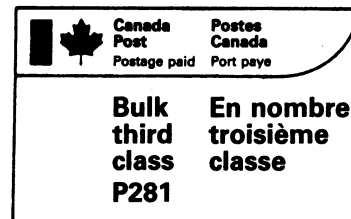
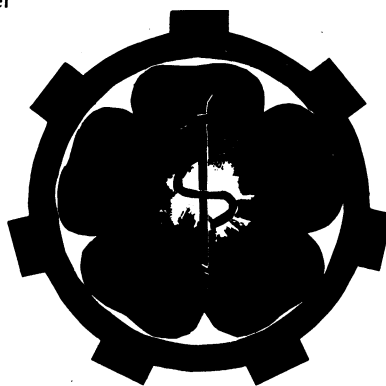


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**Vol. VI, No. 3**

**February 1989**

# ALBERTA OCCUPATIONAL MEDICINE NEWSLETTER

## EDITORIAL COMMENTS

One of the most common health concerns with occupational exposures is their potential for causing cancer. While epidemiologic studies have demonstrated the role of such agents as asbestos and benzene in occupational cancers, these studies are purely observational, and rely on information on exposures to agents already in the workplace. Thus, an important goal of occupational health research is to attempt to *predict* which agents may be carcinogenic, thus preventing any exposure to such agents. Doug Hamm's article is a "user friendly" overview of the biochemistry of carcinogenesis, which relates to this goal.

This newsletter also highlights information available from sources in Alberta. Brian Alleyne has kindly allowed us to print a summary of his reports on Auto-body Shop Workers and the Silicosis/Pneumocosis project. Because these data are collected from Alberta workers, they are of particular value for physicians in the province. In addition, we have printed excerpts of a book review of Tee Guidotti's *Occupational Health Syllabus*.

We have had many requests for references from recent Newsletter issues, indicating a high level of interest in some of the topics presented. These have been sent along to all requesting them. If you wish to comment or obtain further information or references on any of the articles in the Newsletter, please contact the editorial office.

Heather Bryant, M.D., Ph.D., FRCPC

## NEW PUBLICATIONS FROM ALBERTA HEALTH, OCCUPATIONAL HEALTH BRANCH

A recent Newsletter noted the availability of a Medical Guideline regarding "Medical Assessment of the Pregnant Worker." A recent guideline takes the reproductive theme to the consideration of infertility and subfertility. The new guideline (MSB/38) is entitled:

### . Workplace Hazards Affecting Fertility

and can be obtained from:

Alberta Community and  
 Occupational Health  
 Occupational Health Branch  
 10709 Jasper Avenue  
 Edmonton, Alberta  
 T5J 3N3

\* \* \*

## UPCOMING CONFERENCES

### Managing Occupational Health and Safety (OH&S) in the 1990's: Cost Effective Strategies.

Ottawa, Ontario,  
 April 12-14, 1989.

Contact:

Continuing Education  
 University of Ottawa  
 5 Calixa Lavellee/Priv.  
 Ottawa, Ontario K1N 6N5

\* \* \*

### Introduction to Industrial Hygiene.

Seattle, Washington  
 April 5-6, 1989

Contact:

Northwest Center for Occupational  
 Health and Safety  
 Department of Environmental Health,  
 SC-34  
 University of Washington  
 Seattle, WA 98195

\* \* \*

### Biological Monitoring Laboratory.

Seattle, Washington  
 April 7, 1989

Contact:

Northwest Center for Occupational  
 Health and Safety  
 Department of Environmental Health,  
 SC-34  
 University of Washington  
 Seattle, WA 98195

\* \* \*

### Ergonomics.

Seattle, Washington  
 April 21-21, 1989

Contact:

Northwest Center for Occupational  
 Health and Safety  
 Department of Environmental Health,  
 SC-34  
 University of Washington  
 Seattle, WA 98195

\* \* \*

Prepared in the Department of Community Health Sciences, Faculty of Medicine  
 The University of Calgary, through funding by Alberta Occupational Health and Safety

# OCCUPATIONAL CANCER: DNA Adducts and Prospects for "Molecular Epidemiology"

R. Douglas Hamm, M.D., C.C.F.P.\*

The first recognition of an association between occupation and cancer is generally attributed to the English physician, Sir Percival Pott. In 1775, he described scrotal cancer (known as "soot wart") among London chimney sweepers. A century later, the substance we know as DNA (deoxyribonucleic acid) was discovered at the University of Tübingen by Friedrich Miescher. In 1869, he described a new cellular component which he called "nuclein" and the era of nucleic acid research began. However, almost another century passed before the molecular geometry of DNA was determined by Francis Crick and James Watson at Cambridge University. Their proposal for the DNA "double helix" in 1953 is considered the beginning of the science of molecular biology.

As we enter our second century of DNA research and our third century of occupational cancer epidemiology, we are finding these disciplines converging in what is becoming known as "molecular epidemiology". This article will present a conceptual framework for this "newcomer on the block" and will consider some prospects for its application in occupational cancer control from the perspective of DNA adduct formation.

## CHEMICAL CARCINOGENS AND DNA

There are at least 23 chemicals known to be causally associated with human cancer according to the International Agency for Research on Cancer, (see their "Group 1" category in Table 1). Over 60 other chemicals (IARC "Group 2") are considered as probable human carcinogens (cancer causing agents). As the number of natural and man-made chemicals in industry and the environment has rapidly increased, so has the need for ways to prospectively evaluate their human carcinogenic impact.

Based on the fact that most known human carcinogens are genotoxic (injurious to the genetic apparatus) *in vitro* and *in vivo*, many microbial and animal short-term genotoxicity tests have been developed to "screen" for potential human carcinogens. The well known "Ames test" (*Salmonella typhimurium* mutagenesis assay) was one of the first of these. It should be noted that the Ames test has shown genotoxicity for only 18 of the 23 known human carcinogens as Table 1 (Shelby, 1988) indicates. For example, despite the fact that benzene and asbestos are known carcinogens, they do not produce mutagenesis in the Ames test, demonstrating that most, but not all carcinogens are mutagens (mutation inducing agents).

## DNA Specific Reactivity

Short-term genotoxicity tests appear to be most predictive for those chemicals which are DNA-reactive, i.e. they form so-called "reactive electrophiles", either directly or after activation by host metabolism (there is up to 100-fold variation among individuals with respect to the metabolism of specific carcinogens). Covalent bonding of electrophiles to DNA moieties (usually bases) creates "DNA adducts". For example, after bioactivation, vinyl chloride ( $\text{CH}_2=\text{CHCl}$ ) produces an adduct at guanine's N-7, a cyclic bridge between adenine's N-1 and C-6-amino, and another adduct bridging cytosine's N-3 and C-4-amino (refer to the accompanying diagram of DNA in Figure 2 for these sites). Adduct concentrations have been estimated at about one per  $10^5$  to  $10^6$  nucleotides (the human cell contains about  $10^9$  DNA nucleotides) resulting in from 1,000 to 10,000 DNA adducts per cell. Adduct removal requires enzymatic DNA repair processes which, in some cases, produce base mispairs and potential cancer initiation.

The analysis of human and animal genotoxic agents has shown that DNA reactivity is associated with families of chemical groups. Structure activity relationship (SAR) research has been developed for predicting carcinogen potential, a kind of chemical "guilt by association". Using SAR, a composite hypothetical super-carcinogen has been contrived as shown in Figure 1 (adapted from Ashby and Tennant, 1988).

## DNA Structure

Double stranded DNA (dsDNA) is a linear bipolymer composed of four different nucleotide units in varying arrangements, all held in sequence by a phosphodiester "backbone". Each nucleotide is composed of a *purine* (either adenine, 'A', or guanine, 'G') or *pyrimidine* (either cytosine, 'C', or thymine, 'T') *base*, a 2-deoxyribose *sugar* and a terminal *phosphate* molecule. The two polynucleotide strands are held in precise register by hydrogen bonding between the opposing nucleotide bases. It is important to remember that the nucleotides must pair along the "inside" of the DNA molecule so that G is always matched with C (as G-C or C-G) and T with A (as A-T or T-A). The two strands of DNA are thus complementary, a critical aspect of DNA replication. Figure 2 shows how the base pairs "fit" within the double helix and suggests that helical deformation can result from mispairing or steric hindrance due to adduct formation, e.g. alkylation of bases.

DNA base mispairing is a result of either 1) "transitions" due to switching of purines (i.e. G to A or A to G) or pyrimidines (i.e. T to C or C to T) or 2) "transversions"

due to switching of purines to pyrimidines (i.e. G or A to C or T) or vice versa. In its natural state (the "B" form), the two intertwined single stranded DNA (ssDNA) chains form a right handed dsDNA helix with 10 base pairs per "turn" and alternating major and minor grooves along the "outside" of the helix. This helical structure can affect adduct removal e.g. the guanine C-2-amino adduct of benzo(a)-pyrene lies in the minor (narrow) groove whereas the adenine C-6-amino adduct lies in the major groove where it is more susceptible to enzymatic repair.

## DNA 'AP Sites'

We have come to recognize that cellular DNA is not an inherently stable macromolecule. Biochemists even tell us that DNA "breathes" (the helix opens and closes). It is now known that spontaneous *depurination* (i.e. loss of G or A) in dsDNA occurs at 10,000 events/cell/day and dsDNA *depyrimidination* (loss of C or T) occurs at 500 events/cell/day. *Deamination* (loss of G's 2-amino, A's 6-amino or C's 4-amino) occurs at 170 events/cell/day. These rates are greatly increased in ssDNA. Alkylation (e.g. by methylating or ethylating agents) of DNA can increase the rates of depurination/depyrimidination by as much as six orders of magnitude! Depurination/depyrimidination results in *apurinic/apyridinimic sites* ("AP sites") thought to be common intermediates in chemical mutagenesis.

We now know that the N-glycosidic bonds holding the bases to the phosphate-sugar "scaffold" are weakened by base modifications such as ring opening (e.g. at purine 7-8 bonds) and adduct formation (especially at the N-7 purine sites). About a dozen highly active sites have been identified on the nucleotide bases. The most reactive sites (see Figure 2) providing adduct formation are found at:

Guanine's	N-1, C-2-amino, N-3, C-6-oxygen, N-7,
Adenine's	N-1, N-3, C-6-amino, N-7,
Cytosine's	N-3.

Sites of lesser reactivity are found at cytosine's exocyclic oxygen and amino groups and thymine's exocyclic oxygen (favored sites of alkylation). By looking at the diagram, it is apparent that many of these DNA-reactive sites are those involved in complementary base pairing, e.g., the exocyclic groups. It has been determined that the relative rates of N-glycosidic bond cleavage are  $\text{G} \gg \text{A} \gg \text{C} \gg \text{T}$ . Thus purines are favored in AP site formation.

## DNA Repair

In order to correct for AP sites, base mispairing, and adduct formation pro-

duced spontaneously or by chemical damage, the cell must continuously monitor and repair its DNA. There appear to be about 100 DNA repair enzyme molecules per cell but such levels can rise under genotoxic stress. It is thought that the cell's repair enzymes recognize structural distortions in the DNA helix produced by adducts and AP sites. So-called "intercalation" of adducts between base pairs (like adding a coin to a stack) may produce "frame shift" mutations. Cross-linking and strand scission pose even more difficult repair problems.

DNA excision repair enzymes may preferentially recognize certain adducts, perhaps because of differences in helix distortion. Purine (i.e. A and G) C-8 adducts appear to present more conformational "signal" to repair enzymes whereas G's C-2-amino and A's C-6-amino adducts are less recognized, perhaps because of being more "internal". The C-8 adducts are removed relatively rapidly but the internal adducts tend to persist. Thus, particular base positions are favored targets for certain carcinogens and some base-carcinogen adducts may be "hidden" from repair enzymes.

## CANCER INITIATION

DNA adduct formation is considered to be a necessary though not sufficient condition for the initiation of chemically induced cancer. It appears that it is not only the amount of DNA adduct formation and the molecular site of such lesions that is important, but the genomic location of the DNA adducts is critical. This introduces the topic of *oncogene activation* (to be considered in a subsequent article), a recent and rapidly growing field of research in carcinogenesis. It is of interest that a major review of chemical carcinogenesis by Farber in 1982 (*Am. J. of Path.*) does not mention oncogenes whereas an entire review is devoted to the topic of chemical oncogene activation by Balmain and Brown in 1988 (*Adv. Cancer Res.*).

Oncogenes (more than 30 have been identified in humans) are activated cellular genes that are normally present (as proto-oncogenes), coding for protein products that alter the cell's sensitivity to growth factors, hormones or other transmembrane signal pathways. Chemical activation of oncogenes results in altered cellular proteins, e.g. cell receptors, enzymes, DNA binding proteins, etc. which can modify the cell's growth control under suitable conditions (promotion). Malignant cell transformation is known to be a multi-stage process which involves initiation (genotoxicity), differential stimulation-proliferation (tumour promotion), and immune responses (tumour progression). It appears that at least two or more gene changes (e.g. point mutations) may be required for cancer induction.

## MOLECULAR EPIDEMIOLOGY

Weinstein (1988) has suggested that DNA can be used as an "internal dosimetry badge, for assessing types and levels of exposures to carcinogens and mutagens". Levels of DNA adducts above background exposures could then serve as indicators of cancer risk and carcinogen exposures. However, pilot studies have shown that the range of adducts seen in "background" controls overlaps with that observed in "exposed" groups. There is also need to evaluate the relationship of adduct levels in sampled tissues, for example, lymphocytes, to that of target tissues, such as the lung, liver, etc. Koh, Prout, and Groopman (*Adv. Intern. Med.*, 1989) have provided the most recent review of such methodological issues.

Due to DNA repair processes, adduct levels will probably indicate relatively recent exposures rather than distant ones. However, DNA chemotherapy adducts have been measured at autopsy and have been found to be dose related and to persist for up to 22 months from exposure (Santella, 1988). Protein adduction has been found to parallel DNA binding so hemoglobin adducts have been assayed in order to circumvent DNA repair constraints and permit detection of chronic low level exposure over the lifespan of the erythrocyte (compare the use of hemoglobin A1c in diabetes). Other protein assays may provide even longer term measures of exposure.

Since carcinogenesis is a multi-stage process and tumour promoters do not appear to be DNA reactive, DNA adduct monitoring will quantify the initiation stage rather than the promotion stage of cancer development. As noted earlier, different carcinogens attack specific DNA sites and can form multiple types of adducts. Only certain DNA sites may be critical for malignant transformation (mutational "hot spots"). Thus, measures of adduct levels will provide only one dimension in the complex relationship of DNA (and protein) adducts to mutation and cancer. Although there is often a good correlation between DNA adduct levels and the frequency of induced mutations, ratios of adduct to mutation vary considerably between chemical classes or agents.

Methods used for detecting DNA adducts in humans have included Synchronous Fluorescence Spectrophotometry (SFS), Radioactive Immunoassay (RIA), Ultrasensitive Enzymatic Radioimmunoassay (USERIA), Enzyme-Linked Immunosorbent Assay (ELISA), Single Cell Immunofluorescence Assay, and Gas Chromatography/Mass Spectrometry (GC/MS). Such assays now have the sensitivity to measure attomole concentrations of DNA adducts per microgram of DNA, i.e. about one adduct in  $10^8$  nucleotides. Thus we now have the *molecular* tools to detect the earliest indicators of chemical carcinogenesis at a cellular level. It remains

to define the *epidemiology* of such changes in order to estimate cancer risks. Perhaps we will see *molecular epidemiology* incorporated in future designated substance regulations.

Although much is yet to be learned about the relation of DNA adduct levels to cancer risk for specified exposure groups, molecular epidemiology promises to be a rapidly developing resource for occupational cancer control. As occupational health professionals, we should become acquainted with this "newcomer on our block"!

*\*Resident in Community Medicine, Department of Community Health Sciences, Faculty of Medicine, The University of Calgary.*

Table 1

Human Carcinogens (IARC Group 1)	Ames Test Results
4-aminobiphenyl	+
arsenic and compounds	-
asbestos	-
azathioprine	+
benzene	-
benzidine	+
bis(chloromethyl)ether	+
chlornaphazine	+
chlorambucil	+
chromium and compounds	+
conjugated estrogens	-
cyclophosphamide	+
diethylstilbestrol	-
melphalan	+
methoxsalen + UV-A	+
MOPP (chemotherapy)	+
mustard gas	+
myleran	+
2-naphthylamine	+
phenacetin analgesics	+
soots, tars, oils	+
treosulphan	+
vinyl chloride	+

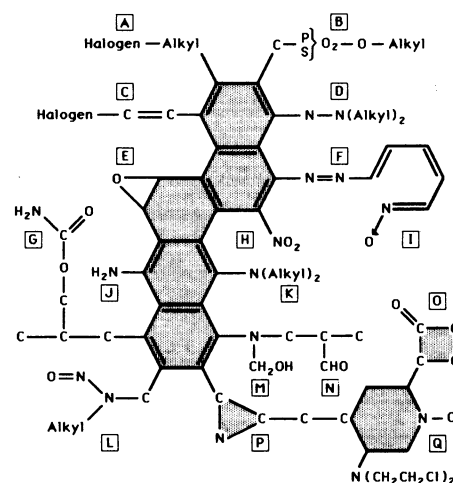
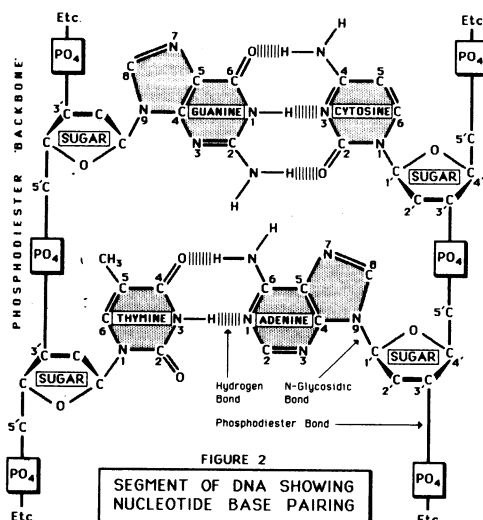


FIGURE 1

HYPOTHETICAL CARCINOGEN WITH DNA-REACTIVE UNITS

## Chemical Groups Identified in Figure 1

- |                                    |                                     |
|------------------------------------|-------------------------------------|
| A alkyl halides                    | J aromatic amines and derivatives   |
| B phosphonic/sulfonic alkyl esters | K aromatic alkylamino groups        |
| C monohaloalkenes                  | L alkyl N-nitrosamines              |
| D alkyl hydrazines                 | M N-methylol derivatives            |
| E epoxides                         | N alkyl aldehydes                   |
| F aromatic azo groups              | O propiolactones and propiosultones |
| G carbamate (urethane) derivatives | P aziridinyl derivatives            |
| H aromatic nitro groups            | Q N-chloramines                     |
| I aromatic ring N-oxides           | R nitrogen and sulfur mustards      |



## AUTOBODY SHOP SURVEY 1986-1987

Brian C. Alleyne, F.I.M.L.S., M.Sc.\*

Over the past two years attention has been focussed on possible health problems that may be associated with exposure to isocyanates or solvents in autobody shop operations.

In early 1987 the Occupational Health and Safety Division of Alberta Community and Occupational Health conducted a survey of 42 randomly selected autobody shops (21 in Edmonton and 21 in Calgary). Participating in the survey were 89 workers in Edmonton and 99 in Calgary. Each worker filled out a questionnaire on health problems involving the skin, breathing and nervous system.

This summary shows the findings of our survey. The complete report is available from Brian Alleyne, Manager, Occupational Epidemiology, 427-8943.

The key findings were:

- Respiratory symptoms in autobody shop workers appear to be higher than reported in other studies of general populations.
- The prevalence and pattern of wheezing suggests that it could be provoked by substances at work that can cause asthma.

- The prevalence of asthma in autobody shop workers in Alberta is estimated to range from 4.3 to 13%.
- Respiratory protection was used by 81% of the workers, but only 15% reported using the respiratory protection (positive pressure-supplied air masks) which is appropriate for working with isocyanates.
- The most common dermatological complaint was dry skin. Complaints of reddened inflamed skin and itchy skin were related to painting.
- Responses related to the nervous system were reported. These responses are similar to those given by other workers who are exposed to solvents. However our survey was not able to detect whether there were any adverse neurological effects resulting from exposure to solvents in autobody shop workers.

### RECOMMENDATIONS

- Autobody shop workers who have
  - wheezing (defined as a whistling sound during breathing) on most days and nights, or
  - difficulty breathing or tightness of the

chest on workdays but not on weekends, or

- had asthma diagnosed by a doctor should see their doctor for a more detailed examination. If it appears that the health problems may be related to work, improved ventilation or respiratory protection may help. If it does not help, a worker would be wise to consider changing occupations before the health problems get worse. Filing a claim with the Workers' Compensation Board should provide advice and assistance in changing employment for health-related reasons.

- Workers and employers should be aware that a positive pressure supplied air type of respirator is the only one approved for work with isocyanates.
- Skin contact with isocyanates or solvents should be avoided, and appropriate gloves or barrier creams should be used when skin contact is unavoidable.
- Evaporation or inhalation of solvents should be minimized by proper work practices, improved ventilation or use of appropriate respirators.

### AVAILABLE INFORMATION

Various bulletins and pamphlets about isocyanates and spray painting were mailed out to all autobody shops last year.

Also available is a guideline on "The Medical Aspects of Exposure to Isocyanates at the Workplace". This is designed to assist general physicians who do not have training in occupational medicine.

\*Occupational Health Epidemiologist, Medical Services Branch, Alberta Community and Occupational Health and Adjunct Lecturer, Department of Community Health Sciences, Faculty of Medicine, The University of Calgary.

\* \* \*

### UPCOMING CONFERENCES

**Achieving Equity in Health: Strategies and Resources.**  
Edmonton, Alberta  
May 8-11, 1989.

Contact:

Trevor D. Hodge, Chairperson  
Scientific Program Committee  
Alberta Public Health Association  
Sturgeon Health Unit  
Box 174  
St. Albert, Alberta  
T8N 1N3

# AN ANALYSIS OF THE FIBROSIS OF THE LUNG PROGRAM

Brian C. Alleyne, F.I.M.L.S., M.Sc.\*

Employers in Alberta with workers exposed to asbestos, silica or coal dust have been participating in the Fibrosis Program for a number of years. We have analyzed the information collected by the program during the years 1981-1984. There were 11,201 workers who participated in the program during that time.

The complete report is available from Brian Alleyne, Manager, Occupational Epidemiology, 427-8943. This summary presents the key findings of our analysis:

- Among the workers in the program 28.5% were exposed to asbestos, 19.4% to silica, 27.8% to coal dust and 24.3% to "other" dust.

- Among the 11,201 workers, 72 (0.6%) were identified as having pneumoconiosis (or fibrosis of the lung): 43 had asbestosis, 16 had silicosis, 11 had coal workers' pneumoconiosis, and 2 had unspecified pneumoconiosis. Expressed as a rate the prevalences were:

- asbestosis 13.6 per 1000 asbestos exposed workers
- silicosis 10.4 per 1000 silica exposed workers
- coal workers' 3.4 per 1000 coal dust pneumoconiosis exposed workers

- Among the 72 workers with pneumoconiosis, there were 17 who were discovered on their first submission to the program: 13 with asbestosis, 2 with silicosis and 2 with coal workers' pneumoconiosis. In some instances the work history would indicate that the worker had his exposure outside Alberta. Expressed as a rate these "new cases" were:

- asbestosis 6 per 1000 "new" asbestos exposed workers
- silicosis 1.2 per 1000 "new" silica exposed workers
- coal workers' 1.6 per 1000 "new" pneumoconiosis coal dust exposed workers

- Among the 72 workers with pneumoconiosis, there were 38 who were in the program prior to 1981 and who were identified as having developed pneumoconiosis during the period 1981 - 1984: 19 developed asbestosis, 10 silicosis, 8 coal workers' pneumoconiosis, and 1 unspecified pneumoconiosis. When we say "developed" we mean that the appearance of their X-rays reached a point where the fibrosis of the lung became apparent enough to meet the International Labour Organization standard that we were using. In fact, the diseases had been slowly developing for many years. Expressed as a rate these incidences were:

- asbestosis 4 per 1000 person years of asbestos exposure
- silicosis 7.2 per 1000 person years of silica exposure
- coal workers' 1.1 per 1000 person years of coal dust exposure

- Lung function test results suggest that there is poor quality control in the implementation of these tests. We found chest X-rays to be more reliable indicators of fibrotic changes.

- We found that radiologists in Alberta were quite accurate in reporting X-ray changes consistent with pneumoconiosis, but they were not always making an association with work exposure.

## UPDATE ON REGULATIONS

The final drafts of the *Asbestos, Silica and Coal Dust Regulations* were prepared following the input from the public review last year. We would like to thank all of those who sent in their comments and suggestions. The Regulation Review Committee decided that it would be appropriate to include these three regulations as Sections of an expanded *Chemical Hazards Regulation*. The expanded Chemical Hazards Regulation would then contain not only Occupational Exposure Limits (OEL's), but would also contain other requirements on specific chemical hazards.

Further work on the expanded Chemical Hazards Regulation has been delayed until work on the Alberta Workplace Hazardous Materials Information System (WHMIS) Regulation is completed. The Chemical Hazards Regulation and the WHMIS Regulation will need to be complementary.

## AVAILABLE INFORMATION

Also available from Alberta Community and Occupational Health are the guidelines:

- "Medical Monitoring of Workers Exposed to Asbestos";
- "Medical Monitoring of Workers Exposed to Silica";
- "Medical Monitoring of Workers Exposed to Coal Dust"; and
- "Medical Assessment of Fitness to Wear Respirators".

These guidelines were prepared to assist physicians and nurses who do not have specialized training in occupational health.

*\*Occupational Health Epidemiologist, Medical Services Branch, Alberta Community and Occupational Health and Adjunct Lecturer, Department of Community Health Sciences, Faculty of Medicine, The University of Calgary.*

## UPCOMING CONFERENCES

**Education & Training for Prevention. An International Section for Information on Prevention of Occupational Risks.**

Paris, France  
May 31-June 2, 1989.

Contact:

Caisse regionale d'assurance maladie d'Ile-de-France  
17-19 place de l'Argonne  
75019 Paris  
France

\* \* \*

## Work with Display Units.

Second International Scientific Conference

Montreal, Quebec  
September 11-14, 1989.

Contact:

WWDU 1989  
General Secretary, IRSST  
Diane Berthelette  
505, boul de Maisonneuve Ouest  
Montreal, Quebec  
H3A 3C2

\* \* \*

## International Symposium on New Trends and Developments in Occupational Health Services.

Espoo, Finland  
October 3-6, 1989.

Contact:

Dr. Esa Rahkonen, Symposium Secretariat  
Topeliuksenkatu 42 aA  
SF-00250 Helsinki, Finland

\* \* \*

## Epidemiology in Occupational Health.

Tokyo, Japan  
October 11-13, 1989.

Contact:

Secretariat  
VII International Symposium on Epidemiology in Occupational Health  
Department of Preventive Medicine & Public Health  
School of Medicine, Keio University  
35 Shinano-machi, Shinjuku Ward  
Tokyo 160, Japan

# A CRITICAL REVIEW OF "OCCUPATIONAL MEDICINE - A SYLLABUS FOR ALBERTA"

Wilfred Ntiamoah\*

"Occupational Medicine - A Syllabus for Alberta" was published in August, 1985. It was prepared mainly for the Medical Students in Phase II at The University of Alberta by Tee L. Guidotti, Professor of Occupational Medicine at the University of Alberta. The monograph has deliberately and successfully incorporated subject materials relevant to the needs of both users and providers of health care in Alberta. The monograph assists the reader in the following ways:

- i) To assist in the understanding of the organization of occupational health services;
- ii) To help identify, assess and control occupational health by observing individuals and groups in relation to their work environment;
- iii) To understand the special problems that arise out of relations between management and workers, emphasizing throughout the impartiality of the occupational physician and the confidentiality of medical information about individual patients;
- iv) To know the methods of seeking information and the importance of currently being aware of new facts.

According to Dr. Guidotti, occupational medicine in practice is part of a much larger network of relationships that is formalized into a complex system. A physician, he states, cannot be effective in managing an occupationally related case alone. Therefore, the solution of occupational health problems at a reasonable cost requires knowledge and technical resources beyond the capacity of the physician. The many professional groups who contribute their knowledge and services to occupational medicine include the following: occupational health nurses, occupational health nurse practitioners, occupational hygienists, safety engineers, rehabilitation counsellors, audiologists and occupational and hearing conservationists.

The difficulties inherent in controlling occupational diseases successfully are characterized as follows:

- i) *Latent period*: Often there is a long delay between the exposure of the occupational agent and the appearance of the first sign or symptom. For most occupational cancers, the latent period ranges from ten to fifty years. In some cases workers may not remember *what* (agent) they were exposed to several years ago, or *when* (at which work place) the exposure occurred.
- ii) *Non Specific Symptomatology*: Occupational diseases or conditions may

not present with specific symptoms or signs. Thus diagnosis may be challenging.

- iii) *Presence of the Agent and Form of the Agent*: In some instances, it is difficult to determine not only what kind of occupational agent a worker has been exposed to, but in what form - gaseous or liquid state.

This monograph illustrates how a worker's occupational history can be used to establish a direct occupational association - a link between work-related risk caused by an agent during the worker's previous or present work, and an occupational disease (health outcome). As the monograph states, "the complexity of interpreting the occupational history discourages many clinicians who might otherwise incorporate it into their practice." But if properly utilized, according to this monograph, the occupational history need not be burdensome. The uses of the occupational history are numerous. They include the following: i) Patient evaluation, ii) Surveillance, iii) Compensation and eligibility, iv) Liability and risk control, v) Community education. The problem with surveillance of occupational diseases is that the agents - chemical, physical, biological - are numerous and yet new products (agents) continue to be produced all year round. The potential for discovering links between causes (agents) and effects (health outcomes), therefore widens. Certain diseases are by their nature strongly associated with occupational exposure. Examples are hemangiosarcoma of liver and mesothelioma of the peritoneum or pleura.

The monograph also elaborates on the relationships of the various interest groups serving the needs of occupational medical care. Whether a service required is geared toward a control or prevention program, team effort is contributed by various members of the interest group. The group membership includes the following: the regulatory agency, the employer, the Worker's Compensation Board, the physician, and the patient (or employee). In every occupational health service, whether ameliorative or preventive, "the physician's responsibility is first to the patient whether in treating occupational disorders or intervening and educating to avoid the development of preventable disease."

Dr. Guidotti's monograph *Occupational Medicine - A Syllabus for Alberta*, offers its readers comprehensive coverage of occupational medical care services with detailed references of various technical staff and informative materials. This monograph reasserts the set of values and philosophy of the activities of occupational

health services, and addresses not only the prevention of occupational hazards but also the promotion of the general health of workers. Prevention programs involving various professional bodies are emphasized by Dr. Guidotti as an ongoing process in the workplace. This unique feature is part of the appeal of this monograph. *Occupational Medicine - A Syllabus for Alberta* is a great source of information to its reader.

(Reference available on request)

## AVAILABLE INFORMATION

Readers interested in ordering a copy should send \$8.55 (\$7.05 plus \$1.50 mailing cost) to:

University of Alberta Health Sciences  
Bookstore  
Walter MacKenzie Centre 1J1  
8440 - 112 Street  
Edmonton, Alberta T6G 2B7

Request by title "Occupational Medicine: A Syllabus for Alberta". (There is another syllabus with a similar name, so "occupational medicine" should be specified prominently.) Payment can be C.O.D. or by Mastercard or Visa. Telephone orders can be placed by calling (403) 492-4696.

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