is applied to malignant tumors originating in individuals during the course, and/or as the result of the regular and usually prolonged exercise of certain occupational activities entailing contact with some exogenous, physical, chemical or parasitic agent acting in proper intensity. Occupational cancers thus are caused by specific agents or etiologically related to well defined occupational conditions which form an integral part of the ordinary working environment. They differ through these aspects from the accidental occupational cancers which result from or follow upon some extraor-

dinary, unforeseen, accidental injury sustained at work and being of a nonspecific nature.

8. Types and Nature of Occupational Carcinogens.—Occupational agents which are known or strongly suspected as having caused cancers of various organs in workers who became exposed to them are largely chemicals. Some of these chemicals are well defined, others consist of variable mixtures of a large number of chemical compounds, a few of which possess carcinogenic properties. A second group of occupational carcinogens is composed of agents characterized by their ionizing or nonionizing radiation. A third group is represented by environmental agents and conditions which sometimes have an occupational association, and which may be related to certain infections or several specific climatic, geologic, dietary, and nutritional conditions, and which in part have more or less indirect relations to carcinogenesis (table 2).

TABLE 2.—*Occupational Carcinogens—Their Route of Exposure and Their Target Organs*

General type	Specific type	Route of exposure	Target organ
Chemical carcinogens:			
Organic chemicals—aromatic, polycyclic, heterocyclic	Benzol	Cutaneous, respiratory.	Blood-forming organs (leukemia, lymphosarcoma, myeloma).
	Beta-naphthylamine, benzidine, 4-aminodiphenylcarbamine, magenta?) (toluidine?).	Respiratory, cutaneous, alimentary.	Urogenous organs (bladder, ureter, kidney).
	Coal tar, pitch, soot, asphalt, tar oil, creosote oil, anthracene oil lamp black, lignite tar and paraffin oil.	Cutaneous Respiratory	Skin. Lung.
	Synthetic coal oil and tar (Bergius).	Cutaneous	Skin.
	Shale oil and paraffin oil	do	Do.
		Respiratory	Larynx.
	Petroleum fuel oil, diesel oil, lubricating oil and grease, cutting oil, carbon black, asphalt, tar, coke, crude paraffin oil.	Cutaneous Respiratory	Skin. Lung.
Aliphatic	Isopropyl oil	do	Nasal sinus, larynx, lung.
Inorganic chemicals.	Arsenic	Cutaneous, ingestive	Skin.
		Respiratory	Lung.
	Nickel	do	Nasal cavity, nasal sinus, lung.
	Chromium	do	Lung.
	Asbestos	do	Do.
Physical carcinogens: Ionizing radiation.	X-radiation	Cutaneous	Skin.
		Transcutaneous	Connective tissue, bone, blood-forming organs.
	Radioactive radiation (alpha, beta, and gamma-radiation).	Cutaneous	Skin.
		Transcutaneous	Connective tissue, blood-forming organs.
		Respiratory	Nasal sinus Lung.
		Ingestive	Bone.
Parasitic carcinogens	Schistosoma haematobium	Cutaneous	Bladder

In addition to these established occupational carcinogens there are a number of potential human carcinogens which have been shown to elicit cancers in experimental animals and to which workers become exposed during their regular occupational activities. These potential agents which also must be considered in the adjudication of compensation claims, are listed in tables 3 and 4.

TABLE 3.—*Potential Environmental Human Carcinogens*

Agent	Site of tumor	Type of exposure	Species	Types of human contacts
Estrogens	Breast	Parenteral	Mouse	Manufacture.
Natural	Lymph nodes	Cutaneous	Rat	Dietary additive.
Synthetic	Uterus	Oral	Rabbit	Cosmetic ingredient.
	Hypophysis		Guinea pig	
Carbon Tetrachloride	Liver	Oral	Mouse	Industrial solvent.
				Degreasing agent.
				Dry-cleaning agent.
				Fire-extinguishing agent.
				Manufacture
				Production of freon.
				Grain fumigant.
				Extractive of oils.
Chloroform	do	Oral	Mouse	Manufacture.
		Parenteral		Anesthetic.
				Solvent and Extractive of oils, resins, rubber, waxes, iodine, alkaloids.
				Ingredient of lacquer, floor polish, cleaning fluid.
				Production of artificial silk, plastics.
DDT (4,4-Bis(p-chlorophenyl-1,1,1-trichloroethane).	do	Oral	Rat	Manufacture.
				Insecticide.
				Food contaminant.
Tannic acid	do	Parenteral	do	Ingredient of foodstuffs (fruits, wine, coffee, and tea).
				Medicinal agent.
				Tanning agent.
Thiourea and derivatives.	do	Oral	do	Citrus fruit preservative.
	Thyroid			Medicinal agent.
p-Phenetylurea (Dulcin).	Liver	do	do	Manufacture.
				Sweetening agent.
Diethylene glycol	Bladder	do	do	Antifreeze.
				Intermediate in explosive manufacture.
				Softener of lacquer inks, wood stains, glue, textile, humectant (tobacco).
Methylated naphthalenes.	Skin	Cutaneous	Mouse	Vehicle of insecticides.
Bergius coal oils and tar.	do	Cutaneous	Mouse	Manufacture.
	Subcutaneous tissue.	Parenteral	Rat	Fuel.
				Lubricants.
				Plastic production.
				Petrochemical.
Fischer-Tropsch coal oils, tars, waxes, greases.	Liver	Cutaneous		Manufacture.
		Parenteral	Mouse	Fuel.
				Lubricant.
				Petrochemical

TABLE 3.—*Potential Environmental Human Carcinogens*—Continued

Agent	Site of tumor	Type of exposure	Species	Types of human contacts
Light green SF brilliant blue FCF fast green FCF,	Subcutaneous tissue.	Parenteral	Rat	Textile dyes Food and cosmetic dyes.
Butter yellow	Liver.	Oral	Mouse Rat	Food dye (orient). Gasoline dye. Flare color.
Cellophane, polyethylene, polyamide (nylon), bakelite teflon, polymethyl methacrylate.	Subcutaneous.	Parenteral	do	Film, fiber, plastic-industrial manufacture and uses. Wrapping material. Medicinal agent.
Beryllium	Bone	Parenteral Respiratory	Rabbit Rat	Metal alloy, X-ray tube, phosphor manufacture. Refractory vessels Atomic energy production.
Selenium	Liver, thyroid	Oral	Rat	Soil contaminant. Coloring matter of glass, ceramics, paint, rubber. Metal alloys. Rubber accelerator. Photoelectric apparatus Decolorizer, Fireproofing agent. Medicinal agent.
Cobalt	Subcutaneous tissue.	Parenteral	Rat	Metal alloys and plating. Coloring matter of glass, ceramics, paint, rubber Medicinal agent.

TABLE 4.—*Carcinogenic Chemicals Eliminated as Environmental Human Carcinogens*

Agent	Site of cancer	Type of exposure	Species	Intended use
Styryl 236	Subcutaneous tissue.	Parenteral	Mouse	Trypanocidal agent.
2-Acetylamino-fluorene	Subcutaneous tissue. Bladder Liver Brain. Thyroid. Acoustic duct. Breast. Leukemia. Kidney. Lung.	Oral Parenteral	do Rat Dog Fowl	Insecticide.

3. Evaluation of Evidence.—For the establishment of causal relations between an exposure to these agents and the subsequent development of a cancer of an occupational genesis and therefore compensable nature, the following prerequisites must be fulfilled:

(*a*) Definite evidence must be available indicating that the claimant had occupational contact with any one of the various agents listed, that this contact was either of prolonged nature extending as a rule over periods of at least 6 months, and was of adequate intensity for eliciting a cancerous response, according to any previous experience which might be available on this point. Single exposures may be considered to be effective if the casual agent consists of external ionizing radiation or the exposure mechanism favors the production of a carcinogenic depot in the tissues from which a prolonged exposure of the host organism may ensue.

(*b*) The route of exposure to or introduction of the carcinogenic agent in addition to its specific chemical properties and metabolic peculiarities determine the site of the organ or tissues in which a cancer is elicited. Therefore, not any cancer, but only cancers of specific sites are related to any particular carcinogenic agent.

(*c*) The type of exposure does not need to be continuous or extend up to the time of appearance of the cancer, but may be intermittent. In fact, any contact with the occupational carcinogenic agent may have ceased many months to years or, occasionally, even decades before the cancer becomes manifest. Effective carcinogenic exposures may be sustained not only by the ordinary operators employed in operations with occupational carcinogenic hazards, but also by workers entering such operations on a transitory basis, such as supervisors, maintenance personnel, repairmen, truckers, loaders, yardmen, and workers employed in rooms and buildings near the hazardous operations, if housekeeping and exhaust disposal methods are inadequate and environmental contaminations result.

(*d*) *Occupational Medical Stigmata.*—For the establishment of causal relations between occupational exposures to carcinogens and subsequently appearing cancers, it is of distinct value to analyze the medical history of the affected persons for functional or anatomic disease manifestations characteristic of a pathogenic action of the particular carcinogen allegedly involved. Such noncancerous and often preparatory or precancerous symptoms of etiologic specificity act as stigmata or biologic finger prints supporting not only the actuality of a previously sustained specific exposure, but also its causal relationship to the cancer present. Symptomatic manifestations possessing such a significance are, for instance, chronic radiodermatitis, chronic tar dermatitis, arsenic dermatosis, chronic solar dermatitis, chronic radiation periostitis, asbestosis of the lung, and leukemoid reactions after exposure to benzol and ionizing radiation. Chemical analyses of tissues for the incriminated chemical also may sometimes be of assistance in establishing previous contact with any specific occupational carcinogens, such as arsenic, chromium, nickel, radioactive substances, and benzol.

It is important, however, to note that both these procedures are not applicable for all occupational cancers. Some of the carcinogens do not produce any etiologically characteristic symptoms, in fact, they may not elicit any preliminary toxic or other disease phenomena despite a persistent and prolonged carcinogenic exposure. Thus, cancers of the bladder in dye and rubber workers may develop in the absence of any preliminary local or general warning symptoms after more than one or two decades of continuous, intermittent, or discontinued exposure to certain carcinogenic aromatic amines.

There also does not exist any parallelism between the toxic and/or irritative properties of any chemical and its potential carcinogenic qualities, nor, is there a relationship between the degree of toxicity of any particular carcinogen and its relative carcinogenic potency.

(e) Extensive experience with occupational cancers indicates that there usually elapses a latent period of years, if not decades, between the onset of exposures to a carcinogenic agent and the appearance of the resulting cancer (table 5). It is for this reason most unlikely that occupational cancers become manifest within the first year following the inception of contact with a carcinogenic agent. In fact, all cancers which make their appearance during the first three years of occupation in a carcinogenic environment should be subjected to severe scrutiny for a more probable nonoccupational origin before they may be considered from any occupational viewpoint.

TABLE 5.—*Latent Periods of Occupational Cancers*

Organ and agent	Average latent period	Range of latent period
Skin		
Arsenic:	*Years*	*Years*
Medicinal	18	3–40
Occupational	25	4–46
Tar	20–24	1–50
Creosote oil	25	15–40
Mineral oil	50–51	4–75
Crude paraffin oil	15–18	3–35
Solar radiation	20–30	15–40
X-radiation	?	1–12
Lung		
Asbestos	18	15–21
Chromates	15	5–17
Nickel carbonyl	22	6–30
Tar fumes	16	9–23
Ionizing radiation	25–35	7–50
Bladder		
Aromatic amines	11–15	3–40

(*f*) A successful production of cancers in experimental animals by the administration of any particular chemical or occupational agent supplies weighty support to a claim that such a factor may have an identical effect upon man, especially if the route of exposure, the physiochemical status of the agent during contact, and the cancer produced in experimental animals are identical with the corresponding aspects in human experience. However, because of the pronounced species specificity of axogenous carcinogenic agents, even positive experimental evidence provides merely strongly suggestive but not entirely conclusive evidence as to the existence of carcinogenic properties of such agents for man. Only human experience gives a definite and irrefutable answer in such matters.

(*g*) Pathologic and histologic evidence is in general of only limited importance in the establishment of the occupational causation of cancers, since neither the clinical behavior nor the gross appearance and histologic structure of cancers are etiology specific. However, in some occupational cancers histologically or histochemically demonstrable manifestations are present which give conclusive or suggestive evidence as to their particular etiology. The presence of asbestos bodies and asbestosis in a lung affected by cancer provides practical and scientific proof as to the asbestotic origin of such a pulmonary carcinoma. Of similar causal significance would be the demonstration of chromite dust by histochemical methods and by incineration of sections of the lung of an individual with pulmonary carcinoma. The histochemical demonstration of arsenic in the skin affected by a carcinoma represents another illustration of the value of histologic studies in the determination of the etiology of a cancer. Likewise the presence of extensive endarteritic changes in a fibrosed subcutaneous tissue containing a carcinoma or sarcoma would arouse the suspicion that X-rays or radioactive energy was causally responsible for the malignant lesion.

It appears from these considerations that a competent and critical evaluation of the entire evidence related to the exposure of the individual the symptomatic manifestations of the preparatory period, the length of the latent period, the site of the cancer, some histologic or histochemical characteristics and the existence of corresponding observations on cancer production in experimental animals aside from pertinent epidemiologic data represents the scientific basis essential for medicolegal decisions in litigations of alleged occupational cancers.

C. Compensation Laws

Workmen's compensation laws enacted in various countries and states reveal a remarkable lack of uniformity as to the types of occupational carcinogenic agents, industrial operations with carcinogenic

hazards, and occupational cancers covered. As of October 1, 1954, full coverage laws applying to all kinds of occupational diseases including cancers had been passed in 26 States of the United States and the District of Columbia; schedule coverage laws not always covering occupational cancers, or all types, were in force in 20 States and no occupational disease laws had been enacted at that time in 2 States (Mississippi and Wyoming).

While workmen's compensation laws based on the principle of schedule coverage may provide some degree of compensation against disability and injuries resulting from occupational cancer hazards, such legal provisions designed for the protection of workers are totally inadequate, if not obsolete, in an era of a rapidly changing and increasing occupational disease panorama. Such static laws reflect a legal and sociologic state of mind which is maladjusted to the dynamic state of modern industry.

However, even the blanket or general coverage compensation law which is sufficiently flexible to include not only the present but also any future occupational cancer hazards, usually has a statute of limitations which is inadequate for providing fair protection under certain and not too unusual circumstances met with in occupational cancer. Since occupational cancers may become manifest many years after cessation of exposure to occupational carcinogenic agents, a statutory time clause of from 6 months to 6 years may be too short for occupational cancers which may appear after a longer period. It is advisable for this reason to adjust all existing laws in such a way that such eventualities are properly covered.

In the workmen's compensation laws existing in most States, certain groups of workers or employees, such as especially farm labor and domestic servants, are excluded from coverage by the law. Since farming as well as housework have become and are increasingly becoming occupations having extensive contact with industrial products (pesticides, herbicides, synthetic fertilizers, antisprouting agents, hormonal fattening agents of livestock, lubricants and fuel of farm machinery, residual oils for smudge pots, soil improvers, antibiotics, detergents, waxes, polishes, dry cleaning agents), there is no sound scientific reason for excluding members of such occupations from coverage by the workmen's compensation laws and from adequate protection against cancers arising from exposure to potential and actual occupational carcinogenic agents.

D. Labor Codes and Standards

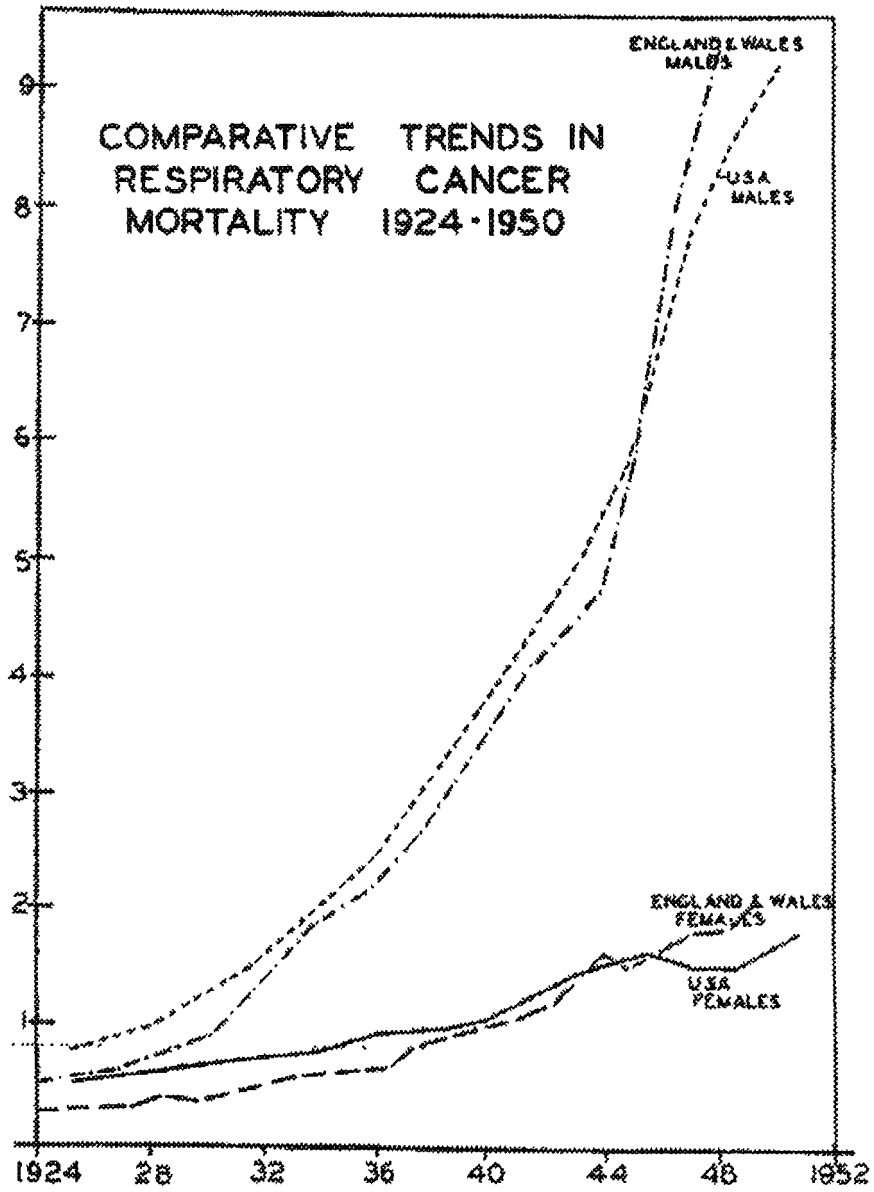
Any attempt toward achieving an adequate compensation by financial means for a cancerous condition acquired through contact with occupational agents not only presents considerable economic difficulties but is also of questionable humane merit. While such a procedure is evidently the best that can be done after an occupational cancer has become an accomplished fact, the wiser, more rational, and in the long run, more economical approach to a reasonable control of occupational cancer hazards lies obviously in their prevention wherever this is possible.

This goal can be achieved to a large degree by the enforcement of proper work conditions in operations in which cancer hazards exist, so that either any contact of workers with such agents is eliminated or reduced to an ineffective level. As to the fundamental step for accomplishing such a control of occupational cancer hazards, it would be advisable to have all factories, workshops, laboratories and other establishments producing, handling or using known or suspected carcinogenic materials to register with State public health and labor agencies. This information would provide the basis for establishing the supervision of plants by health and labor agencies and would create the proper machinery for familiarizing managers of such operations with the potential carcinogenic properties of the substances and devices used in their establishments and with the methods and procedures by which they might be controlled or avoided.

The relative effectiveness of any medical, sanitary and technologic control measures to be adopted can best be determined by ascertaining the changes in the occupational cancer incidence among the total population at risk occurring during some 10 to 30 years after their introduction. It is therefore desirable that provisions be made for routinely recording all cancer cases occurring among worker groups exposed to occupational carcinogens. All precancerous and cancerous lesions observed among members of such groups should for this reason be made notifiable with the State health agency. All persons having knowledge of those cancers should be held responsible for such reporting. The adoption of such measures represents an important step from a public health viewpoint, because there is a growing urgency to determine the environmental causes responsible for significant changes in the incidence rates of several cancers.

A great deal of circumstantial and direct evidence incriminates recent changes in the industry-related environmental factors for the startling and progressive increase in the lung cancer frequency observed

in all industrialized countries during the past 50 years (graph 1). A similar, while not as marked, rise in mortality rates has been observed during the last two decades for cancers of the blood forming tissues, especially leukemias, for which the crude mortality rate increased from 1.0 per 100,000 of the population in 1900 to 4.3 per 100,000 in 1944. This development coincides with a marked increased use of ionizing radiation as well as with a widespread industrial and medicinal employment of chemicals having distinct hematotoxic effects.



GRAPH 1.

It also may be of significance that the age-specific, total and age-adjusted mortality rates per 100,000 population for skin cancer in white males and white females in the United States have shown between 1930 and 1950 a marked shift in favor of the younger age groups (table 6). Whether such changes in the age distribution of skin cancers for both sexes may have resulted from the modern habit of sun bathing or a more intense environmental, occupational and dietary contact of the general population with arsenicals, or with products of the coal tar and petroleum industry, remains at present a matter of speculation. For determining the possible causal significance of such factors in this development, it would be necessary to ascertain data on occupational, dietary, environmental and habitual contacts of the affected individuals, their complexion and race, and

TABLE 6.—*Age-specific, Total, and Age-adjusted Mortality Rates Per 100,000 Population for Skin Cancer in White Males and White Females, United States, 1930, 1945, and 1950*

Age	White males			Percent change 1930–50
	1930	1945	1950	
Under 15	0.06	0.08	0.03	−62
15–24	.12	.11	.34	+209
25–34	.22	.46	.81	+76
35–44	.75	.85	1.4	+65
45–54	2.5	2.1	2.7	+29
55–64	8.0	6.3	5.9	−11
65–74	23.7	16.0	12.8	−20
75–84	68.2	52.3	32.6	−38
85 and over	194.2	172.4	77.5	−55
All ages	3.3	3.2	2.9	
Age adjusted [1]	4.7	3.6	2.9	
	White females			
Under 15	0.07	0.07	0.02	−71
15–24	.06	.13	.31	+138
25–34	.23	.23	.73	+213
35–44	.63	.55	1.2	+118
45–54	1.5	1.0	1.8	+80
55–64	3.6	2.9	3.6	+24
65–74	11.4	8.7	6.8	−32
75–84	44.6	26.3	18.8	−29
85+	162.7	132.7	68.5	−48
All ages	2.1	2.0	2.0	
Age adjusted [1]	2.9	2.1	1.9	

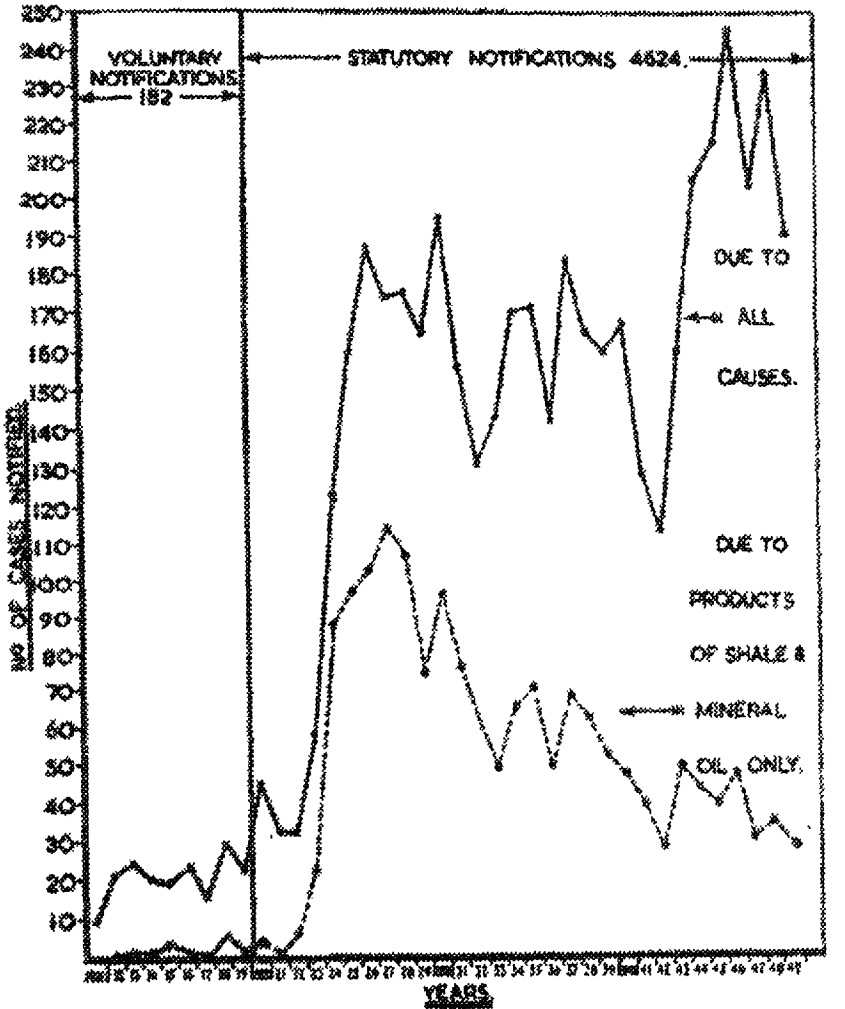
[1] Adjusted for age on total population of the United States, 1950.

on the location of the cancers in the exposed or unexposed parts of the skin (site distribution).

All such data could readily be obtained for occupational factors if all occupational cancers would be made notifiable. At the same time this measure would be instrumental in ascertaining to a much greater degree than heretofore the scope of occupational cancer hazards and of the cancers resulting therefrom. The effectiveness of this procedure is strikingly illustrated by a graph (graph 2) published several

CUTANEOUS CANCER IN RELATION TO OCCUPATION

GRAPH SHOWING THE NUMBER OF CASES OF CUTANEOUS CARCINOMA IN MALES & FEMALES IN ENGLAND, WALES & SCOTLAND NOTIFIED ANNUALLY FROM 1911 – 1949 INCLUSIVE.



THE DOTTED PORTION OF THE GRAPH DENOTES THE NUMBER OF CASES CONTRIBUTED BY SHALE OIL & MINERAL OIL ONLY.

Graph 2.

years ago by Henry in which he showed the striking rise in the number of cases of cutaneous carcinoma in males and females in England, Wales and Scotland attributable to occupational exposures to coal tar, pitch, shale oil and mineral oil after statutory notification went into effect in 1920.

Conclusions

1. The rapid development of modern industrialism has been associated with the appearance of many new occupational health hazards of which those causing cancers occupy an important position.

2. The increasing rate at which occupational carcinogens have been discovered during recent decades suggests that only a fraction of the actually existing industrial carcinogens is known and that heretofore unknown and new occupational carcinogens may be added to the present industrial environment through the development and use of new chemicals and production methods.

3. There is a great deal of evidence indicating that only a relatively small fraction of the cancers produced through contact with occupational carcinogens has been recognized and/or placed on record. Because of the established widespread industrial contact of large worker groups with known occupational carcinogens, the potential scope of the occupational cancer problem evidently is much larger than apparent from the reported number of occupational cancer cases and hazardous industrial operations.

4. Medicolegal decisions in compensation procedures of occupational cancer claims should be based on a competent and critical evaluation of the entire evidence related to the exposure conditions of the claimant, the presence or absence and the type of any etiology, specific symptomatic manifestations observed during the latent and developmental periods of the cancer, the length of the latent period, the site of the cancer, any specific histologic and histochemical characteristics, the production of corresponding cancers in experimental animals by the incriminated agent and pertinent epidemiologic data.

5. Schedule coverage laws are inadequate for providing proper protection against occupational cancer hazards because of the dynamic state of modern industry resulting in the constant addition of new chemicals and production methods which may entail heretofore unknown occupational cancer hazards. The statute of limitations contained in the general coverage laws represents its main defect in the fair and equitable application of this type of workmen's compensation law to occupational cancer hazards.

6. Since the prevention of occupational cancer hazards and not financial compensation for established occupational cancers appears to be the more sensible and humane method of occupational cancer

control, labor codes should be properly adjusted and enforced so as to eliminate or reasonably contain occupational cancer hazards in modern industries.

7. For ascertaining the actual scope of the occupational cancer problem and for the institution of adequate control measures, it is essential that occupational cancers and precancerous conditions are made notifiable with local and State health and labor authorities.

8. A comprehensive study of occupational cancer hazards will provide a sound basis for an equally urgent investigation of the apparent extension of industry-related cancer hazards to large parts of the general population.

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The Accident Prevention Associations Under the Quebec Workmen's Compensation Law

L. H. T. Clegg, *President, Quebec Association for the Prevention of Industrial Accidents*

[In the absence of Mr. Clegg, his statement was read by J. Armand Desrochers, vice president, Quebec Association for the Prevention of Accidents]

I will outline briefly the setup of the various safety associations, and later, being more familiar with the organization which I represent, I shall endeavour to elaborate on a few of its major promotional activities.

APPENDIX C

Official Attendance at the Fortieth Annual Meeting of the International Association of Industrial Accident Boards and Commissions

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Adam Bell, Chairman, Workmen's Compensation Board, Vancouver.
C. W. Pritchard, Commissioner, Workmen's Compensation Board, Vancouver.

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J. A. LeBlanc, Claims Officer, Workmen's Compensation Board, St. John.

Newfoundland

Clarence V. Hancock, Commissioner, Workmen's Compensation Board, St. John's.

Nova Scotia

Arthur Perrin, Commissioner, Workmen's Compensation Board, Halifax.
Frank Rowe, Chairman, Workmen's Compensation Board, Halifax.

Ontario

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F. Fincham, Workmen's Compensation Board, Toronto.
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E. E. Sparrow, Chairman, Workmen's Compensation Board, Toronto.
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Dr. E. C. Steele, Workmen's Compensation Board, Toronto.
Dr. Bruce Young, Director, Rehabilitation, Workmen's Compensation Board, Toronto.

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Dr. Bertrand Bellemare, Workmen's Compensation Commission, Quebec.
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Jacques Bernard, Montreal.
Dr. L. A. Brochu, Workmen's Compensation Commission, Quebec.
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Arthur Foster, Vice President, Workmen's Compensation Commission, Quebec.
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William A. Callahan, Chairman, Industrial Accident Commission, Salem.

Pennsylvania

F. R. Barnard, Manager, Compensation and Safety, Bethlehem Steel Co., Bethlehem.
Frank K. Beal, Deputy Secretary, Department of Labor and Industry, Harrisburg.
A. C. Christman, Chief Supervisor, Indemnity Insurance Co. of North America, Philadelphia.
W. D. Coakley, Secretary, Fire Association of Philadelphia, Philadelphia.
E. Gwilliam, Manager, State Workmen's Insurance Fund, Harrisburg.
R. M. Hartman, Assistant Manager, Compensation and Safety, Bethlehem Steel Co., Bethlehem.

ROBERT C. HERON, Superintendent, Compensation Claim Department, American Casualty Co., Reading.
RUSSEL C. KEISER, Assistant Manager, State Workmen's Insurance Fund, Harrisburg.
WALTER LINN, Secretary-Treasurer, National Council of State Self-Insurers Association, Philadelphia.
JOHN L. NAYLOR, Director, Bureau of Workmen's Compensation, Harrisburg.
F. PRITCHARD, Chief Safety Inspector, State Workmen's Insurance Fund, Harrisburg.
A. SCHROEDER, General Attorney, Pennsylvania Railroad Co., Philadelphia.
DAVID M. WALKER, Secretary, Department of Labor & Industry, Harrisburg.

Puerto Rico

MISS RAQUEL NEGRONI, Member, Industrial Commission, San Juan.
EDUARDO A. SANTIAGO, Actuary, State Insurance Fund, San Juan.

Rhode Island

ANTONIO DI PINTO, Executive Assistant Curative Center, Department of Labor, Providence.
DR. JOHN E. DONLEY, Medical Director, Curative Center, Department of Labor, Providence.

South Carolina

MISS FAITH CLAYTON, Commissioner, Industrial Commission, Columbia.
JAMES J. REID, Vice Chairman, Industrial Commission, Columbia.
MRS. H. S. TATE, Columbia.

Tennessee

W. H. PARHAM, Commissioner, Department of Labor, Nashville.
PAUL A. PHILLIPS, Safety Engineer, Department of Labor, Nashville.
DEWEY WHITTLE, Director, Division of Workmen's Compensation, Department of Labor, Nashville.

Texas

DR. V. C. BAIRD, Chief Physician, Humble Oil & Refining Co., Houston.
R. L. ECKMAN, Vice President, Petroleum Casualty Co., Houston.
L. H. FLEWELLEN, Chairman, Industrial Accident Board, Austin.
FELIX A. RAYMER, President, Petroleum Casualty Co., Houston.

Virginia

MRS. C. B. HITCH, Richmond.
W. H. NICKELS, JR., Chairman, Department of Workmen's Compensation, Richmond.

Washington

ELMER H. KENNEDY, Supervisor, Rehabilitation Center, Department of Labor and Industries, Olympia.

West Virginia

DR. JOHN P. BRICK, Chairman, Occupational Disease Medical Board, Charleston
FRANKLIN W. KERN, Executive Secretary, Workmen's Compensation Fund, Charleston.
RUDOLPH J. MURENSKY, Legal Assistant, Workmen's Compensation Fund, Charleston.
GEORGE W. STOKES, Commissioner, Workmen's Compensation Fund, Charleston

Wisconsin

Stephen J. Haddock, Attorney, Milwaukee.
B. E. Kuechle, Vice President, Employers Mutuals, Wausau.
Mortimer Levitan, Assistant Attorney General, Madison.
J. E. Lohrer, Claims Counsel, Employers Mutuals, Wausau.
Harry A. Nelson, Director, Workmen's Compensation, Industrial Commission, Madison.
Votta Wanders, Chairman, Industrial Commission, Madison.

Wyoming

William P. Perry, Manager, Workmen's Compensation Commission, Cheyenne.