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Alberta Occupational Medicine Newsletter: Summer 1985

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ALBERTA OCCUPATIONAL MEDICINE NEWSLETTER

EDITORIAL COMMENTS

In this issue, the editor has taken the liberty of summarizing a statement from MMWR (Morbidity and Mortality Weekly Report) about the possible relationship of work and *cardiovascular diseases,* and workplace exposures to possible etiologic agents. The issue is far from settled, but certainly contains "food for thought." As well, this issue of the Newsletter contains a followup article to one which appeared in Vol. III, No. 1 (Spring 1985), dealing with *Occupational (Industrial) Hygiene,* written by Mr. Paul Gray. This article completes the series on aspects of industrial hygiene for those readers who have enquired about the role of this group of specialists in the workplace setting.

Again, our thanks is expressed to all our contributors — new and regular. We encourage our readers to correspond with us regarding: what you find of value in the Newsletter, what your needs are, and requests (or submissions) for future topics.

Have a good summer!

* * * * *

W.M. Csokonay MD, CCFP(C),
DTM&H, FRCP(C)
Editor,
Alberta Occupational Medicine Newsletter
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NOTICE BOARD — UPCOMING MEETINGS AND CONFERENCES

September 1985

Third Annual Conference of the Occupational Medical Association of Canada; Sept. 11-13, 1985; Calgary, Alberta; Contact: Margaret-Anne Stroh; Conference Coordinator, Conference Office, Faculty of Continuing Education, University of Calgary, 2500 University Drive N.W., Calgary, Alberta, T2N 1N4.

FROM THE JOURNALS: CARDIOVASCULAR DISEASES — THE ROLE OF OCCUPATIONS

(This article is taken from the *Morbidity and Mortality Weekly Report,* Vol. 34/no. 16, April 26, 1985, and is part of an ongoing series entitled Leading Work-Related Diseases and Injuries in the USA. References may be found in the original article).

The role of occupation as a factor in cardiovascular disease is far from clear. Most investigators believe that personal risk factors, such as cigarette smoking, blood pressure, diet, personality, and heredity, are more important than environmental factors. Specific data are sparse on the role of occupational factors. Nevertheless, some occupational factors are clearly associated with heart diseases, and evidence on other factors is accumulating. Because heart diseases are still so prevalent identifying and preventing occupational factors that result in even a small increase in the relative risk of cardiovascular disease would involve large numbers of persons. Thus, preventing any occupational contribution to this problem would be an important public health measure.

In 1978, an ad hoc task force was formed by the American Heart Association to review the data regarding the environmental impact on cardiovascular disease. Its report, "the Impact of the Environment on Cardiovascular Disease" was published in 1981. The task force identified and reviewed six environmental factors that have potential impact on cardiovascular health: water hardness; trace elements; inhalant occupational exposure; carbon monoxide; noise and radiofrequency; and physical and psychosocial stress. The workplace is a specific source of potential exposure for all but the first.

A. METALS, DUSTS, TRACE ELEMENTS: The development of congestive heart failure that results from restrictive lung disease (cor pulmonale) has been observed in studies of occupational respiratory diseases, such as chronic beryllium disease and silicosis. Other metals, such as antimony, cobalt and lead, have been implicated as possible causes of cardiovascular diseases.

B. OCCUPATIONAL INHALANTS AND OTHER CHEMICAL EXPOSURES: These include:

1. *Carbon monoxide:* CO decreases the oxygen-carrying capacity of hemoglobin and thus reduces the oxygen supply available to heart muscle and other tissues. In persons with preexisting coronary artery disease, occupational exposures to carbon monoxide may precipitate acute cardiovascular events, such as untoward changes in cardiac rhythm. In animal studies, life-threatening arrhythmias, such as ventricular tachycardia and ventricular fibrillation, have been observed in response to exposures to CO that produced a carboxyhemoglobin concentration of 9% and above.

In one study of workers, short-term exposure to CO at levels within the current Occupational Safety and Health Administration permissible exposure limit (50ppm) was associated with decreased exercise tolerance and EKG evidence of myocardial ischemia. In another study among Finnish foundry workers exposed to CO, the overall prevalence of angina pectoris was increased: this was most pronounced among workers who also smoked. Among British steelworkers, investigators found end-of-shift carboxyhemoglobin saturations substantially higher among blast furnace workers than among steelworkers in other jobs. This was also observed for both smoking and nonsmoking employees.

2. *Carbon disulfide:* Carbon disulfide, a widely used solvent, has been shown to increase the risk of cardiovascular disorders, including coronary artery disease and hypertension, in both epidemiologic and experimental studies. It has also been shown to pose a significant risk for coronary death. The atherogenic potential of carbon disulfide involves both cerebrovascular and cardiovascular systems.

3. *Halogenated hydrocarbons:* Acute exposures to many common industrial solvents (eg choroform, trichloroethylene) and fluorocarbon aerosol propellants have precipitated sudden death probably due to cardiac arrhyth-

mias in workers exposed at high levels. Other common aerosols or solvents may be arrhythmogenic at concentrations permitted by current occupational exposure standards. A recent study of pathologists exposed to monochlorodifluoromethane (a fluorocarbon aerosol propellant) in hospitals showed an increased incidence of "palpitations" at levels of exposure far below the recommended standard.

4. *Nitroglycerin and nitrates:* Workers exposed to nitroglycerin and nitrates during the manufacture of explosives experienced increased risk of cardiac chest pain, myocardial infarction and sudden death, particularly after a period of time away from exposure. The mechanism is thought to be "rebound vasospasm" as a consequence of withdrawal from exposure.

5. *Noise:* Tens of millions of workers are exposed to high levels of sustained and/or intermittent noise in the workplace. A number of studies have demonstrated that single exposures to noise cause transient increases in blood pressure. Chronic exposure to occupational noise has also been associated with sustained increases in blood pressure, particularly in workers with noise-induced hearing loss. Increases in serum cholesterol and changes in circulating hormones have been observed in humans in association with noise. In studies of animals, abnormalities in platelet aggregation have been documented following exposure to noise.

6. *Psychosocial stress:* Stress has long been thought to adversely affect the cardiovascular system. A relationship between psychologic factors and cardiovascular disease is supported by the correlation between "Type A personality" and such disorders. A 1976 assessment suggested the "work-overload", role conflicts, and thwarted career goals were related to evidence of cardiovascular disease. A prospective evaluation of health changes among air traffic controllers, published 1978, showed an increased prevalence of hypertension among controllers, attributed by the authors to difficulties in coping with working conditions.

An updated analysis of the Framingham heart study in 1980, indicated that rates of coronary heart disease were nearly twofold greater among women employed in clerical jobs than among housewives. Significant predictors of the risk of coronary heart disease included a "nonsupportive supervisor" and decreased job mobility. Occupation may also affect the risk of cardiovascular disease in a spouse. Men whose wives worked in white collar jobs were observed to experience heart disease at a rate three times greater than men whose wives worked in clerical or blue collar jobs or were housewives. Similarly, men appeared to have a higher risk of cardiovascular disease if they had well-educated, working wives who reported nonsupportive supervisors or few opportunities for job promotion. These and similar results suggest that adjustments to the conflicting demands of job and family may be important factors in the risk of cardiovascular disease.

Recent evaluations of data from a large random sample of the Swedish working male population and from other surveys, also suggest that certain working conditions, such as limited autonomy and heavy workloads, are associated with clinical indicators of coronary heart disease.

Epidemiologic studies are clearly needed to define the significance of these and other occupational stress factors in the etiology of cardiovascular diseases. Such physical stresses as noise, vibration, and heat also merit investigation for possible interaction with the psychologic risk factors of cardiovascular diseases.

OCCUPATIONAL (INDUSTRIAL) HYGIENE, ITS ROLE IN OCCUPATIONAL HEALTH PART II

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TLV's and OEL's (Threshold Limit Values, and Occupational Exposure Limits) — THE REGULATED LIMITS OF EXPOSURE

To assess the potential hazard of exposure to a substance, comparison with an established, chemical-specific numerical standard is required. To determine such a standard, the best available data are considered and may, therefore, vary as new information becomes available.

This article discusses the regulatory limits utilized by Occupational Hygienists when conducting surveys to appraise and evaluate employees' health risks.

A. HISTORY

In 1942, at the fifth annual meeting of the American Conference of Governmental Industrial Hygienists (ACGIH), the concept of establishing a committee to determine standards for air contaminants in the workplace was considered. The committee later became known as the Threshold Limits Committee.

The first compilation of occupational hygiene numerical exposure standards in the U.S. was compiled in 1945 when Warren Cook published a list of maximum allowable concentrations for several industrial atmosphere contaminants.

After many years of researching/developing and revising numerical standards, and applying them in workplace contaminant evaluation, the ACGIH Threshold Limit Values (TLV's) were used, in conjunction with the Walsh-Healy (Public Contracts) Act, as initial standards for the Occupational Safety and Health Act.

The ACGIH TLV's are used extensively throughout Canada and, although a number of provinces have changed the name of these standards (in Alberta, they are called Occupational Exposure Limits (OEL's), the values involved are basically the same. The Alberta Occupational Health and Safety Division has the authority, with adequate substantiation, to revise these values as they best see fit. The Alberta OEL's are published in the Chemical Hazards Regulation under the Occupational Health and Safety Act.

B. DEFINITION OF STANDARDS

The terms Threshold Limit Value (TLV) and Occupational Exposure Limit (OEL) can be considered synonymous. Three variations of these limits exist, and are defined as:

1. *TLV-TWA (TLV-Time Weighted Average)* **or** Prescribed 8-hour OEL:
   — the time-weighted average concentration for a normal 8-hour workday and 40-hour workweek, to which nearly all workers may be repeatedly exposed, day after day, without adverse effect.
2. *TLV-STEL (TLV-Short Term Exposure Limit)* **or** Prescribed 15-minute OEL:
   — the 15-minute time-weighted average concentration to which a worker can be exposed 4 times per day providing at least one hour exists between each exposure above the TWA but below the STEL. In addition, the 8-hour TLV-TWA (8-hour OEL) is not exceeded.
   — this limit allows workers to be exposed to contaminant concentrations in excess of the 8-hour TWA limit for a short period of time but are established so that the worker will not suffer:
   1) irritation
   2) chronic or irreversible tissue damage
   3) narcosis of sufficient degree to increase the likelihood of accidental injury, impair self-rescue or materially reduce work efficiency.
3. *TLV-C (TLV-Ceiling)* **or** Ceiling OEL:
   — the maximum concentration to which a worker can be exposed and which cannot, even instantaneously, be exceeded without the wearing of proper personnal protective equipment (eg. respirators).

Some chemical substances such as irritant gases (formaldehyde, ethylene glycol vapour, hydrogen cyanide etc.) have only a single limit (the ceiling limit) listed in the tables. For most other chemicals, an 8-hour TWA (OEL) and/or a 15-minute STEL (OEL) value exists, depending upon the chemicals physiological action. A potential hazard exists when any of these three categories of TLV's (OEL's) is exceeded.

It is imperative to understand that these standards are only guidelines. The variability and imprecise nature of a biological response, compounded by the subjective measurement which occurs when analyzing biological and environmental data, allows us only to set standards that are as accurate as practicable. This means that if the TLV-TWA (8-hour OEL) is 100 ppm; 99 ppm is not always "safe" and 101 ppm is not always "harmful". The 100 ppm standard is a guideline only and any exposures above this level indicate that some sort of control be implemented.

From a legal point of view however, the published standards do represent more precise markers. The Occupational Health and Safety Division use them although still as guidelines, to determine whether or not a worker's exposure to a chemical is excessive.

Individual variation to chemical insults significantly affect a person's degree of reaction to a contaminant. Standards are set so that the majority of workers will show no effect. Obviously, the ideal is to set a limit where no person will be negatively affected but his would impose too severe restrictions upon industry. The standards set will protect, as the definitions noted, "nearly all workers," even on repeated exposure. The working population will, to any given substance, show degrees of

response which, if plotted, would approach a bell distribution. Approximately 5 percent of the population will be hypersensitive, another 5 percent will be hyposensitive, and the remainder will react in approximately the same manner. The standards then, should protect at least 95 percent of the population — the average group (90%) and the hyposensitive group (5%). The hypersensitive individuals may or may not react at or below the set standard. If such a reaction does occur, the occupational hygienist must reply on the third primary job function — control of the hazard by engineering, administrative or personal protective equipment controls.

* * * * *

SETTING THE STANDARD

A. Extrapolation and Experimentation

Before workers are exposed to a chemical agent in the workplace, it is necessary to know what toxic properties that agent may possess so that appropriate control procedures and medical protocols can be established. Where new chemicals are used, there is frequently little or no information available upon which to act.

Toxicologists rollow two routes to ascertain some of this information: 1) extrapolation — the quality of the response to the chemical is assumed to be analogous to that produced by similarly structured chemicals; 2) experimentation — conducting animal toxicological experimentation under controlled conditions resembling the conditions of industrial usage.

Inconsistent effects, both type and degree, are inherent limitations of the extrapolation procedure. It is interesting to note that approximately one-quarter of all TLV's published by the ACGIH in 1968 were based upon extrapolation.

Standard animal toxicological experimentation involves; 1) skin testing for determination of possible absorption and systemic toxicity; 2) primary irritation or sensitization; and 3) mucous membrane and eye testing to identify similar reaction effects. In addition, animals are exposed to gases, vapours, mists and aerosols of the chemical so as to determine the pulmonary effects and systemic toxicity. Experimentation involving gastro-intestinal and subcutaneous absorption are less frequently used except for range-finding toxicity purposes.

Three *major limitations* of animal experimentation are obvious. *Firstly,* consider the accuracy of the extrapolation to man of the observed toxicological effects. Since man does not react as does a single species of animal to every chemical, it is common practice to study the toxicological effect on numerous animal species. In setting human exposure limit standards, man is normally considered to react in the same manner as does the most sensitive of the animal species tested.

A *second limitation* is the number of animals required to make the toxicological finding statistically significant. Normal biological variance within any one group of animals necessitates an adequate sample. With this in mind, statistical techniques are utilized in determining what confidence limits exist for a given sample size.

A *third limitation* is determining what dose ranges are required during experimentation. Large doses help define which organ/system is susceptible to damage since gross structural change will often occur. The large doses do not however, provide the more specific information regarding functional change in the organ. Through detection of altered organ function prior to pathological organ alteration, the organism has a greater probability of avoiding permanent damage. The functional changes are normally detected through identification of a metabolic alteration of the absorbed chemical agent as produced by one of the detoxification organs. As long as the rate and concentration of chemical impingement on the detoxification organ does not exceed its metabolic capabilities, the organism can cope with the chemical insult. Production of more toxic metabolites than the original chemical by these organs is also of vital importance in the recognitition/evaluation of functional change. Animal experimentation must therefore be designed to identify these parameters, thereby assisting in the definition of a safe "dose."

Neurofunctional response is now being studied with great interest due to the possibility of this response being an early indication of change.

Finally, carcinogenicity, mutagenicity, and teratogenicity potentials must be identified at all dose levels.

B. DATA APPLICATION

As previously noted, environmental standards must be based on the assumption that man responds in a manner similar to the most sensitive species.

By varying the dosage and dose rates during experimentation, a dose-response curve can be established. Again, using the most sensitive animal species, a no-effect level can be predicted for man.

To further decrease the chance of negative toxicological effect in man, a safety factor is incorporated between the dose showing the first effect and the TLV (OEL). The size of the safety factor depends upon the response identified at the lowest effective dose. The TLV (OEL) calculation therefore becomes the lowest effective dose divided by a safety factor.

C. HUMAN DATA

Although animal experimentation has many shortcomings, it is invaluable in the toxicologist's efforts to establish realistic occupational exposure standards. The ability to study contaminant action and evaluate exposures/effects in humans, provides the most useful information in standard development: Such data result from inadvertent (ie. Bhopal, India Union Carbide incident) or intentional experimental exposure (considered only after stringent limits have first been identified through animal experimentation and then, only with volunteers).

The combination of animal toxicology, human exposures, experience, toxicology, and safety factors allow occupational exposure limits to be developed. The variability of these limits, as a whole, is significant but this is expected with the ever-increasing data bank available regarding chemicals in use today.

Experts in industrial hygiene and toxicology review standards set for more than 400 substances annually. On the basis of this continually increasing amount of data available, TLV's are recommended. Interested persons and organizations can comment on the proposed change to a currently listed TLV. These "proposed" additions/changes are listed in the ACGIH TLV Handbook under "Notice of Intended Changes" for a period of 2 years and if, after this length of time no evidence comes to light to cast doubt on the appropriateness of the value, the value is considered for the "Adopted" TLV list. Documentation for each limit set is available in the ACGIH "Documentation of TLV's" book.

SUMMARY

Under the American EPA's Toxic Substances Control Act (TOSCA), a total of between 50,000 and 60,000 materials are identified as being used in industry. The NIOSH Registry of Toxic Effects of Chemical Substances (RTECS) currently lists some 40,000 chemical substances. We now have established standards (TLV/OEL) for 500-600 chemical substances.

The need for additional data to be developed for use in the setting of standards is a gross understatement but many factors such as cost, facilities, human resources etc. limit our ability to greatly improve upon this imposing ratio. Occupational hygienists must therefore use a great degree of care and sound scientific judgement when evaluating personal exposures to all chemical substances. Occupational hygienists are not solely responsible for this evaluation. Significant input is required from all Health professionals, primarily occupational health nurses and physicians, in their role of accurately diagnosing and determining the health status of the worker.

* * * * *

ANNOUNCEMENT

"Impact of Air Toxics on the Quality of Life" is the theme of the 1985 annual meeting of the Pacific Northwest International Section of the Air Pollution Control Association. This reflects the increasing emphasis in society on human health, aesthetics, and those things that are summarized in the expression "Quality of Life".

We would like to share your insights, discoveries, and experience at our annual meeting which has become a valued medium of exchange of ideas between people from the public, government, the academic world, and industry.

Input from the following industries would be most welcome.

Utilities
Sour Gas
Heavy Oil and Oil Sands
Pulp and Paper
Transportation
Potash and Coal
Petrochemicals and Refineries
Hazardous Waste Disposal
Fertilizers and Plastics

Some of the topics suggested are:

Acid Rain
Patterns of Health Effects (Epidemiology)
Greenhouse Effect
Stack Monitoring
Wood Burning
Deposition Modelling
Risk Analysis
Indoor Air Quality
Air Quality and Modelling
Emergency Response
Regional Haze
Weather Modification
Waste Management
Occupational Hygiene
Government Affairs
Air Toxics & Athletic Performance

PLEASE ADDRESS ENQUIRIES TO:

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237 - 4 Ave. S.W., Calgary, Alta. T2P 0H6

MALIGNANT MELANOMA — AN OCCUPATIONAL SKIN CANCER?

PHYSICIANS ARE AT GREATEST RISK FOR THE DEVELOPMENT OF MALIGNANT MELANOMA!!

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Dermatologist, Calgary

Cutaneous malignancies were the first occupationally related cancers to be documented. Sir Percivall Pott recorded the development of scrotal cancers in the chimney sweeps of Great Britain in 1775. Since that time scientists have attempted to link other cutaneous cancers to occupations. The skin is the barrier separating us from our environment and we are in direct contact with a great many chemicals that are potentially carcinogenic. A surprising few have been firmly associated with cutaneous malignancy (Table 1). Ultraviolet light is the agent most commonly suggested as the cause of non-melanoma skin cancers thought to be occupationally related. This is supported by the fact that over 90% of basal cell cancers occur on 10% of the body surface (head and neck) and the incidence of squamous cell carcinomas doubles in incidence with each 10 degree change in latitude as one approaches the equator, clearly outdoor workers are the most susceptible to these carcinogenic effects.

The incidence of malignant melanoma is rapidly rising in our society. The proof of a causative role for ultraviolet light remains elusive and contradictory. The occurence of melanoma as an ultraviolet induced occupationally related malignancy is being investigated. Twenty years ago studies indicated that the risk of melanoma increased with outdoor work. Recent epidemiologic studies refute this and suggest the greatest risk is among indoor workers of high social class. The evidence that is being presented is unexpected and is of great interest to us all.

In New Zealand a study of 500 Melanoma patients found the incidence of Melanoma highest among professional, technical, administrative and managerial workers with the lowest incidence among production, transport workers and laborers. These differences appeared to be due to an increase in risk with higher socioeconomic status. They did not find a difference in risk between outdoor and indoor workers of similar social class. In Western Australia the incidence of Melanoma from 1975-1976 was highest in residents of high social class areas. These findings are supported by similar large studies reviewing data from England and North America.

Other interesting statistics have been generated by these studies. One study found that when women with malignant Melanoma were classified by the occupation of their husbands they showed a similar relationship to social status. It has also been shown that the prognosis for unskilled patients is much worse than that for other occupations.

How are these findings to be explained? The experts disagree. Could it be related to the recreational sun exposure of relatively untanned skin? Is it related to the different health care utilization patterns among different social classes? Do the office buildings that we work in contain yet another potential carcinogen?

TABLE 1.
CHEMICAL AGENTS ASSOCIATED WITH SKIN CANCER

AGENT	OCCUPATION
Machine(Cutting) Oil	Machinist
Coal Tar, Pitch	Coking
Soot	Chimney Sweep
Chloroprene	Synthetic Rubber
PCB(Melanoma)	Petrochemical
Vinyl Chloride(Melanoma)	Manufacture
Arsenic	Agriculture

A study published in the Lancet entitled "Malignant Melanoma and Exposure to Fluorescent Lighting at Work" is worth considering because of the recent widespread concern about our working environments. In a study of 274 female Melanoma patients residing in New South Wales, Australia exposure to florescent light at work was associated with a doubling of Melanoma risk when compared with matched controls. The risk increased with increased duration of exposure and was higher in women who had worked mainly in offices than in women who worked indoors but not in offices. These findings could not be explained any other factor.

When the distribution of lesions was related to the type of work and fluorescent light exposure an increased number of lesions occurred on the trunk in indoor office workers with florescent light exposure. The recreational sun exposure habits were assessed and surprisingly the Melanoma risk was lower in those women who had apparently been most exposed to sunlight. Women born in Australia who spent a lot of time outdoors and whose main outdoor activity at age 20 was sunbathing had the lowest risk. Those women who reported a greater than average number of nevi had an increased Melanoma risk.

A review of 27 men and age matched controls revealed a 4-fold increase in Melanoma risk in those individuals with fluorescent light exposure of greater than 10 years.

Fluorescent lights do emit wavelengths of light that extend into the ultraviolet spectrum and they are capable of producing photosensitive skin reactions. The weave of clothing is important in its ultraviolet blocking capabilities and light, loosely woven blouses and shirts offer only partial protection. Do these factors explain the increased risk of developing a truncal Melanoma with an increased exposure to fluorescent lighting? Should one encourage a "truncal" suntan by having patients wear potent sunscreens on their faces and less potent ones on the trunk? Should our preemployment screening assess the number of nevi and preclude those individuals with an above average number from office work under fluorescent lighting? These questions remain unanswered.

Clean air, light and water are necessary to sustain life. Our indoor air is polluted and our indoor light may be carcinogenic. How is your water?

Indoor office work is a dangerous occupation!!

SILICA AND CANCER

A SUMMARY OF CURRENT EVIDENCE

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University of Alberta Faculty of Medicine

Until quite recently, the recognized link between silica exposure and cancer risk could be summarized succinctly: "There isn't any!" That conventional view has been challenged by toxicologic and epidemiologic investigations just now becoming available. Most of these data have been presented in one of two forums in 1984: the International Conference on Silica, Silicosis, and Cancer, in Chapel Hill, North Carolina, or the International Epidemiology Association Triennial meeting in Vancouver. A book will soon appear incorporating the proceedings of these meetings; the book will also preview papers to be presented in a risk assessment symposium to be convened in May by the American Association for the Advancement of Science.

The principal findings to be highlighted in the book, entitled *Silica, Silicosis, and Cancer* (Praeger), are summarized on page 5.

Discussion-Experimental Results

The experimental results of Holland et al. (1983), Broth et al. (1984), and Dagle et al. (1984) are intriguing for several reasons.

1) These experiments are the first demonstration that silica (Min-U-Sil) produces ~~respiratory tumors~~ in animals without prior initiation.
2) The results were all observed in animals exposed to Min-U-Sil as a positive control for fibrosis.
3) The doses were very high, especially in the inhalation studies.
4) This is the first time that tumors were produced using inhalation, paralleling the route of exposure in humans.
5) The excess of tumors appears to predominate in female rats.
6) The results of Holland et al. were not observed in hamsters (Holland et al., 1983); the results of Wagner et al. (1980) could not be reproduced in mice (Wagner — personal communication).
7) A higher proportion of squamous cell carcinomas (compared to adenocarcinomas) was produced in these positive studies of silica.

Discussion-Epidemiologic Findings

Several aspects of studies of dusty industries and of silicotics tend to question silica's etiologic role in the observed excesses of lung cancer.

1) Smoking is unadjusted in almost every study, although smoking plays an insignificant role in the risk of silicosis (Rice et al., 1984; Sluis-Cremer, 1984). However, as observed by Fletcher and Ades (1984) in their study of foundry workers, nearly the total cohort would need to be smokers to produce on SMR of 512 ($p < 0.001$).
2) Foundry workers, besides being exposed to silica, work in a complex environment

Evidence From Bioassays

Very recent research indicates that silica (in the form of Min-U-Sil, a highly purified quartz used for making glass), *without* initiation from benzo [a] pyrene, is a carcinogen and a mutagen. The evidence is summarized in Table 1.

Table 1.
SUMMARY OF POSITIVE TRANSFORMATION/TUMORIGENESIS STUDIES OF MIN-U-SIL

Authors (date)	Route of Administration	Dose	Species	Results vs. Controls
Hestergerg et al. (1984)	Mixed with cells in culture	Between 2 and 200 mg/cm^2	Syrian hamster embryo	Positive but much less than asbestos or glass fibers
Groth et al. (1984)	Single dose by intratrachael injection	20 mg	male Fischer 344 rats	13% lung tumor vs 0% ($p=0.0007$)
Holland et al. (1983)	Intratrachael installation•	7 mg in saline for 10 weeks	Sprague-Dawley rats	15% lung tumors vs 0%
	Nose-only inhalation	12 mg/m^3 6 hrs/day 4 days/wk for 24 months	Fischer 344 female rats	27% lung tumors vs 3%
Dagle et al. (1984)	Whole body inhalation	50 mg/m^3 6 hrs/day 5 days/wk for 24 months	Fischer 344 rats	19% and 2% lung tumors in females and males vs 0%
Wagner et al. (1980)	Intrapleural Instillation	20 mg. suspended in saline	Wistar rats	Range of 34 to 5% malignant histocytic lymphomas vs 8 to 0%.

Dusty Trades Evidence

Workers in occupations characterized by high silica exposure (excluding coal and uranium mining and asbestos work) have excess mortality from silicosis, respiratory tuberculosis and other nonmalignant respiratory disease, although the risk seems to be declining in recent years due to improved ventilation. Paralleling these excesses are significantly elevated relative risks for lung cancer ranging from 2 to 7. Industries and occupations where these excesses have been reported include sandblasting, foundries, metal ore mining, firebrick making, ceramics manufacture, and quarrying and tunneling (see Table 2).

Table 2.
INDUSTRIES AND OCCUPATIONS WITH SIGNIFICANT RISK RATIOS FOR LUNG CANCER

Industries/Occupation	Range of Risk Ratios	Comment
Sandblasting	1.9 (NS) to 3.8	Possible confounding by asb in shipyards
Foundry (ferrous and nonferrous)	1.5 to 7.1	Stomach cancer excesses als
Crane operators	1.2 (NS) to 7.1	7.1 based on ‹5 deaths
Fettlers	2.0 to 3.1	Highly exposed to particulat
Molders	1.3 (NS) to 3.1	Huge variety of mold binder
Casters	1.5 (NS) to 4.6	
Masons/furnace repairmen	2.0 to 2.1	Highly exposed to silica bric
Metal ore mining	1.3 to 3.5	Highest in chrome mine; possible confounding by rad daughter exposure
Gold mining	1.0 (NS) to 3.1	Stomach and hematopoietic cancer excesses also found
Firebrick making	1.8 to 2.0	
Ceramics	1.8	
Granite/crushed stone/tunneling	1.0 (NS) to 1.6	Possible excess stomach can

Adapted from Goldsmith et al., 1982 and Goldsmith et al., 1984.
[NS = not significant statistically]

Silicosis Evidence

Only since 1980 has there been any followup of silicotics. However, every study has demonstrated that workers disabled with silicosis have a significant lung cancer risk of between 1.3 and 5.9. These results are summarized in Table 3.

Table 3.
SIGNIFICANT LUNG CANCER EXCESSES AMONG SILICOTICS

Author (date)	Risk Ratio	Comment
	(Confidence interval not available)	
Westerholm (1980); Westerholm et al. (1984), Sweden	5.9	Mining, quarrying and tunneling, (MQT) 1931-48
	3.8	MQT, 1949-69
	5.4	MQT, 1961-80
	2.2	Steel and iron industries, (SII) 1949-69
	3.8	SII, 1961-80
Finkelstein et al. (1982), Ontario, Canada	2.0	Overall, 1940-75
	3.0	1940-49
	2.0	1950-59
Schüler and Rüttner (1984), Switzerland	2.3	All silicotics, 1960-78
	3.3	Foundry
	2.3	Miners
	2.4	Other occupations
Neuberger et al. (1984), Austria	1.40	Overall, 1955-79
	1.31	1955-59
	1.32	1960-64
	1.32	1965-69
	1.38	1970-74
	1.42	1975-79
Gudbergsson et al. (1983), Finland	3.0	Overall, 1964-74

containing carcinogens such as polycyclic aromatic hydrocarbons, fumes from a variety of mold binders, and metallic oxides (Palmer and Scott, 1984).

3) Underground metal ore miners are likely to have exposure to radon daughters, and thus increase their risk of lung cancer (Archer et al., 1984).

4) Excess lung cancer among sandblasters, especially those employed in shipyards, may be confounded by asbestos exposure.

5) Gold miners, usually working with highly silicaceous ores, have a mixed lung cancer risk profile with elevations reported in Ontario, Australia, and USSR but not in South Dakota or South Africa (summarized in Goldsmith et al., 1984).

6) In occupations where silica exposure predominates, the risk is slightly elevated among tunnel and ceramic workers but not elevated in Vermont granite workers.

7) Cohorts of dusty trades workers and those with silicosis are selected populations; are the cancer risks higher or lower because of their being selected and what are the rates for ex-workers or undiagnosed silicotics?

8) In occupations characterized by excess mortality from silicosis and other non-malignant respiratory disease, what are the effects among smoking silica-exposed workers (Goldsmith and Guidotti, 1984).

REFERENCES AVAILABLE on request.

SPONDYLOLISTHESIS — ETIOLOGY & PATHOGENESIS — "STATE OF THE ART"

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Introduction

The inter-relationship of pre-existing back disease and work related injury to the spine remains a complicated and confusing issue. When faced with the question of the relative roles of a work related trauma and a diagnosis of spondylolisthesis, a better understanding of spondylolisthesis would be helpful in clarifying the matter.

Definition

Spondylolisthesis is forward movement of the body of one of the lower lumbar vertebrae on the vertebra below, or upon the sacrum. The condition is staged according to the percentage of slippage that has occurred.

General

Because of the complexity of spondylolisthesis, signs and symptoms will not be described separately but will be considered under the different forms of the condition. (See classification below) Treatment must be directed at the cause of the symptoms rather than the radiological signs of spondylolisthesis.

Classification

Because of the forwardly declining plane of the lower lumbar intervertebral discs, there is a gravitational shear strain, to move the lower lumbar vertebrae forward. At the same time, there are normal "blocks" which prevent this forward slippage. These include:

A. The posterior facet joints
B. An intact neural arch and pedicle
C. The bonding of the intervertebral discs to the vertebral bodies at the cartilagenous end plates

Spondylolisthesis can be classified according to the breakdown of the normal "blocking" mechanism.

1. Congenital malformations
2. Abnormalities of the neural arch
3. Degenerative changes at the intervertebral disc and facet joints

Before discussing these three different types, it should be pointed out that various forces play a part. These include:

a. Acute or repetitive hyperextension injuries
b. Direct blows to the posterior aspect of the lumbar spinal curve
c. Postural exaggeration of the normal lumbar lordosis
d. Chronic repetitive lifting trauma
e. Gravitational force of daily weight bearing

1. *Congenital Malformations*

Congenital abnormalities of the lumbar spine may range from minor findings such as spina bifida or poorly developed facet joints to major developmental defects in which scoliosis or neurological problems predominate. These congenital abnormalities do not play a major role in the etiology of backache in adult patients with spondylolisthesis.

2. *Abnormalities of the Neural Arch*

A. **Attenuation of the pedicles or pars interarticularis.**

Normal bone plasticity resists stretching. Attenuation, as the result of increased stresses early in life, may occur with disruption of the hyaline cartilage end plates. This is most likely to occur prior to skeletal maturation.

B. **Spondylolysis**

The cause of the bony defect of the pars interarticularis remains largely unknown.

In some instances stress fractures may occur at this stie as the result of the forces outlined above. When stress fractures occur, these almost always unite satisfactorily after a period of rest and immobilization.

It is believed that most cases of spondylolysis with a fibrous syndesmosis occur prior to skeletal maturation and are often asymptomatic. The condition is usually not diagnosed until the third or fourth decade when a patient presents with symptoms of backache. In these cases the spondylolysis is probably an incidental finding.

Special care should be taken when evaluating the x-ray finding of spondylolysis. All to often the physician is relieved to find "a radiological reason" for the patient's backache. Statistical evidence suggests that the incidence of spondylolysis does not parallel the incidence of back symptomatology. Some Eskimo communities have a 50% incidence of spondylolysis, but are by no means incapacitated by backache to the same extent.

Studies have suggested a high probability of an association between spondylolysis and backache in individuals under the age of 26 years. Such a relationship is reduced to a slight possibility between the ages of 26 and 40. A relationship is quite unlikely in individuals 40 years and older.

A firm syndesmosis seems more likely with advancing age whereas a more loose fibrous union in younger patients may be associated with some degree of instability, leading to pain following repetitive strains.

Forward slippage rarely increases after the age of 20 and will not occur without degenerative changes in the disc below the affected vertebra. Nerve root entrapment may occur either as the result of anatomical slippage (spondylolisthesis) or disc prolapse. Instability, disc degeneration or nerve root entrapment may cause pain.

It is interesting to note that disc degeneration is more common at the level above the spondylolisthesis and therefore probably entirely unrelated to the condition.

In summary one should remember that many cases of spondylolisthesis are entirely asymptomatic (moreso in older patients) and another cause for the backache should be sought. Surgery should be directed at the cause of pain and not simply be performed because of a radiological diagnosis of spondylolysis.

C. **Degenerative Spondylolisthesis**

This is most frequently encountered at the mobile section of the lumbar spine (L4-5). There is no associated defect in the neural arch, but the condition is frequently associated with fairly marked instability of the spinal segment. It occurs more often in females and the onset is frequently not until the 5th or 6th decade.

There is always marked disc degeneration and facet joint damage. Nerve root entrapment may occur as a combination of disc bulging, instability, loss of disc height and osteophyte formation (both at the disc margins and around the facet joints).

The treatment of degenerative spondylolisthesis is clearly the treatment of degenerative back conditions, and should be directed at the cause of the pain rather than the radiological diagnosis of spondylolisthesis.