Study of the Occupational Disease Law in Maine

Submitted to the Joint Standing Committee on Labor

January 1999

Maine Workers' Compensation Board

Department of Human Services, Bureau of Health

Department of Labor, Bureau of Labor Standards

Department of Professional and Financial Regulation, Bureau of Insurance

Maine Institute for Occupational Health Education

DEPARTMENT OF LABOR
BUREAU OF LABOR STANDARDS
45 STATE HOUSE STATION
AUGUSTA, MAINE
04333-0045

VALERIE R. LANDRY
COMMISSIONER

ANGUS S. KING, JR.
GOVERNOR

ALAN C. HINSEY
DIRECTOR

January 25, 1999

Sen. Neria R. Douglass, Chair
Rep. Pamela H. Hatch, Chair
Joint Standing Committee on Labor
119th Maine Legislature
Augusta, ME 04333

Dear Senator Douglass, Representative Hatch, Members of the Joint Standing Committee on Labor:

Attached please find a copy of the "Study of the Occupational Disease Law in Maine." Last session the Legislature commissioned that a study of the Occupational Disease Law be completed and submitted to the Labor Committee in January of 1999 (L.D. 835). I have had the honor of chairing the "study group" that has spent many hours over the past year compiling and analyzing the material that you will find in the attached "study." I would like to take this opportunity to thank the members of the "study group" who constructed this document. Their knowledge, dedication and plain hard work has resulted in a document that we believe will serve the Labor Committee well whenever it has cause to consider the provisions of the Occupational Disease Law in Maine.

I, and my fellow members of the "study group," would be happy to address the Labor Committee in person to present the recommendations of the "study group" and to answer any questions that the Committee might have. Please notify me if you would like the "study group" to address the Committee and I will arrange to have all the members present.

If you have any questions about this report or the process that the "study group" employed to compile the information, please feel free to call me (287-7067).

Sincerely,

Isabella Tighe

Isabella Tighe
Maine Workers' Compensation Board

AC/ln
attachment

Study of the Occupational Disease Law in Maine

Table of Contents:

Attachments:

A. L.D. 835 - "Resolve, Instructing the Workers Compensation Board to Study and Make Recommendations Regarding the Occupational Disease Law" - 1998

B. "Staff Study of Occupational Disease Laws" - Jan. 1998, Heather Henderson, Office of Policy and Legal Analysis, Maine State Legislature

C. Report to the Labor Committee Regarding Occupational Disease Laws in Maine - the Commission on Health and Safety in the Maine Workplace, Beth Stowell Chair, Jan., 1998.

D. Report to the Labor Committee Regarding Occupational Disease Laws in Maine - Isabella Tighe, Alan Hinsey, Allison Hawks, MD., Eric Cioppa, and Karen Packard - Feb.., 1998

E. 1996 Survey of Occupational Health Practices, March 24, 1996

F. Maine Law Review - "What is wrong with Maine's Occupational Disease Law?", Thomas R. Watson, Volume 34, Number 1, 1982

G. "Clinical Aspects of Occupational Medicine," by Harold R. Imbus, M.D., Sc.D., from Occupational Medicine, 3rd Edition, 1994 (article supplied by Craig Curtis, M.D., Health Works, Occupational and Sports Medicine)

H. "Chemically Injured Workers Ignored, Disenfranchised and Thrown Away ... the Overall Problem" - excerpt from the Maine Injured Workers Association Website

I. Suggested Language Changes to the Workers Comp Act - Occupational Disease Law; M.R.S.A. 39A Section 606 - Date from which compensation is computed, employer liable & M.R.S.A. 39A Section 607 - Notice of incapacity; filing of claim

J. Table of Selected Job Categories, Exposures, and Associated Work-related Diseases and Conditions, taken from, "Environmental Medicine - Integrating a Missing Element into Medical Evaluation," National Academy Press, 1995.

K. New OD Reporting Forms:

- WCB M-1
- Bureau of Health - Occ Disease Reporting Program Form

L. Case Studies in Environmental Medicine

- Cadmium Toxicity, Benzene Toxicity, Chromium Toxicity, Neuropsychological cases

Study of the Occupational Disease Law in Maine

Submitted to the Joint Standing Committee on Labor

January 1999

1. Introduction:

In the spring of 1998 the Legislature enacted L.D. 835, a resolve, "Instructing the Workers' Compensation Board to Study and Make Recommendations Regarding the Occupational Disease Law." The resolve specified that representatives from the Workers' Compensation Board, the Bureau of Health, the Bureau of Insurance and the Bureau of Labor Standards should conduct the study and report their findings and recommendations to the Labor Committee by January 15, 1999. The resolve further specified that the following particular topics be included in the study and report:

- issues related to proof of causation
- unique problems that arise when there is a prolonged period between exposures and onset of the disease
- apportionment of liability
- provision of benefits where there has been no lost time
- discussion of Occupational Disease issues
- alternative approaches that can be taken to address problems
- a recommended definition for Occupational Disease
- a recommendation for tracking Occupational Disease data in Maine
- plus any other relevant recommendations

The report and recommendations that follow attempts to address all of the issues identified in L.D. 835.

Over the past year a "study group" has been assembled and has met periodically to collect the attached material and to construct the following report. The membership of this group has varied over the course of the year. The following is a listing of all persons who were a part of the group and who contributed to this study and recommendations:

Isabella Tighe, Workers' Compensation Board

Julia Finn, Workers' Compensation Board

Frank Kimball, Bureau of Insurance

Eric Cioppa, Bureau of Insurance

Allison Hawkes, M.D., Bureau of Health

Wendy Davis, Bureau of Health

Joyce Roy, Bureau of Health

Phillip Haines, Bureau of Health

Karen Packard, Maine Institute for Occupational Health Education

Alan Hinsey, Bureau of Labor Standards

Since much of the research and analysis on the subject of Occupational Disease Law has already been compiled, the study group decided to focus its attention on the collection and synthesis of those various studies and analyses. Therefore, the following report is designed to keep the recommendations made by the group as clear and concise as possible. All of the relevant information and research that was reviewed in the course of performing this study are attached to the recommendations. The "Recommendation" section of the report on the following page is straightforward and simple. The study group will be happy to elaborate on these recommendations when they present this report in person to the Labor Committee.

In addition to the recommendations, the study group has also include a section in the report that presents several Occupational Disease case studies and scenarios for policy makers to consider as well as a section that summarizes the key findings of the "1996 Survey of Occupational Health Practices."

The Occupational Disease Law in Maine is complex. Any attempt to change the existing law must be done with great caution by lawmakers. The study group offers the following "Cautions" to the policy maker as he or she begins to consider changes to the Occupational Disease Law in Maine.

CAUTIONS:

- **Be on the look out for any unintended consequences** - seemingly minor changes in the law may have significant effects on injured workers, employers, insurance providers and regulators.

- During work session last year, the Labor Committee specifically prohibited the study group from considering the issue of "costs to the system" when reviewing the issues laid out in L.D. 835. **The study group did not "cost out" any of the recommendations but STRONGLY URGES that lawmakers fully analyze any potential changes to the Occupational Disease Law so that the full impact of potential increases in cost may be understood.**

- In it's original report to the legislature in Feb. 1998 (see Attachment D.), the study group recommended that standard diagnosis codes be used on all Occupational Disease report forms. It was also recommended that Occupational Disease data should be collected for a full two years based on these standard diagnosis codes before any changes be proposed to the Occupational Disease Law. **The study group still believes that the standardized diagnosis data should be collected for a full two years and thoroughly analyzed BEFORE any substantive changes are made to the Occupational Disease Law.**

2. Recommendations of the "Study Group":

After a comprehensive review and much discussion of all the material collected and displayed in the attachments to this document, the study group recommends that the following nine (9) points be considered by policy makers whenever they have cause to review the provisions of the Occupational Disease Law in Maine.

1.We recommend that the definition of Occupational Disease in the law be extended by one additional sentence, as follows:

> The definition of occupational disease will read "means only a disease that is due to causes and conditions characteristic of a particular trade, occupation, process or employment and that arises out of and in the course of employment. "Occupational disease" includes, but is not limited to, the diseases described in §§ 612-615 of this chapter."

It is the belief of the study group that the Occupational Disease Law was not intended to be confined to just those diseases described in §§ 612-615 (hearing loss, silicosis, asbestos related diseases, and disability due to radioactive properties). We further recommend that the Bureau of Health adopt this definition.

2. The Occupational Disease Reporting Law (22 MRSA Chapter 259-A Subsection 1491 - 1495) that currently governs the Bureau of Health Occupational Health Program must be monitored for compliance.

3. The medical licensing boards, professional organizations, and other appropriate governmental agencies should be used to educate all physicians on the importance of reporting occupational diseases.

4. All health care providers will report occupational diseases to the Bureau of Health on the form designated by the Bureau of Health. The Bureau of Health will forward copies of these forms to the Workers' Compensation Board.

5. Rewrite and/or clarify the Occupational Disease Law. See redrafts of §§ 606, 607 in Attachment I.

6. Revisit the three-year limitation contained in § 609. Maine law requires that employees must become incapacitated within 3 years of the last injurious exposure to qualify as a valid, compensable Occupational Disease claim in Maine. While the 3 year "latency period" in Maine is liberal, relative to many other states (34 states have latency periods of less than two years), the study group simply recommends that lawmakers revisit the 3 year latency standard whenever they have cause to reconsider the provisions of the Occupational Disease law. After reconsideration, the lawmakers may in fact wish to retain the 3 year latency standard.

7. Revisit the medical/scientific literature on occupational diseases. Explore the changes in current thinking that have occurred since the law was originally written regarding latency periods, etc.

8. With respect to apportionment, the statute currently provides that the carrier on the risk during the last injurious exposure (provided that exposure lasted at least 60 days) is responsible for the claim. No changes are recommended in this regard.

9. With respect to payments for medical bills, no compensation, either for medicals or indemnity, is due until the employee becomes incapacitated. See 39-A MRSA §§ 206, 209. Policy makers should be aware that this provision differs from payments for personal injuries under the Workers' Compensation Act in that medical only claims are payable without incapacity. It is important to note that the vast majority of states treat this issue exactly as Maine does, i.e., no benefits are paid if the employee is not incapacitated by the occupational disease (only 4 states pay any non-lost work time benefits). The study group is not recommending that "Medical only claims" be paid under the Occupational Disease law, but simply that as lawmakers have cause to reconsider the provisions of the law that they revisit this issue to ensure that it still represents the thinking of the legislature.

3. Occupational Disease Case Histories and Case Scenarios:

The study group has provided numerous Occupational Diseases Cases Histories and Case Scenarios for you to review in Attachment L at the end of this document. We urge you to take some time to look over these case histories and examples of how occupational disease can manifest itself in employees, even after substantial latency periods have occurred. While there is much technical information in this material, the Executive Summary at the beginning of the Attachment L section will provide you with useful insights into how various occupational diseases can effect employees.

Attachments

A. L.D. 835 - "Resolve, Instructing the Workers Compensation Board to Study and Make Recommendations Regarding the Occupational Disease Law" - 1998

B. "Staff Study of Occupational Disease Laws" - Jan. 1998, Heather Henderson, Office of Policy and Legal Analysis, Maine State Legislature

C. Report to the Labor Committee Regarding Occupational Disease Laws in Maine - the Commission on Health and Safety in the Maine Workplace, Beth Stowell Chair, Jan., 1998.

D. Report to the Labor Committee Regarding Occupational Disease Laws in Maine - Isabella Tighe, Alan Hinsey, Allison Hawks, MD., Eric Cioppa, and Karen Packard - Feb.., 1998

E. 1996 Survey of Occupational Health Practices, March 24, 1996

F. Maine Law Review - "What is wrong with Maine's Occupational Disease Law?", Thomas R. Watson, Volume 34, Number 1, 1982

G. "Clinical Aspects of Occupational Medicine," by Harold R. Imbus, M.D., Sc.D., from Occupational Medicine, 3rd Edition, 1994 (article supplied by Craig Curtis, M.D., Health Works, Occupational and Sports Medicine)

H. "Chemically Injured Workers Ignored, Disenfranchised and Thrown Away ... the Overall Problem" - excerpt from the Maine Injured Workers Association Website

I. Suggested Language Changes to the Workers Comp Act - Occupational Disease Law;
M.R.S.A. 39A Section 606 - Date from which compensation is computed, employer liable &
M.R.S.A. 39A Section 607 - Notice of incapacity; filing of claim

J. Table of Selected Job Categories, Exposures, and Associated Work-related Diseases and Conditions, taken from, "Environmental Medicine - Integrating a Missing Element into Medical Evaluation," National Academy Press, 1995.

K. New OD Reporting Forms:
- WCB M-1
- Bureau of Health - Occ Disease Reporting Program Form

L. Case Studies in Environmental Medicine
- Cadmium Toxicity, Benzene Toxicity, Chromium Toxicity, Neuropsychological cases

ATTACHMENT A

STATE OF MAINE

IN THE YEAR OF OUR LORD
NINETEEN HUNDRED AND NINETY-EIGHT

H.P. 610 - L.D. 835

Resolve, Instructing the Workers' Compensation Board to Study and Make Recommendations Regarding the Occupational Disease Law

Sec. 1. Study of Occupational Disease Law. Resolved: That the Workers' Compensation Board, the Bureau of Health, the Bureau of Insurance and the Bureau of Labor Standards shall study the unique issues involved in providing workers' compensation benefits to employees under the Occupational Disease Law and make recommendations as necessary to ensure that the purposes of the Workers' Compensation Act of 1992 are achieved with respect to occupational diseases. The particular topics to study must include issues related to proof of causation when the occupational disease is thought to arise from exposure to hazardous materials; unique problems arising when there is a prolonged period between exposure to the hazardous material and the onset of disease; apportionment of liability for occupational diseases; and the provision of benefits in cases where the employee has not lost any time from work. The groups may rely on their staffs to conduct this study and may also request the assistance without pay of any other person or group in providing information for this study; and be it further

Sec. 2. Report and recommendations. Resolved: ~~That~~ the groups named in section 1 shall report by January 15, 1999 to the joint standing committee of the Legislature ~~having jurisdiction~~ over labor matters on the study conducted pursuant to section 1. The report must contain a discussion of the issues, alternative approaches that may be taken to address any problems identified, a recommended definition for occupational disease, a recommendation for tracking data on occupational diseases and any other recommendations considered necessary. The groups may submit with the report legislation necessary to implement their recommendations.

Staff Study of Occupational Disease Laws In the United States

Latency Periods

The latency period is the time within which an employee must file a claim for compensation for an occupational disease.

Most states require claims to be filed within 1 year (12 states) or 2 years (22 states) of a specific starting point. Maine requires claims to be filed within 3 years as do 7 other states.

Maine State Legislature

OFFICE OF POLICY AND LEGAL ANAL

13 State House Station, Augusta, Maine 04333-0013
Telephone: (207) 287-1670
Fax: (207) 287-1275

ATTACHMENT B

January 22, 1998

To: Joint Standing Committee on Labor

From: Heather Henderson, Legislative Analyst HH

Re: Staff Study of Occupational Disease Laws (LD 835)

Last May, the Committee voted to carry over LD 835, Resolve, Instructing the Workers' Compensation Board to Study and Make Recommendations Regarding the Occupational Disease Law. The Committee felt it needed more information before it could make a decision on the bill, and it requested three pieces of information from three different groups:

1. The Workers' Compensation Board was asked to provide historical information on occupational disease claims in Maine;

2. The Commission on Health and Safety in the Workplace was asked to assess the availability of Maine data on chemical exposures and evaluate whether advances in the scientific field of occupational disease have any bearing on the operation of our occupational disease law; and

3. The Office of Policy and Legal Analysis was asked to compile laws from the other states regarding specific occupational disease issues.

Attached to this memo is the OPLA component. The study ("Comparison of Occupational Disease Laws") lists the pertinent laws from each of the other states with regard to proof of causation, latency periods, apportionment of liability and payment of benefits if no work time is lost. The highlights in each of those categories are discussed below.

Proof of Causation

The first column states the various definitions of occupational disease and any burdens of proof or presumptions that exist in each state. Forty states, including Maine, require that the disease arise out of and in the course of employment and be peculiar to (or result from the nature of) the employment. An additional eight states require an occupational disease to arise out of and in the course of the employment, without the "peculiar to"

requirement. Two states (New York and North Carolina) only recognize diseases specifically listed in statute as being occupational diseases. Ohio, Pennsylvania and Rhode Island enumerate occupational diseases in statute but also recognize other diseases that meet the specific criteria.

The employee or claimant bears the burden of proof in every state. A number of states have presumptions regarding occupational diseases, both for and against employees. For example, Louisiana states that a disease contracted by an employee who has been working for a particular employer for less than one year is presumed to be non-occupational, and Virginia declares flatly that conditions of the neck, back or spinal column, hearing loss and carpal tunnel syndrome are not occupational diseases. However, eight states (Michigan, Minnesota, Nevada, New Hampshire, Oregon, South Carolina, Virginia and Washington) generally presume that lung and respiratory diseases are occupational diseases for firefighters and/or police officers.

Latency Periods

The second column addresses the period of time within which an employee must file a claim for compensation for an occupational disease. Most states require general claims to be filed within 1 year (12 states) or 2 years (22 states) of a specific starting point, but the range is 90 days (Nevada) to 7 years (Georgia, Virginia).

Four states have extended filing periods when a disease results from certain events. For example, Illinois permits claims up to 25 years after the last day of the last exposure to radiological materials or equipment, Indiana permits claims up to 35 years after the last exposure to asbestos, Kentucky permits claims up to 20 years after the last exposure to radiation or asbestos, and New Mexico permits claims up to 10 years after the last day of work on which the employee was exposed to radioactive or fissionable materials. In addition, several other states (Alaska, Kansas, South Carolina and Wyoming) simply state that their time limitations do not apply for certain diseases.

The states measure their filing periods from different starting points. Sixteen states measure from the date of actual disability; 17 states count from the date the claimant knew or should have known of the disability and its relationship to the employment; and 14 states measure from the date of the last injurious exposure. Standing alone are New Mexico (measuring from the last day the employee worked for the employer against whom compensation is claimed--particularly narrow, considering its 1-year filing period) and Washington (measuring from the date the employee receives written notice of the disease from a physician).

Some states will measure from more than one date. For example, Maine takes a two-step approach. It states that an employee must become incapacitated within three years of the last injurious exposure, but that claims may be filed up to 2 years after incapacity. Louisiana measures from either the date of disability, the date the diseases manifests

itself, or the date the employee knew or should have known of the disease and its relationship to the employment--perhaps to compensate for its very short filing period of six months.

Apportionment of Liability

The third column shows how each state apportions liability for occupational disease claims among employers. Twenty-five states place liability on the employer in whose employment the worker was last injuriously exposed to the hazards of the disease. Of those, Idaho and Maine state that the exposure must have lasted at least 60 days. In addition, seven states (Colorado, Illinois, Indiana, Kansas, New Mexico, Oklahoma and South Dakota) require that the exposure last at least 60 days for specific diseases, such as silicosis or asbestosis.

Five states (Connecticut, Michigan, Nevada, New York and Rhode Island) impose liability on the employer who last employed the worker. California assigns liability to anyone who employed the worker during the year immediately prior to the date of injury or the last date on which the worker was employed in the occupation creating the risk of the disease. Sixteen remaining states do not specifically state which employer bears the liability; the presumption was made that liability falls on the employer out of whose employment the occupational disease arose.

Benefits If No Lost Work

The last column addresses the issue of whether compensation benefits are paid when the employee does not lose work. The vast majority of states treat this issue the way Maine does: if the disease does not incapacitate the employee from working, no benefits are awarded. In fact, many states require that the employee be actually disabled from work for a specified period of time before receiving benefits (typically three to seven days).

States that take a different approach are:

- Arkansas — If an employee is not actually disabled but is affected by silicosis or asbestosis to the point that it is hazardous for the employee to continue in the work, the Workers' Comp Commission can remove the employee from the work and pay benefits for 26 weeks or until the employee has found other work, whichever is earlier.

- Iowa — Employees who are able to continue in employment receive (only) medical service benefits.

- Nevada: — Medical benefits must be paid from the date of application for payment (the customary 5-day disability requirement does not apply).

- New Jersey: A condition that does not impair an employee's ability to work will be compensable only if it is serious enough to interfere substantially with other aspects of the employee's life.

- North Carolina: An employee may receive medical benefits when there is no actual disability, but only if the employee suffers damage to organs as a result of the occupational disease.

G:\OPLAGEA\COMMTTEE\LAB\CORRESP\ODMEMO.DOC

COMPARISON OF
OCCUPATIONAL DISEASE LAWS

State	Proof of Causation	Latency Periods	Apportionment of Liability	Benefits If No Lost Work
Alabama	An OD arises out of and in the course of employment, without regard to fault, is due to hazards in excess of those in general, and is peculiar to the particular occupation. No presumption that disablement or death is result of OD. A person claiming benefits for OD has the burden of establishing entitlement.	Claims forever barred unless parties agree to compensation or complaint is filed within 2 years of the date of injury or, in the case of death, within 2 years of the date of death, provided the death results proximately from the OD and occurs within 3 years of the date of injury. Date of injury is the date of the last exposure to the hazards of the disease.	Liability only for employer in whose employment occurred the last exposure. For pneumoconiosis or radiation, liability for employer in whose employment occurred the last exposure to the hazards of the disease in each of at least 12 months, within a period of 5 years prior to the date of the injury, and for employer who furnished workers' compensation during that period,.	An injury by accident occurs if an employee dies or is disabled as the result of an OD. No compensation unless employee experiences "loss of ability to earn". Bentley Pontiac/Cadillac Inc. v. Adams, 646 So.2d 155 (1994).
Alaska	An OD arises naturally out of and in the course of employment	Claims barred unless filed within 2 years after employee knows the nature of the disability and its relation to the employment. Death benefits barred unless filed within 1 year after death or, if payments have been made without an award, within 2 years after the date of the last payment. In the case of latent defects, the board determines the employee's right to claim, time limitations notwithstanding.	Liability for employer out of whose employment the OD arose.	Disability means incapacity to earn the wages the employee was receiving at the time of injury in the same or other employment. Disability benefits are premised on the loss of earning capacity, not merely medical impairment. Cortay v. Silver Bay Logging, 787 P.2d 103 (1990). The law does not contemplate the payment of compensation in addition to the payment of wages. Hanson v. Benson, 179 F. Supp. 130 (1959).
Arizona	An OD is deemed to arise out of employment only if there is a direct causal connection between the work and the OD; the OD can be seen to have followed as a natural incident of the work; the OD can be fairly traced to the employment; the OD does not come from a hazard to which workers would have been equally exposed outside the employment; the OD is incident to the character of the business; and the OD appears to have had its origin in a risk connected with the employment, although it need not have been foreseen.	Claims must be filed within 1 year after claimant knows or should know that the employee sustained a compensable OD.	Liability only for employer in whose employment occurred the last injurious exposure. For silicosis or asbestosis, liability only for employer in whose employment occurred the last exposure to harmful quantities of silicon dioxide dust during a period of 2 years or more.	Lost earning capacity is the basis for benefits. Compensation is awarded in lieu of lost wages, not as damages for pain and suffering. Bugh v. Bugh, 608 P.2d 329 (19890).
Arkansas	An OD arises out of and in the course of employment or naturally follows or unavoidably results from an injury. An OD must be due to the nature of an employment in which the hazards of the disease actually exist and are	Disablement or death must result within 1 year or, in the case of silicosis or asbestosis, within 3 years of the last injurious exposure to the disease. Death compensable if it follows continuous disability for which compensation has	Liability for employer in whose employment occurred the last injurious exposure and for the insurance carrier on the risk at that time.	Compensation payable for disablement or death. If an employee is not actually disabled but is found to be affected by silicosis or asbestosis to the degree that it is hazardous to continue in the employment involving exposure to the

State	Proof of Causation	Latency Periods	Apportionment of Liability	Benefits If No Lost Work
Arkansas cont'd	characteristic of that employment. A claimant must show a causal connection between the employment and the OD by clear and convincing evidence. In the absence of conclusive evidence in favor of the claim, silicosis and asbestosis are presumed not to be OD's unless the employee was exposed to silica dust or asbestos dust for at least 5 of the 10 years immediately preceding the date of disablement, 2 years of which must have been in the state.	been made and results within 7 years of the last exposure. Limitations do not apply to OD caused by exposure to x-rays, radioactive substances or ionizing radiation.		disease, the commission may remove the employee from the employment and pay benefits until the earlier of 26 weeks or the date the employee finds other steady employment.
California	An OD must arise out of employment. Claimants must prove their cases by a preponderance of the evidence.	Claims must be brought within 1 year of the date of death, 1 year of the date of last furnishing benefits if death occurs more than one year from the date of injury, or 240 weeks from the date of injury. The date of injury is the date the employee first suffered disability and knew or should have known the disability was caused by the employment.	Liability for employers who employed the employee during the year immediately prior to either the date of injury or the last date on which the employee was employed in an occupation exposing him/her to the hazards of the disease, whichever occurs first. If no employer was covered by workers' compensation during that time, liability is imposed on the last employer that exposed the employee to the OD and was covered by worker's compensation. If more than one employer can be held liable, the liability is joint and several.	Benefits are a substitute for lost wages. Livitsanos v. Superior Court, 828 P.2d 1196 (1992).
Colorado	An OD results directly from employment, can be seen to have followed as a natural incident of the work, can be fairly traced to the employment as a proximate cause and does not come from hazards to which the employee would have been equally exposed outside employment. Mental or emotional stress is not an OD unless it is shown by competent evidence that the stress is proximately caused solely by hazards to which the employee would not have been equally exposed outside the employment. A heart attack is not an OD unless it is shown that the attack was proximately caused by an unusual exertion arising out of and within the course of employment. For permanent total disability, the employee has the burden of proving inability to earn any wages in the same or other employment.	Disability beginning more than 5 years after the date of injury (the date of the last injurious exposure) is conclusively presumed not to be due to the injury, except in the case of disability or death resulting from exposure to radioactive materials, to fissionable materials or to uranium, or resulting from asbestosis, silicosis or anthracosis. Death occurring more than 2 years after the date of injury is rebuttably presumed not to be due to the injury, except in the case of silicosis, asbestosis, anthracosis or disability or death resulting from exposure to radioactive materials, fissionable materials or uranium.	Liability only for employer in whose employment the employee was last injuriously exposed and suffered a substantial permanent aggravation thereof. Liability also for insurance carrier on the risk at that time. In the case of silicosis, asbestosis or anthracosis, liability for employer and insurance carrier when employee was last exposed to harmful quantities of silicon dioxide dust, asbestos dust or coal dust on each of at least 60 days or more.	Benefits protect against an actual loss of earnings. Ice v. Industrial Comm'n, 207 P.2d 963 (1949).

State	Proof of Causation	Latency Periods	Apportionment of Liability	Benefits If No Lost Work
Connecticut	An OD must arise out of and in the course of business and must originate while the employee was engaged in the line of duty. It must be peculiar to the occupation and due to causes in excess of the ordinary hazards of employment.	Claim must be filed within 1 year from the date of the injury (the date of incapacity to work), within 3 years of the first manifestation of a symptom of the OD, or within 1 year of death, if the death occurred within 2 years of the manifestation of the OD.	Initial liability for the employer who last employed the employee prior to the filing of the claim, and the employer's insurer. The commissioner must then determine whether prior employers are also liable; if so, they must reimburse the initially liable employer for their portion of the liability.	Disability means total of partial incapacity to work.
Delaware	An OD arises out of and in the course of employment.	Claims forever barred unless, within 2 years of disability or death, the parties agree upon compensation or a claim is filed. Claims for OD due to ionizing radiation forever barred unless employee files petition within 1 year of the date the employee first knew the disability was or could have been caused by employment or, in the case of death, within 1 year of the date the petitioner knew or should have known the possible relationship of the death to the employment.	Liability for employer out of whose employment the OD arose.	A claimant must prove an actual reduction in earnings to qualify for benefits. Ernest DiSabatino & Sons v. Apostolico, 260 A.2d 710 (1969).
Florida	An OD results from the nature of the employment, meaning that the occupation has a particular hazard of the OD or the incidence of the OD is substantially higher in that occupation. Presumptions in favor of Worker's Compensation claimants do not apply to claims for OD's.	Death must follow continuous disability from an OD for which a timely claim has been made and must result within 350 weeks after the last exposure.	Liability only for employer in whose employment occurred the last injurious exposure and the insurance carrier on the risk at that time.	Disablement means becoming actually incapacitated, partially or totally, from performing the work in the last occupation in which the injurious exposure occurred.
Georgia	An OD arises out of and in the course of the employment. The claimant must prove that there was a direct causal connection between the employment and the OD, that the OD was a natural incident of exposure during employment, that the OD is not one to which the employee might have had substantial exposure outside employment, and that the OD appears to have had its origins in and was a natural consequence of a risk connected with the employment.	Claims must be filed within 1 year of the date the employee knew or should have known of the disablement and its relationship to the employment. No claim may be filed more than 7 years after the last injurious exposure to the hazard of the disease. Employees with asbestosis or mesothelioma related to exposure to asbestos have 1 year from the date of first disablement after diagnosis to file a claim. In the case of death, claims must be filed within 1 year of the date of death, assuming the claim was not barred during the employee's life.	Liability only for employer in whose employment occurred the last injurious exposure and the insurance carrier by whom the employer was insured at that time.	Disability means becoming actually disabled to work. Where there is no loss of wages, there is no disablement. Yates v. United States Rubber Co., 112 S.E.2d 182 (1959).
Hawaii	An OD is proximately caused by or results from the nature of the employment.	Claims barred unless filed within 2 years after the effects of the OD become manifest and within 5 years after the date of the occurrence that caused the OD. For OD's caused by exposure to x-rays,	Liability for employer out of whose employment the OD arose.	The right to compensation presupposes a disability to work, either total or partial. 34 H. 317.

State	Proof of Causation	Latency Periods	Apportionment of Liability	Benefits If No Lost Work
Hawaii cont'd		radium, ionizing radiation, radioactive substances, arsenic, asbestos, benzol, beryllium, zirconium, cadmium, chrome, lead, fluorine or other substance with carcinogenic properties, claims must be made within 2 years after knowledge that the OD was proximately caused by or resulted from the nature of the employment.		
Idaho	An OD is due to the nature of an employment in which the hazards of the OD actually exist, are characteristic of and peculiar to the occupation. It must be actually incurred in the employment. An OD does not include psychological injuries, disorders or conditions unless certain conditions are met. Compensation shall be paid for specified diseases contracted in specified ways or specified professions, as listed in statute. The list is not exhaustive, but any other OD may not result from hazards common to the public in general.	Claims forever barred unless: (1) written notice of the manifestation of the OD is given to the employer within 60 days of the first manifestation or the death or, if the employer cannot be reasonably located, to the industrial commission within 90 days after the first manifestation or the death; and (2) a claim for benefits is filed with the industrial commission within 1 year of the first manifestation or the death.	Liability for employer, or the surety on the risk for the employer, in whose employment occurred the last injurious exposure, provided the employee was exposed for at least 60 days by that same employer. The 60-day limitation does not apply to cardiovascular, pulmonary or respiratory diseases contracted by paid firefighters.	Benefits paid when an employee is disabled from performing work in the last occupation in which the injurious exposure occurred. Disability means a decrease in wage-earning capacity.
Illinois	An OD is deemed to arise out of employment if there is a rational, causal connection between the conditions under which the work is performed and the OD. An employee is conclusively deemed to have been exposed to the hazards of an OD when, for any length of time, he or she is employed in an occupation in which the hazards of the disease exist. In a claim of exposure to atomic radiation, the exposure must be verified by the records of the central registry of radiation exposure maintained by the state. If a miner suffers from pneumoconiosis and was employed 10 or more years in one or more coal mines, there is a rebuttable presumption that the pneumoconiosis arose out of the employment. If a deceased miner was employed 10 or more years in one or more coal mines and died from a respirable disease, there is a rebuttable presumption that the death was due to pneumoconiosis.	Disablement must occur within 2 years of the last day of the last exposure to the hazards of the disease. In cases of berylliosis or the inhalation of silica dust or asbestos dust, disablement must occur within 3 years of the last day of the last exposure. In the case of exposure to radiological materials or equipment, disablement must occur within 25 years after the last day of the last exposure.	Liability for employer in whose employment occurred the last exposure, regardless of the length of time of the exposure. In cases of silicosis or asbestosis, liability only for the last employer in whose employment occurred the last exposure that lasted 60 days or more.	Disablement means being impaired, temporarily or permanently, in the function of the body or becoming disabled from earning full wages at the work in which the employee was engaged when last exposed to the OD or any other suitable work.

State	Proof of Causation	Latency Periods	Apportionment of Liability	Benefits If No Lost Work
Indiana	An OD arises out of employment only if there is a rational, direct, causal connection between the conditions under which the work is performed and the OD was a natural incident of the work, can be fairly traced to the employment as the proximate cause, does not come from a hazard to which workers would have been equally exposed outside the employment, and is incidental to the character of the business. An employee is conclusively deemed to have been exposed to the hazards of an OD when, for any length of time, he or she is employed in an occupation in which the hazards of the disease exist. In cases involving silicosis or asbestosis, an exposure lasting less than 60 days is not considered a last exposure.	Disablement must occur within 2 years of the last day of the last exposure. For OD's caused by the inhalation of silica dust or coal dust, disablement must occur within 3 years of the last exposure. For OD's caused by exposure to radiation, disablement must occur within 2 years of the date on which the employee knew or should have known of the nature of the OD and its causal relationship to the employment. For OD's caused by the inhalation of asbestos dust, disablement must occur within 35 years of the last exposure. Death must occur within 2 years of the date of disablement, except that there is no bar to compensation if: (1) death occurs during the pendency of a timely claim that has not been resolved or is being appealed or (2) death occurs within 2 years after the end of a fixed period of compensability, but no later than 300 weeks after the date of disablement.	Liability for employer in whose employment occurred the last exposure, regardless of the length of time of the last exposure. For silicosis or asbestosis, liability only for the last employer in whose employment occurred the last exposure lasting 60 days or more. The insurance carrier whose policy was in effect on the last day of the exposure rendering the employer liable is also liable. If an employer, at the time of the last exposure, was exposed in the joint service of 2 or more employers, the employers must contribute to the compensation in proportion to their wage liability to the employee.	Disability means being incapacitated from earning full wages at the work in which the employee was engaged when last exposed by the employer from whom compensation is claimed.
Iowa	An OD must have a direct causal connection with the employment and must follow as a natural incident from injurious exposure occasioned by the nature of the employment. It need not have been foreseen, but it must appear to have had its origin in a risk connected with the employment and to have resulted from that risk as a rational consequence. A disease resulting from a hazard to which the employee could have been equally exposed outside of the employment is not an OD.	Disablement or death must result within 1 year or, in the case of pneumoconiosis, within 3 years of the last injurious exposure. Death must follow continuous disability (for which a claim was timely made) and must result within 7 years of the last exposure. If disablement or death is caused by latent or delayed pathological conditions, blood or other tissue changes or malignancies due to occupational exposure to x-rays, radium, radioactive substances/machines or ionizing radiation, claims must be filed within 90 days of disablement, death or the date the employee knew or should have known the disablement was caused by overexposure to those substances and the relation to the employment.	Liability for employer in whose employment occurred the last injurious exposure.	Disablement means actually incapacitated from performing work or from earning equal wages in other suitable employment. Employees who are able to continue in employment receive only medical services for the OD.
Kansas	An OD must arise out of and in the course of the employment, result from the nature of the employment, and be contracted during the employment.	Disablement or death must result within 1 year or, in the case of silicosis, within 3 years of the last injurious exposure. Death must follow continuous disability	Liability for employer in whose employment occurred the last injurious exposure, and the insurance carrier on the risk at that time. In the case of	Disablement means actually incapacitated, partially or totally, from performing the work required by the occupation in which exposure to the

State	Proof of Causation	Latency Periods	Apportionment of Liability	Benefits If No Lost Work
Kansas cont'd	"Nature of the employment" means there is a particular and peculiar hazard of the OD that attaches to the employment, which distinguishes it from other employment and creates a hazard of the OD in excess of the hazard in general. The OD must appear to have had its origin in a risk connected with the employment and must result as a reasonable consequence of the risk. Compensation is not payable for emphysema unless it is proved by clear and convincing medical evidence to a reasonable probability that it was caused solely by the employment.	from the disease and result within 7 years of the last exposure. The time limits do not apply for disablement or death due to occupational exposure to ionizing radiation.	silicosis, liability only for employer in whose employment the employee was last injuriously exposed for at least 60 days, and the insurance carrier on the risk at that time.	hazards of the disease occurred.
Kentucky	An OD arises out of and in the course of the employment. An OD is deemed to arise out of employment if there is a causal connection between the work conditions and the OD, the OD can be seen to have followed as a natural incident to the work, and the OD can be fairly traced to the employment as the proximate cause. An OD must be incidental to the character of the business. It need not have been foreseen, but it must appear to be related to and to have flowed from a risk connected with the employment.	Claims forever barred unless filed within 3 years of the last injurious exposure or the date the employee first experienced a distinct manifestation of the OD, whichever occurs last. In any event, the claim must be filed within 5 years of the last injurious exposure. If death results during that period, a claim must be filed within 3 years of the death. In cases of radiation disease or asbestos-related disease, a claim must be filed within 20 years of the last injurious exposure. For pneumoconiosis resulting from exposure to coal dust, the employee must have been exposed in the state for at least 2 continuous years during the 10 years immediately preceding the last exposure, or for any 5 of the 15 years immediately preceding the last exposure.	Liability for employer in whose employment occurred the last exposure and the insurance carrier at that time. Benefits for coal-related occupational pneumoconiosis are paid 50% by the employer and 50% by the state coal workers' pneumoconiosis fund.	Disability is measured by the loss of the employee's earning capacity. Commonwealth Transportation Cabinet v. Blackburn, 890 S.W.2d 627 (1994).
Louisiana	An OD must be due to causes and conditions characteristic of and peculiar to the particular occupation or employment in which the employee was exposed to the OD. An OD contracted by an employee who has been engaged in work for a particular employer for less than 12 months is presumed to be non-occupational and not to have been contracted in the course of the employment. The presumption may be rebutted with an overwhelming preponderance of evidence that the OD was contracted within the 12 months.	Claims barred unless filed within 6 months of the date the OD manifests itself, the date the employee is disabled from working as a result of the OD, or the date the employee knows or should know that the disease is occupationally related. Death claims barred unless filed within 6 months of the date of death or the date the claimant had reasonable grounds to believe the death resulted from an OD.	Liability for employer out of whose employment the OD arose.	Benefits are payable if an employee dies or becomes disabled.

State	Proof of Causation	Latency Periods	Apportionment of Liability	Benefits If No Lost Work
Maine	An OD is due to causes and conditions characteristic of a particular occupation and arises out of and in the course of employment. Silicosis is presumed not to be an OD unless the employee was exposed to the inhalation of silica dust over a period of at least 2 of the 15 years immediately preceding the date of disability.	Incapacity must result within 3 years of the last injurious exposure. That limitation does not apply to full-time firefighters who file claims for occupationally-related cancer and were last injuriously exposed after 1-1-85. The limitation also does not apply to asbestos-related diseases. Claims are barred unless filed within 2 years of the date of incapacity. For an OD due to exposure to radioactive substances, claims must be filed within 2 years of the later of the date of incapacity or the date the claimant knew or should have known of the relationship between the OD and the employment. The 2 years are tolled: (1) until the employer files a report of injury; or (2) for any period during which the employee is unable to file because of physical or mental incapacity. If an employee fails to file within 2 years due to mistake of fact as to the cause and nature of the OD, a claim may be filed within a reasonable time. Claims for further compensation are barred if not filed within 1 year, or within 40 years for an asbestos-related OD, of the last previous payment.	Liability for employer in whose employment occurred the last injurious exposure lasting at least 60 days and the insurance carrier on the risk at that time. For asbestos-related diseases, liability only for employer in whose employment occurred the last injurious exposure to asbestos and the insurance carrier at that time.	A "personal injury" occurs only when an employee dies or becomes partially or totally incapacitated from working. No basis for award unless the employee is actually incapacitated. Manzo v. Great Northern Paper Co., 615 A.sd 605 (1992).
Maryland	An OD must be due to the nature of the employment in which hazards of the OD exist and must have manifestations that are consistent with those known to result from exposure to a biological, chemical or physical agent that is attributable to the type of employment in which the employee was engaged before disablement. The employee must have been employed in that employment prior to disablement.	Claims barred unless filed within 2 years of disablement or death or, for pulmonary dust disease, within 3 years of the date of disablement or death or the date the claimant had actual knowledge that the disablement was caused by the employment.	Liability for employer in whose employment occurred the last injurious exposure and for the insurer liable for the risk at that time.	Disablement means becoming partially or totally incapacitated from performing the work of the last occupation in which the employee was injuriously exposed to the hazards of the OD.
Massa-chusetts	An OD is considered a personal injury if the nature of the employment is such that the hazard of contracting the OD is inherent in the employment.	An employer must submit notice of injury to the division of administration within 7 calendar days (not including Saturdays, Sundays and legal holidays) of receiving notice of an injury arising out of and in the course of employment.	Liability for employer out of whose employment the OD arose.	Compensation is awarded solely for impairment of earning capacity. Federico's Case, 1286 N.E. 599 (1933). An employee must be incapacitated from earning full wages for at least 5 calendar days.

State	Proof of Causation	Latency Periods	Apportionment of Liability	Benefits If No Lost Work
Michigan	An OD must be due to causes and conditions characteristic of and peculiar to the business and arise out of and in the course of the employment. An ordinary disease of life to which the public is generally exposed is not compensable. Mental disability and conditions of the aging process, including but not limited to heart and cardiovascular conditions, are compensable if significantly contributed to or aggravated or accelerated by the employment. Mental disability must arise out of actual events of employment, not unfounded perceptions. A hernia must be clearly recent in origin, result from a strain arising out of and in the course of the employment and be promptly reported to the employer. Respiratory and heart diseases are deemed to arise out of and in the course of employment, in the absence of evidence to the contrary, for full-time firefighters.	Claims must be made within 2 years after the date the claimant knew or should have known that the OD or death was work related.	Liability for employer who last employed the employee in the employment to the nature of which the OD was due and in which it was contracted.	Disability means a limitation in wage earning capacity in work suitable to the employee's qualifications and training. It is not sufficient for claimant to simply prove work-related injury. Michales v. Morton Salt Co., 538 N.W.2d 11 (1995).
Minnesota	Disputed issues of fact are determined by a preponderance of the evidence. An OD arises out of and in the course of employment peculiar to the occupation and is due to causes in excess of the ordinary hazards of employment. Ordinary diseases of life are not compensable, unless the disease follows as an incident of an OD or unless the exposure peculiar to the occupation makes the disease an OD hazard. A disease arises out of the employment only if it is causally connected to the conditions under which the work is performed and if the OD follows as a natural incident of the work. An employer is not liable for any OD that cannot be traced directly and proximately to the employment, is not recognized as a hazard characteristic of and peculiar to the employment or results from a hazard to which the worker would have been equally exposed outside the employment.	No compensation unless claimant gives notice of the OD within 180 days after the occurrence of the injury. A claimant unable because of mental or physical incapacity to give notice within 180 days from the injury must give notice within 180 days after the incapacity ceases.	Liability for employer in whose employment occurred the last significant exposure and for the insurer who was on the risk at that time.	Benefits are paid only for periods of disability.

State	Proof of Causation	Latency Periods	Apportionment of Liability	Benefits If No Lost Work
Minnesota cont'd	If, immediately preceding the disability or death, the employee was on active duty with a fire or police department and had on file a doctor's written report stating that the employee did not have myocarditis, coronary sclerosis, pneumonia or its sequel, the disease is presumptively an OD. If an emergency medical care provider, a police officer or a firefighter contracts an infectious or communicable disease due to exposure in the course of employment outside of a hospital, the disease is presumptively an OD. A firefighter who has a disabling cancer caused by exposure to heat, radiation or a known or suspected carcinogen is presumed to have an OD.			
Mississippi	An OD must arise out of and in the course of employment, without regard to fault as to its cause. An OD is deemed to arise out of and in the course of employment when there is evidence that there is a direct, causal connection between the work performed and the OD.	Claimant must notify employer of the OD within 30 days after the occurrence of the injury. Claims barred unless application for benefits filed within 2 years after the date of injury or death. The 2-year provision begins to run when a compensable OD becomes reasonably apparent. Tabor Motor Co. v. Garrard, 233 So.2d 811 (1970).	Liability for employer out of whose employment the OD arose.	Disability means incapacity to earn the wages the employee was receiving at the time of the disablement in the same or other employment. No benefits unless disability lasts at least 5 days.
Missouri	An OD arises with or without human fault, out of and in the course of employment. Ordinary diseases of life are not compensable, unless the disease follows as an incident of an OD. An OD need not be foreseen, but it must appear to have had its origin in a risk connected with the employment and to have flowed from that source as a rational consequence. An OD must be clearly work related and is not compensable merely because work was a triggering or precipitating factor. An employee is conclusively deemed to have been exposed to the hazards of an OD when for any length of time, employed in an occupation in which the hazard of the disease exists.	Claims barred unless filed within 2 years after it becomes reasonably discoverable and apparent that a compensable OD has been sustained.	Liability for employer in whose employment the employee was last exposed, regardless of the length of time of the last exposure. If: (1) the OD is the result of repetitive motion, (2) exposure to repetitive motion lasted less than 3 months, and (3) exposure to repetitive motion with a prior employer was the substantial contributing factor to the injury, the prior employer is liable.	No benefits unless employee is disabled for at least 3 regularly scheduled work days.
Montana	An OD is harm, damage or death arising out of or contracted in the course and scope of employment and caused by events occurring on more than a single	Claims must be presented within 1 year of the date the claimant knew or should have known the condition resulted from an OD. Claims for death benefits must	Liability for employer in whose employment occurred the last injurious exposure, and the insurer providing coverage at the time. In the case of	Disablement means becoming physically incapacitated from performing work in the workers job pool. No compensation is payable for partial disability.

State	Proof of Causation	Latency Periods	Apportionment of Liability	Benefits If No Lost Work
Montana cont'd	day or work shift. The term does not include a physical or mental condition arising from emotional or mental stress or from a nonphysical stimulus or activity. OD's are deemed to arise out of employment only if there is a direct causal connection between the conditions under which the work is performed and the OD, the OD can be seen to have followed as a natural incident of the work, the OD can be fairly traced to the employment as the proximate cause, the OD did not come from a hazard to which workers would have been equally exposed outside of the employment, and the OD is incidental to the character of the business and not independent of the employment relationship.	be presented within 1 year of the date the claimant knew or should have known the death was related to an OD.	pneumoconiosis, both the coal mine operator at the time of the exposure and any coal mine operator who later acquires the mine are liable.	
Nebraska	An OD arises out of and in the course of employment. The claimant must prove by a preponderance of the evidence that the employment caused the OD.	Claims must be made within 6 months after disability. Raymond v. Buckridge, Inc., 237 N.W.2d 412.	Liability for employer out of whose employment the OD arose.	No benefits unless employee is actually disabled for at least 7 calendar days. Disability is defined in terms of employability and earning capacity rather than in terms of loss of bodily function. McGee v. Panhandle Technical Sys., 387 N.W.2d 709 (1986).
Nevada	Claimant must establish by a preponderance of the evidence that the OD arose out of and in the course of the employment. If an employee files a claim after employment has been terminated for any reason, there is a rebuttable presumption that the OD did not arise out of and in the course of the employment. An OD arises out of and in the course of employment if: there is a direct causal connection between the work conditions and the OD; the OD followed as a natural incident of the exposure occasioned by the work; the employment is the proximate cause; and the OD is not the result of a hazard to which workers would have been equally exposed outside the employment. In cases of radium poisoning or exposure to radioactive properties or substances or to x-rays or ionizing radiation, the OD must have been contracted in Nevada.	Claims must be filed within 90 days of the date the employee knew of the disability and its relationship to the employment. Claims for death benefits must be filed within 1 year of the death of the employee.	Liability for employer that employed the worker at the time of disablement and falls within the provisions of the chapter or accepts the terms of the chapter.	Disablement means being physically incapacitated from engaging in any occupation for which the employee is or becomes reasonably fitted by education, training or experience. No compensation may be paid for disability that does not incapacitate the employee for at least 5 cumulative days within a 20-day period from earning full wages. That limitation does not apply to medical benefits, which must be paid from the date of application for payment of medical benefits.

State	Proof of Causation	Latency Periods	Apportionment of Liability	Benefits If No Lost Work
Nevada cont'd	Disabling cancer is presumed to have developed out of and in the course of employment for any person who has been a firefighter for at least 5 years and was exposed, in the course of employment, to a known carcinogen, which is reasonably associated with the cancer. A disease of the lungs is conclusively presumed to have arisen out of and in the course of employment if a person was a full-time, salaried police officer for 5 continuous years before the disablement or was a firefighter for 2 years. Diseases of the heart are conclusively presumed to have arisen out of and in the course of employment for a person who was a full-time, salaried firefighter or police officer for 5 continuous years before the disablement.			
New Hampshire	An OD must arise out of and in the course of employment and be due to causes and conditions characteristic of and peculiar to the employment. It does not include diseases that existed at the beginning of the employment. A presumption that heart or lung disease is an OD exists for firefighters until one month after the firefighter's 65th birthday or 5 years after the firefighter's retirement. A presumption that cancer is an OD exists for firefighters until 20 years after the firefighter's retirement.	Claims barred unless filed within 3 years after the last date of injurious exposure or the date on which the claimant first knew or should have known of the OD and its relationship to the employment, whichever is later.	Liability for employer out of whose employment the OD arose.	No benefits unless the employee is actually disabled for at least 3 days.
New Jersey	An OD must arise out of and in the course of employment and be due in a material degree to causes and conditions that are or were characteristic of or peculiar to the particular employment.	Claims barred unless a petition is filed within 2 years of (1) the date on which the claimant first knew of the nature of the disability and its relation to the employment, (2) the date the employer failed to make payment pursuant to the terms of an agreement, or (3) the date of the last payment of compensation.	Liability for employer out of whose employment the OD arose.	A condition that does not impair an employee's ability to work will be compensable only if it is serious enough to interfere substantially with other aspects of the employee's life; injury or disease that merely detracts from former efficiency is not compensable unless it is more than minor. Perez v. Pantasote, Inc., 469 A.2d 22 (1984).
New Mexico	Disablement must arise out of and in the course of employment and must be proximately caused by an OD that is not intentionally self-inflicted. OD or death from silicosis or asbestosis must be the result of at least 1250 work shifts in the state in the 10 years immediately	Silicosis or asbestosis must result in disablement, and death from silicosis or asbestosis must occur, within 2 years of the last day on which the employee actually worked for the employer against whom compensation is claimed. Death from a cause other than silicosis or	Liability only for employer in whose employment the employee was last injuriously exposed. For silicosis or asbestosis, only the employer in whose employment the employee was last exposed to harmful quantities of silicon dioxide dust or asbestos dust during a	Disablement includes both total incapacity to perform any work and partial incapacity to perform some percentage. Disablement must last at least 7 days.

State	Proof of Causation	Latency Periods	Apportionment of Liability	Benefits If No Lost Work
New Mexico cont'd	preceding the OD or death. "Arising out of the employment" exists only if there is a direct causal connection between the work conditions and the OD, which can be traced to the employment as the proximate cause. In all cases where the employer denies that an OD is the material and direct result of the work conditions, the employee must establish that causal connection as a medical probability by medical expert testimony.	asbestosis must occur within 1 year of the last day on which the employee actually worked for the employer against whom compensation is claimed. OD or death resulting from exposure to radioactive or fissionable materials must occur within 10 years of the last day on which the employee actually worked for the employer against whom compensation is claimed.	period of at least 60 days.	
New York	If the employee was engaged in a specified activity at or immediately prior to the date of disablement, and the disease is one scheduled for that activity, the disease is presumed to have been due to the nature of the employment. Absent substantial evidence to the contrary, any exposure to the hazards of compressed air or to harmful dust for at least 60 days is presumed to be injurious exposure.	Notice of the OD must be filed within 2 years of the later of the date of disablement or the date the claimant knew or should have known that the OD was due to the employment.	Liability for employer who last employed the employee in the employment to the nature of which the disease was due and in which it was contracted. If the OD was contracted while the employee was in the employment of a prior employer, the last employer can appeal to the board for an apportionment of benefits, unless the OD is silicosis, a dust disease or oompressed air illness.	Disablement means being disabled from earning full wages at the work at which the employee was last employed.
North Carolina	Only diseases and conditions listed in statute are deemed to be OD's. OD's caused by chemicals are deemed to be due to exposure only when, as a part of the employment, the employee is exposed to the chemicals in a form, quantity and frequency that will cause the OD. Exposure to the hazards of asbestosis or silicosis for at least 30 working days within 7 consecutive calendar months is deemed injurious.	Claims must be filed within 2 years after the employee is disabled by the OD.	Liability for employer in whose employment the employee was last injuriously exposed, and the insurance carrier on the risk at that time.	Disability means incapacity to earn the wages previously earned. Disability is defined in terms of a diminution in earning power. Pruitt v. Knight Publishing Co., 218 S.E.2d 876 (1975). Medical benefits paid where awards are made for either disability or for damage to organs resulting from an OD.
North Dakota	An OD arises out of and in the course of employment and must be established by medical evidence supported by objective medical findings. An OD must result from a hazard to which the employee is subjected in the course of employment. It must be incidental to the character of the business and not independent of the employment. Ordinary diseases of life to which the general public outside of employment is exposed are not OD's. The claimant bears the burden of proving entitlement to benefits.	Claims for disability must be filed within 1 year after the claimant knew or should have known that the employee had a work-related OD and either lost wages as a result of the disability or received medical treatment. Claims for death must be filed within 2 years after the death.	Liability for employer out of whose employment the OD arose.	Claims may be filed when an employee has either lost wages or received medical treatment as a result of the OD. Disability means loss of earnings capacity and may be total (temporary or permanent) or partial. Date of first disability means the first date the employee was unable to work due to an OD.

State	Proof of Causation	Latency Periods	Apportionment of Liability	Benefits If No Lost Work
Ohio	An OD must be contracted in the course of employment that results in a hazard that distinguishes it from employment generally and creates a risk of contracting the OD in greater degree and in a different manner from the public in general. Scheduled diseases are deemed OD's, but the schedule is not exclusive.	Claims are forever barred unless filed within 2 years of death or the first diagnosis of the OD by a licensed physician.	Liability for employer out of whose employment the OD arose.	No benefits are allowed for the first week after the OD is contracted. Purpose of disability benefits is to compensate the employee for lost earnings. State ex rel. Brown v. Indus. Comm. (623 N.E.2d 55 (1993). Benefits for cardiovascular, pulmonary or respiratory diseases of firefighters and police officers are payable only for total disability or death.
Oklahoma	An OD is a disease due to causes peculiar to the particular occupation that arises out of and in the course of employment. There is a rebuttable presumption that an OD does not arise out of and in the course of employment if notice is not given to the employer within 90 days of the employee's separation from employment.	Claims must be filed within 2 years after the date of the last trauma or hazardous exposure or the date of death. For asbestosis, silicosis or exposure to nuclear radiation, a claim may be filed within 2 years of the date of last hazardous exposure or the date the OD first became manifest.	Liability for employer in whose employment occurred the last injurious exposure and the insurance carrier on the risk at that time. For silicosis or asbestosis, the employer in whose employment occurred the last exposure lasting at least 60 days and the insurance carrier on the risk at that time.	No compensation allowed for the first 3 calendar days of disability.
Oregon	An OD is any disease arising out of and in the course of employment, caused by substances or activities to which an employee is not ordinarily subjected or exposed, requiring medical services or resulting in disability or death. The worker must prove that the employment conditions were the major contributing cause of the disease. For preexisting diseases, the worker must prove that employment conditions were the major contributing cause of the combined condition and pathological worsening of the disease. An OD must be established by medical evidence supported by objective findings. For persons employed 5 or more years as firefighters, diseases of the lungs or respiratory tract, hypertension and cardiovascular-renal disease are OD's and are presumed to result from employment.	Claims must be filed within 1 year of: the date the employee first discovered or should have discovered the OD; the date the claimant became disabled; the date a physician diagnosed the OD; or the date the claimant knew or should have known that the employee's death was due to an OD.	Liability for employer out of whose employment the OD arose.	No compensation during the first 3 calendar days after the worker leaves work or loses wages as a result of the OD.
Pennsylvania	OD's specifically enumerated and OD's to which the employee is exposed by reason of employment, which are peculiar to the occupation and which are not common to the general population are compensable OD's. An OD is rebuttably presumed to arise out of and	Claims must be filed within 16 months after compensable disability begins or death occurs. No compensation allowed unless notice is given within 120 days after the beginning of compensable disability.	Liability for employer out of whose employment the OD arose.	For an OD to constitute a disability and a basis for compensation, it must impair the claimant's earning capacity. Ryden v. Johns-Manville Products, 518 F.Supp. 311 (D.C. 1981).

State	Proof of Causation	Latency Periods	Apportionment of Liability	Benefits If No Lost Work
Pennsylvania cont'd	in the course of employment if the employee was employed, at or immediately before the date of disability, in any occupation in which the OD is a hazard. Benefits for silicosis, anthraco-silicosis, coal-worker's pneumoconiosis or asbestosis are paid only when the employee was employed in the state for at least 2 of the 10 years immediately preceding the date of disability, in an occupation having a silica, coal or asbestos hazard.			
Rhode Island	An OD is a disease due to causes peculiar to a particular occupation. A scheduled disease is deemed an OD if the disease is due to the nature of the employment in which the employee was engaged and was contracted therein.	Suits must be filed within 2 years of the date of disablement (partial or total incapacity to work).	Liability for employer who last employed the employee in the employment to the nature of which the disease was due and in which it was contracted. The employer may petition the court for an apportionment among the other employers who employed the employee in the employment causing the disease.	Disability means being disabled from earning full wages at the work at which the employee was last employed.
South Carolina	An OD is a disease arising out of and in the course of employment, due to hazards peculiar to the occupation. as a direct result of continuous exposure to the normal working conditions of the occupation. An OD is not an ordinary disease of life to which the general public is equally exposed. Any heart disease or respiratory disease is presumed to have arisen out of and in the course of employment if the employee is a firefighter, the employee was under 37 years old when first hired as a firefighter, the employee successfully passed a physical exam upon hire, a written report of that exam was filed with the fire department, no evidence of heart or respiratory disease was mentioned in the report, and the disease developed while, or within 24 hours of, actively fighting a fire. For byssinosis, the employee must have been exposed to dust in employment for at least 7 years. There is no presumption that disablement from any cause is the result of an OD, nor that an OD will result in disablement.	An OD must be contracted within 1 year of the last exposure to the hazard peculiar to the occupation that caused the disease. For pulmonary disease arising out of the inhalation of organic or inorganic dusts, the period is 2 years. The time limitation does not apply to diseases due to exposure to ionizing radiation.	Liability for employer out of whose employment the OD arose.	Disablement means being actually incapacitated, partially or totally, because of an OD, from performing work in the last occupation in which occurred injurious exposure to the disease. Partial disability means the inability to work in the particular occupation, and total disability means the inability to perform work in any occupation.

State	Proof of Causation	Latency Periods	Apportionment of Liability	Benefits If No Lost Work
South Dakota	An OD is a disease peculiar to the occupation in which the employee was engaged and due to causes in excess of the ordinary hazards of employment, including any disease due to exposure to or contact with any radioactive material.	Claims must be filed within 2 years after the employee becomes disabled from the OD or within 2 years after the date of death. No compensation unless notice of the OD is given to the employer within 6 months after the employment has ceased or within 90 days after death. For exposure to ionizing radiation, the time for filing claims and notices does not begin to run until 1 year after the date the employee first suffered incapacity and knew or should have known that the OD was caused by employment.	Liability for employer in whose employment occurred the last injurious exposure. For silicosis, the only employer liable is the last employer in whose employment occurred the last injurious exposure that occurred for 60 days or more.	Disability means becoming actually and totally incapacitated from performing work in the last occupation in which injurious exposure to the OD occurred.
Tennessee	An OD is any disease arising out of and in the course of employment. A disease is deemed to be an OD only if it followed as a natural incident of the work, the employment is a proximate cause, it did not originate from a hazard to which workers would be equally exposed outside employment, it originated from a risk connected with the employment, and there is a direct causal connection between the conditions under which the work was performed and the OD. Diseases of the heart and lung and hypertension arising out of and in the course of any employment are deemed OD's. An employee (or dependents) entitled to benefits under the federal Coal Mine Health and Safety Act of 1969 and the Black Lung Benefits Act of 1972 are deemed totally disabled from coal worker's pneumoconiosis.	Claims forever barred unless filed within 1 year of the beginning of the incapacity or death. For coal worker's pneumoconiosis, claims must be filed within 3 years of discovery of total disability or death.	Liability for employer out of whose employment the OD arose.	No compensation allowed for the first 7 days of disability. The purpose of the laws is to provide compensation for loss of earning power or capacity. Mathis v. J. L. Forrest & Sons, 216 S.W.2d 967 (1949).
Texas	An OD is a disease arising out of and in the course of employment that causes damage or harm to the physical structure of the body. It is not an ordinary disease of life to which the general public is exposed outside employment.	Claims must be filed within 1 year of the date on which the employee knew or should have known the disease was related to the employee's employment.	Liability for employer in whose employment occurred the last injurious exposure.	Disability means the inability to obtain employment at wages equivalent to the pre-injury wage.
Utah	An OD is any disease or illness that arises out of and in the course of employment and is medically caused or aggravated by that employment.	Claims must be filed within 6 years after the date the cause of action arose. For death benefits, claims must be filed within 1 year of the date the claimant knew or should have known that the death was caused by an OD, but in no	Liability only for employer in whose employment occurred the last injurious exposure, if the exposure with that employer was a substantial contributing cause of the OD and the employee was employed by that employer for at least 12	Benefits payable if employee dies or becomes disabled.

State	Proof of Causation	Latency Periods	Apportionment of Liability	Benefits If No Lost Work
Utah cont'd		case more than 6 years after the cause of action arose.	consecutive months. If neither of those 2 conditions is met, liability is apportioned between employers based on the employers' causal contribution to the OD.	
Vermont	An OD is a disease arising out of and in the course of employment, due to causes and conditions characteristic of the particular occupation and to which an employee is not ordinarily exposed outside employment.	Disablement must result within 5 years of the last injurious exposure. Death must occur during employment or follow continuous disability (meeting above criteria) and result within 12 years of the last injurious exposure. The time limits do not apply for OD's due to exposure to ionizing radiation; in that case, a claim must be filed within 1 year of the date upon which the employee first suffered incapacity and knew or should have known that the OD was caused by the employment.	Liability only for employer in whose employment occurred the last injurious exposure, and the insurance carrier on the risk at that time.	Benefits paid starting the 8th day after the employee is disabled. Total disability means the incapacity to perform any kind of available work. Partial disability means the ability to perform gainful work at some suitable occupation but for less compensation than that received from the occupation in which the OD was contracted.
Virginia	An OD is a disease arising out of and in the course of employment, provided there is a direct causal connection between the work and the OD, the OD followed as a natural incident of the work, the employment is the proximate cause, the employee did not have substantial exposure to the OD outside the employment, the OD is incidental to the character of the business, and the OD had its origin in a risk connected with the employment. The OD need not have been foreseen. An OD not be an ordinary disease of life if the claimant shows by clear and convincing evidence that the disease arose out of and in the course of employment and did not result from causes outside the employment, and it either (1) follows as an incident of OD, (2) is an infectious disease contracted during employment in the direct delivery of health care or (3) is characteristic of the employment and was caused by conditions peculiar to the employment. Conditions of the neck, back or spinal column, hearing loss and carpal tunnel syndrome are not OD's. Respiratory diseases, leukemia and certain cancers caused by documented contact with a	Claims forever barred unless filed within specified time limits. For coal miners' pneumoconiosis, 3 years after diagnosis or 5 years from the date of the last injurious exposure, whichever occurs first. For byssinosis, 2 years after diagnosis or 7 years from the date of the last injurious exposure, whichever occurs first. For asbestosis, 2 years after a diagnosis. For symptomatic or asymptomatic infection with human immunodeficiency virus, including AIDS, 2 years after a positive test for infections. For all other OD's, 2 years after diagnosis or 5 years after the last injurious exposure, whichever occurs first. If death results from an OD within any of the above periods, a claim must be filed within 3 years of the death.	Liability only for employer in whose employment occurred the last injurious exposure, and the insurance carrier on the risk at that time. For coal mining businesses, both the operator at the time of the last injurious exposure and the subsequent operator are liable.	No compensation allowed for the first 7 calendar days of incapacity.

State	Proof of Causation	Latency Periods	Apportionment of Liability	Benefits If No Lost Work
Virginia cont'd	toxic substance, among firefighters or emergency services hazardous materials officers are rebuttably presumed to be OD's. Hypertension and heart disease among firefighters, police officers and emergency services hazardous materials officers are rebuttably presumed to be OD's.			
Washington	An OD is a disease arising naturally and proximately out of employment. Respiratory disease is rebuttably presumed to be an OD for firefighters. The presumption exists for up to 60 months following the last date of employment.	Claims must be filed within 2 years of the date the employee received written notice from a physician that (1) the OD exists and (2) a claim for benefits may be filed. In the case of death, claims must be filed within 2 years of the date of death.	Liability for employer out of whose employment the OD arose.	Payments cease once the employee's earning power is restored.
West Virginia	An OD is incurred in the course of and results from employment. It is not an ordinary disease of life, unless it follows as an incident of an OD. A disease is deemed to have been incurred in the course of or to have resulted from the employment only if: (1) there is a direct causal connection between the work and the OD; (2) it can be seen to have followed as a natural incident of the work; (3) it can be fairly traced to the employment; (4) it does not come from a hazard to which workers would be equally exposed outside the employment; (5) it is incidental to the character of and not independent of the business; and (6) it appears to have had its origin in a risk connected with the employment and to have flowed from that risk as a natural consequence.	Claims must be made within 3 years after the day the employee was last exposed or the day the employee knew or should have known of the OD. For pneumoconiosis, claims must be filed within 3 years after the last day of the last continuous exposure lasting 60 days or more or the day the employee knew or should have known of the OD. Claims for death benefits must be made within 1 year after the death.	For pneumoconiosis, liability is allocated (based on time and wages) among all employers who employed the employee for as much as 60 days during the 3 years immediately preceding the date of last exposure. In general, liability for employers in whose employment the employee was exposed to the OD.	No award allowed if the disability does not last longer than 3 days.
Wisconsin	An OD arises out of employment and causes mental or physical harm.	Claims must be filed within 2 years from the date the claimant knew or should have known of the disability and its relation to the employment. No compensation paid unless the employer is notified within 30 days after the claimant knew or should have known of the disability and its relation to the employment.	Liability for employer out of whose employment the OD arose.	Compensation paid only if the disability exists after 7 calendar days from the date the employee leaves work as a result of the OD. Physical incapacity to work resulting in a wage loss is necessary to sustain a compensation award for disability. Montello Granite Co. v. Industrial Commission, 232 N.W. 542 (1930).
Wyoming	An OD results when the risk of contracting it is increased by the nature of the employment. The claimant bears the burden of proving that the OD arose	Claims must be filed within 1 year after a diagnosis is first communicated to the employee or within 3 years from the date of last injurious exposure, whichever	Liability for employer out of whose employment the OD arose. If no single employer can be charged, liability is assigned to each employer equal to the	Legislature intended for employees to be compensated until their earning power is substantially restored. State ex rel. Wyo. Workers' Comp. Div. v. Ohnstad, 802

State	Proof of Causation	Latency Periods	Apportionment of Liability	Benefits If No Lost Work
Wyoming cont'd	out of and in the course of employment, there is a direct causal connection between the work and the OD, the OD followed as a natural incident of the work, the employment is a proximate cause of the OD, the OD does not result from a hazard to which employees would have been equally exposed outside the employment, and the OD is incidental to the character of the business.	occurs later. The 3 year limitation does not apply to OD's caused by ionizing radiation. Claims for death benefits must be filed within 1 year after the date of death.	percentage that employment with that employer contributed to the cause of the OD.	P.2d 865 (1990).

Angus S. King, Jr.
Governor

ATTACHMENT C

Commission on Safety & Health in the Maine Workplace

January 21, 1998

Sen. Mary R. Cathcart, Chair
Rep. Pamela H. Hatch, Chair
Joint Standing Committee on Labor
118th Maine Legislature
Augusta, ME 04333

Dear Senator Cathcart, Representative Hatch, Members of the Joint Standing Committee on Labor:

I am pleased to respond to your request for information and input from the Commission on Safety and Health in the Maine Workplace. In response to your request regarding the availability of the data relative to chemical exposures and occupational disease in Maine, we offer the following information:

I. Availability of Data:

The data on occupational disease and chemical exposures in the workplace in Maine come form three primary sources:

- **Maine Bureau of Health,**
- **Workers' Compensation Board,**
- **Bureau of Labor Standards**

Physicians in Maine are required by law to report cases of occupational disease to the **Bureau of Health** under the ***Occupational Disease Reporting*** program (see attached form). The Bureau of Health passively collects the data from reporting physicians and then compiles and analyzes the information. The last comprehensive report was produced in 1994 covering the period of 1986 through 1993. Reports of 683 occupational disease cases were received by the Bureau of

Health during that 7 year period. The Bureau of Health plans to publish the next annual report on their occupational disease findings in 1998.

The **Workers' Compensation Board** collects data on occupational diseases when Workers' Compensation Claims are filed with the Board (see attached "First Report of Occupational Injury or Disease" form). In box #5 of the First Report form, the employer or insurer is asked to indicate the reason for the claim. One selection that is available to employers/insurers is the category, "Occupational Disease." For the period of 1993 through 1996 only 108 cases were recorded by the Workers' Compensation Board as "Occupational Disease" cases. Further, the data indicates that there have been no occupational disease cases reported to the Board since 1994. Of the 108 cases reported in that 3 year period, only eight reported any information showing indemnity (lost time wage benefits). It appears that the occupational disease data captured by the Workers' Compensation Board may either be incomplete or there have been data system problems that have not allowed the accurate recording of occupational disease cases.

The **Bureau of Labor Standards** (BLS) conducts the annual USDOL survey in Maine of OSHA recordable injuries and illnesses. Because OSHA uses an entirely different definition of occupational disease, this data does not directly correlate with either the Workers' Compensation claim data or the data collected by the Bureau of Health on occupational disease cases reported by physicians.

However, the Bureau of Labor Standards does code all the injury types and diagnoses reported on all Workers' Compensation claims. Each claim is analyzed individually by BLS staff and coded to display the nature of the injury or illness. By analyzing the claim forms and coding them, BLS staff can identify disease claims and claims related to chemical exposure, even if those illnesses were not specifically marked as "occupational disease" on the Workers' Compensation First Report.

Attached are two tables that show the **"Number of Chemical Exposure Claims by Industry, 1993-1996"**; and, **"Reported Disease Claims by Industry, 1993-1996."** This data gives a snapshot of the number of chemical exposure claims and disease claims filed during that 3 year period. Based on the BLS coding of Workers' Compensation claims, there were 332 claims that indicated chemical exposure from 1993 through 1996. During that same period, BLS coding indicates that there were 2,491 "disease" claims reported.

The data extracted by BLS is only a starting point for understanding occupational disease in Maine. Because the Workers' Compensation data may not identify the claim being reported as an occupational disease claim, we must rely on the interpretation of BLS staff to infer that the claim is a "disease" claim or a claim as the result of "chemical exposure."

II. Recommendations:

The Commission on Safety and Health in the Maine Workplace offers the following recommendations relative to the collection of accurate, complete and useful data on occupational disease and workplace chemical exposures in Maine:

- ✓ The various definitions of Occupational Disease must be clarified and, to the extent possible, made more uniform among the Maine state agencies responsible for collecting this data. To the extent possible, data collected in Maine should be correlated with other national data sources (National Institute of Occupational Safety and Health [NIOSH], Occupational Safety and Health Administration [OSHA], USDOL Bureau of Labor Statistics, etc.)

- ✓ The Workers' Compensation Board should continue to be the primary central data collection point for information relating to occupational disease claims in Maine.

- ✓ The Bureau of Health should continue to collect data on occupational disease cases as reported by physicians in Maine.

- ✓ As the Workers' Compensation Board implements a new computer system design and work flow procedures (as recommended in the Coopers and Lybrand study), they must include all necessary data collection components and procedures for occupational disease data and chemical exposure data related to claims that are filed in Maine.

- ✓ The Research and Statistics Division of the Bureau of Labor Standards should take a lead role in coordinating the above recommendations and ensuring that the data collected in Maine on occupational disease is accurate and reliable. In its annual report to the Labor Committee and the full Legislature, the Bureau of Labor Standards should include a specific section analyzing occupational disease and chemical exposures in Maine

On behalf of the members of the Commission on Safety and Health in the Maine Workplace, I would be pleased to address the Labor Committee about any of the above recommendations. In addition, as this issue continues to unfold, please feel free to call upon us for research, analysis and input.

Sincerely,

Elizabeth Stowell

Elizabeth Stowell, Chair
Commission on Safety and Health in the Maine Workplace

cc. Valerie Landry, Commissioner, Maine DOL
Kevin Concanon, Commissioner, Maine DHS
Paul Dionne, Executive Director, Workers' Compensation Board
Alan Hinsey, Director, Bureau of Labor Standards
Dora Mills, MD, Director, Bureau of Health

MAINE OCCUPATIONAL DISEASE REPORTING PROGRAM

Please complete this form on all patients with a reportable occupational disease.
Return form to: Occupational Health Program, Bureau of Health
SHS #11
151 Capitol St.
Augusta, ME 04333
For any questions: (207) 287-5378

CLINICIAN OR FACILITY
NAME:
ADDRESS:
PHONE #

PATIENT NAME (Last) (First) (Middle) (Maiden or aliases)

PATIENT'S ADDRESS AT DIAGNOSIS (Street, City, State, Zip Code)

RACE (check one)	SOCIAL SECURITY NUMBER	DATE OF BIRTH (Month, Day, Yr)	SEX (check one)
☐ Caucasian/White ☐ Black ☐ American Indian ☐ Asian ☐ Unknown ☐ Other		/ /	☐ 1 Male ☐ 2 Female

Does patient currently smoke? ☐ No ☐ Yes **If yes,** how many pack(s) a day?

Is there any reason we should not contact this patient directly? ☐ OK to contact patient

☐ Please do not contact the patient for the following reason(s):

PATIENT'S TELEPHONE NUMBER (including area code)

OCCUPATION

For fishers, please indicate the method of fishing employed, e.g. diving, trawling, digging, gillnetting, dredging, etc.

INDUSTRY

For fishers, please indicate the type of fish caught or harvested, e.g., scallops, lobster, haddock, etc.

NAME OF EMPLOYER and ADDRESS

TELEPHONE NUMBER OF EMPLOYER (including area code)

REPORTABLE DISEASE (please check) ☐ Diagnosed ☐ Suspected Date of Diagnosis

Date of Service

Check all that apply.

☐ Agriculturally -related injury (includes farming, logging, and fishing) . **Please describe how injury occurred, and the physical findings of the injury.**

☐ Asbestosis
☐ Byssinosis
☐ Carpal Tunnel Syndrome
☐ Heavy Metal Poisoning ☐ Lead (level) ____ ☐ Mercury (level) ____ ☐ Arsenic (level) ____ ☐ Cadmium (level) ____
☐ Hypersensitivity Pneumonitis (caused by)
☐ Mesothelioma
☐ Occupational Asthma (caused by)
☐ Outbreaks (agent)
☐ Pesticide Poisoning (name of pesticide)
☐ Silicosis
☐ Solvent Toxicity (name of solvent)
☐ Toxic Gas Poisoning (name of gas)
☐ Other (please describe)

PLEASE CHECK ONE OF THE FOLLOWING: ☐ Work-Related ☐ Not Work-Related ☐ Suspect Work-Related ☐ Unknown

COMMENTS:

FORM COMPLETED BY: **DATE:**

CONFIDENTIAL INFORMATION

White (Occupational Health) Yellow (Your File)

1. INSURER FILE NUMBER:	**EMPLOYER'S FIRST REPORT OF OCCUPATIONAL INJURY OR DISEASE** STATE OF MAINE WORKERS' COMPENSATION BOARD STATION 27 AUGUSTA, MAINE 04333	4. WCB FILE NUMBER:
2. EMPLOYER FILE NUMBER:		5. REASON FOR REPORT: CHECK ALL THAT APPL LOST TIME - ONE OR MORE DAYS ☐ MEDICAL/HEALTH CARE ☐ OCCUPATIONAL DISEASE (Note item 42 below) ☐ CORRECT PRIOR REPORT ☐
3. EMPLOYER UNEMPLOYMENT INSURANCE ACCOUNT NUMBER (UIAN):		

EMPLOYER INFORMATION

6. EMPLOYER NAME:

7. EMPLOYER MAILING ADDRESS AND PHONE:

8. LOCATION IF DIFFERENT FROM MAILING ADDRESS:

9. NATURE OF BUSINESS:

10. NAME OF WC INSURER:

11. POLICY NUMBER:

EMPLOYEE INFORMATION

12. LAST NAME: | FIRST NAME: | M.I.: | 13. SOCIAL SECURITY NUMBER:

14. ADDRESS - NUMBER AND STREET:

15. CITY: | STATE: | ZIP:

16. HOME PHONE: | 17. DATE OF BIRTH: | 18. AGE: | 19. SEX: MALE ☐ FEMALE ☐

20. OCCUPATION:

21. DEPARTMENT:

22. DATE OF HIRE: | 23. DATE CURRENT DUTIES BEGAN: | 24. DOES EMPLOYEE WORK FOR ANOTHER EMPLOYER? YES ☐ NO ☐

25. WEEKLY WAGE AT TIME OF INJURY:

INJURY OR EXPOSURE INFORMATION

26. DATE AND TIME OF INJURY: : AM ☐ PM ☐

27. DID INJURY OR EXPOSURE OCCUR ON EMPLOYER'S PREMISES SHOWN ABOVE? YES ☐ NO ☐

28. IF NO, PLACE WHERE INJURY OR EXPOSURE OCCURRED:

29. DESCRIBE THE EVENTS WHICH RESULTED IN THE INJURY OR DISEASE. (GIVE FULL DETAILS ON ALL FACTORS THAT LED OR CONTRIBUTED TO THE INJURY OR THE ONSET OF DISEASE.)

N
P
S
T
A

30. NAME THE OBJECT, SUBSTANCE, OR EXPOSURE WHICH DIRECTLY BROUGHT ABOUT THE INJURY OR DISEASE.

31. DESCRIBE THE INJURY OR DISEASE AND INDICATE PART OF BODY AFFECTED.

32. PHYSICIAN (NAME AND ADDRESS):

33. ☐ FIRST AID ☐ HOSPITAL ☐ EMERGENCY ROOM ☐ OUT-PATIENT

34. HOSPITAL (NAME AND ADDRESS):

35. DATE EMPLOYER NOTIFIED:

36. TIME EMPLOYEE WORKDAY BEGAN: AM ☐ PM ☐

37. DID EMPLOYEE LOSE ONE OR MORE DAYS WORK? YES ☐ NO ☐ IF NO, SKIP BOXES 38, 39 40 AND 41

38. WAS EMPLOYEE PAID FOR 1/2 DAY OR MORE ON DAY OF INJURY? YES ☐ NO ☐

39. DATE INCAPACITY BEGAN:

40. HAS EMPLOYEE RETURNED TO WORK? YES ☐ NO ☐ IF YES, GIVE DATE:

41. DID EMPLOYEE DIE? YES ☐ NO ☐ IF YES, GIVE DATE:

42. FOR OCUPATIONAL DISEASE DATE OF LAST EXPOSURE: | DATE OF DIAGNOSIS AS OCCUPATIONALLY RELAT

PREPARER INFORMATION

43. PREPARER NAME AND TITLE (TYPE OR PRINT):

SIGNATURE (FORM MUST BE SIGNED):

DATE:

THIS DOCUMENT MAY BE PRODUCED IN ALTERNATIVE FORMATS SUCH AS BRAILLE, LARGE PRINT AND AUDIOTAPE.

Number of Chemical Exposure Claims by Industry, 1993-1996

SIC	Title	Frequency	Cumula-tive Frequency	Percent	Cumulative Percent
5812	Restaurants	46	46	5.5	5.5
3731	Shipbuilding	36	82	4.3	9.9
8211	Schools	33	115	4.0	13.8
8062	Hospitals	30	145	3.6	17.4
2621	Paper Manufacturing	28	173	3.4	20.8
1611	Highway & Street Const.	27	200	3.2	24.0
7011	Hotels	18	218	2.2	26.2
7349	Building Cleaning & Maintenance Services	16	234	1.9	28.1
5411	Grocery Stores	15	249	1.8	29.9
3089	Manufacturing Plastic Products	14	263	1.7	31.6
9221	Police Protection	14	277	1.7	33.3
8051	Nursing Homes	13	290	1.6	34.9
1721	Painting & Paper Hanging Contractors	11	301	1.3	36.2
8221	Colleges & Universities	11	312	1.3	37.5
2037	Mfg. Frozen Fruits, Fruit Juices & Vegetables	10	322	1.2	38.7
9441	Adm. of Social, Human Resources, & Income Maintenance Programs	10	332	1.2	39.9

Source: Workers' Compensation Board Claims database as coded and tabulated by the Maine Department of Labor, Bureau of Labor Standards.

Reported Disease Claims by Industry, 1993-1996

SIC	Title	Frequency	Cumula-tive Frequency	Percent	Cumula-tive Percent
5411	Grocery Stores	247	247	5.2	5.2
8062	Hospitals	222	469	4.6	9.8
3731	Shipbuilding	192	661	4	13.8
5812	Restaurants	153	814	3.2	17
2621	Paper Manufacturing	151	965	3.2	20.2
8211	Schools	145	1,110	3	23.2
3144	Women's Footwear, Except Athletic	106	1,216	2.2	25.4
1611	Highway & Street Construction	92	1,308	1.9	27.4
8051	Nursing Homes	86	1,394	1.8	29.2
3143	Men's Footwear, Except Athletic	77	1,471	1.6	30.8
3111	Leather Tanning & Finishing	71	1,542	1.5	32.3
5311	Department Stores	59	1,601	1.2	33.5
7011	Hotels & Motels	56	1,657	1.2	34.7
2499	Wood Products Mfg., NEC	54	1,711	1.1	35.8
8221	Colleges	48	1,759	1	36.8
1521	Residential Home Building	47	1,806	1	37.8
9441	Admin. of Social, Human Resources, Income Maint. Programs	47	1,853	1	38.8
3089	Plastic Products Mfg.	45	1,898	0.9	39.7
5141	Wholesale Groceries	43	1,941	0.9	40.6
8082	Home Health Care Svcs.	39	1,980	0.8	41.4
2051	Bread & Bakery Prods., Excl. Cookies, Crackers	38	2,018	0.8	42.2
2411	Logging	37	2,055	0.8	43
8111	Legal Services	37	2,092	0.8	43.8
5961	Catalog, Mail Order Houses	36	2,128	0.8	44.5
9224	Fire Protection	36	2,164	0.8	45.3
4213	Trucking, Except Local	35	2,199	0.7	46
5511	Motor Vehicle Dealers	35	2,234	0.7	46.8
7349	Building Cleaning & Maintenance Svcs.	35	2,269	0.7	47.5
8361	Residential Care	34	2,303	0.7	48.2

Reported Disease Claims by Industry, 1993-1996

SIC	Title	Frequency	Cumula-tive Frequency	Percent	Cumula-tive Percent
1794	Excavation Work	33	2,336	0.7	48.9
2231	Broadwoven Fabric Mills, Wool	32	2,368	0.7	49.6
5146	Wholesale Fish & Seafoods	32	2,400	0.7	50.2
4212	Trucking, Local	31	2,431	0.6	50.9
4911	Electric Services	30	2,461	0.6	51.5
7363	Help Supply Services	30	2,491	0.6	52.1

Source: Workers' Compensation Board Claims database as coded and tabulated by the Maine Department of Labor, Bureau of Labor Standards.

DEPARTMENT OF LABOR
BUREAU OF LABOR STANDARDS
45 STATE HOUSE STATION
AUGUSTA, MAINE
04333-0045

ATTACHMENT D

ANGUS S. KING, JR.
GOVERNOR

ALAN C. HINSEY
DIRECTOR

February 5, 1998

Sen. Mary R. Cathcart, Chair
Rep. Pamela H. Hatch, Chair
Joint Standing Committee on Labor
118th Maine Legislature
Augusta, ME 04333

Dear Senator Cathcart, Representative Hatch, Members of the Joint Standing Committee on Labor:

At the Labor Committee work session on 1/22/98 the Committee asked that representatives from the Bureau of Labor Standards, Bureau of Insurance, Bureau of Health and the Workers' Compensation Board convene to pull together additional information and recommendations for the Labor Committee on the subject of occupational disease data and occupational disease definitions in Maine. In addition, Karen Packard, Executive Director of the Maine Institute for Occupational Health Education was asked to participate in the discussions. That group did meet on 1/27/98 to discuss these issues.

After much discussion the work group decided that because of the lack of complete and uniform data on occupational diseases in Maine, it would be inappropriate to make a recommendation to the Legislature on a standardized definition for occupational disease in Maine at this time. However, the work group did see that there is an immediate need to change the occupational disease data collections systems and coordinate the various occupational disease data bases that currently exist in Maine. All members of the work group agreed that changing the data collection forms to ensure that diagnosis codes are required by physicians would be a significant step toward the construction of a common language from which to collect and analyze occupational disease data.

The group makes the following specific recommendations and further, plans are now being developed to ensure that theses changes are realized as soon as possible:

1) Modify the M-1 Form used by the Workers' Compensation Board:

Modify the existing M-1 form that the Workers' Compensation Board requires physicians to submit on all claims. The form must require that the physician provide the ICD-CM diagnosis code. The ICD-CM code is recognized as the national standard for coding health incidents in the medical community at large. The Workers' Compensation Board will ensure that these forms are

submitted and data entered on all workers' compensation claims filed with the Workers' Compensation Board. The Workers' Compensation Board will use the enforcement penalty provisions in the Workers' Compensation Act to ensure that a M-1 form is submitted on all claims. The Workers' Compensation Board will place a high priority on the collection of this M-1 ICD-CM data and will ensure that timely and accurate data entry takes place. This ICD-CM data will be made available to the Bureau of Health and Bureau of Labor Standards.

2) Modify the Occupational Disease Reporting Form used by Bureau of Health:

Modify the existing Occupational Disease Reporting Form that physicians are required to submit to the Bureau of Health anytime that a physician identifies occupational disease. The form must require that the physician provide the ICD-CM diagnosis code when indicating the type of occupational disease being reported. This ICD-CM data collected by the Bureau of Health will be made available to the Workers' Compensation Board and the Bureau of Labor Standards.

3) Eliminate Duplicate Reporting:

If the occupational disease identified by the physician is related to a workers' compensation case and an M-1 form must be filed by that physician with the Workers' Compensation Board, then the physician WILL NOT have to submit the OD Reporting Form to the Bureau of Health. The data collected directly by the Bureau of Health will be occupational disease information not otherwise associated with a workers' compensation claim and, as such, will not represent duplicative data or duplicate filing requirements for physicians.

4) Standardization and possible combination of OD reporting forms:

In the process of modifying the Workers' Compensation Board M-1 form and the Bureau of Health Occupational Disease Reporting form, consideration will be given to combining the forms into one uniform data collection document that meets both the Workers' Compensation Board and Bureau of Health needs, if possible. While the modified forms will require physicians to indicate the ICD-CM code, which had not been previously required, every attempt will be made to make the reporting forms easy to use and the appropriate ICD-CM code easy to identify. The Bureau of Health and Workers' Compensation Board will begin work on modifying the forms and considering a standardized form as soon as possible.

5) Outreach and Education with Physicians Groups, the Medical Community and Workers' Compensation Insurance Carriers and Self-Insureds:

Isabella Tighe of the Workers' Compensation Board and Alison Hawkes, MD, of the Bureau of Health will meet with various physician groups, medical service providers and workers' compensation insurance carriers and self-insureds to begin the outreach and education process regarding the need for complete occupational disease data. They will also use that point of

contact to explain the modifications of forms, the addition of ICD-CM coding, and how the shared data will be used.

6) Electronic Data Sharing:

The Bureau of Labor Standards will take the lead in identifying ways to effectively and efficiently share the electronic data collected by the Bureau of Health and the Workers' Compensation Board. An analysis of the respective data bases will be conducted and the Bureau of Labor Standards Research and Statistics staff (with the assistance of the MDOL Office of Information Processing) will make recommendations for the best way to electronically share the data while protecting the confidentiality of the data and the security of the data bases.

NOTE: Special care will be taken to ensure that any proposed data sharing agreement between the Bureau of Health, the Bureau of Labor Standards, the Workers' Compensation Board, the Bureau of Insurance, or any other governmental agency, will fully comply with all confidentiality restrictions placed on this data, as stated in Maine statutes.

7) Collection of Occupational Disease ICD-CM Data for Two Years:

The work group recommends that the new occupational disease data that has been ICD-CM coded should be collected and analyzed for at least two (2) years BEFORE that data is used by policy makers for the purpose of amending exist state statutes or proposing new occupational disease laws in Maine.

8) Uses of ICD-CM Data:

When the ICD-CM diagnosis data has been collected for at least two years, that data can be used to understand the scope and severity of occupational disease in Maine. It can also be used by policy makers to consider the potential impacts (both positive and negative) to injured workers and employers if changes are proposed to the existing Occupational Definitions in state law. The ICD-CM data will allow researchers to more accurately forecast the incidence of occupational disease in Maine, as well as providing a better understanding of all of the costs associated with occupational disease.

9) Occupational Disease Prevention Strategies:

Finally (but certainly not least), the collection of ICD-CM diagnosis data will significantly improve our ability to develop effective occupational disease prevention strategies. A better understanding of the nature and causation of occupational diseases (that can only be accomplished through complete and thorough reporting by physicians), we all will be able to implement programs and practices that reduces the incidence of occupational disease in all workplaces in Maine.

The nine recommendations made above will serve as the basic action plan for the combined efforts of the Workers' Compensation Board, the Bureau of Health, the Bureau of Insurance, the Bureau

of Labor Standards, and the Maine Institute for Occupational Health Education. These five agencies are committed to working together to accomplish a common goal - namely, the complete and accurate collection of occupational disease data that can be used to analyze trends, design prevention strategies and guide policy makers on these very difficult issues.

Given the recommendations made above and the commitment of the five agencies, we further recommend that L.D. 835 be voted "Ought Not to Pass" by the Labor Committee. We believe that the recommendations, plans and strategies given above will result in the data and analysis that L.D. 835 was attempting to achieve. Specific legislation should no longer be needed to accomplish the goals of L.D. 835. We also recommend that the work group made up of the Workers' Compensation Board, Bureau of Labor Standards, Bureau of Health, Bureau of Insurance, and Maine Institute for Occupational Health Education continue to meet regularly on this issue to ensure that all of the above recommendations are implemented. Isabella Tighe of the Workers' Compensation Board will be the coordinator of the work group's activities. A status report on the recommendations will be incorporated into the legislatively mandated report on the "Status of the Maine Workers' Compensation System" which is presented to the Legislature, with a formal report to the Labor Committee, each year.

We appreciate the opportunity to work on this important issue and report to the Labor Committee. Please feel free to contact any of us at any time.

Sincerely,

Isabella Tighe, Workers' Compensation Board

Alison Hawkes, MD, Bureau of Health

Alan Hinsey, Bureau of Labor Standards

Eric Cioppa, Bureau of Insurance

Karen Packard, Maine Institute for Occupational Health Education

cc: Katy Longley, Commissioner, Dept. Professional & Financial Regulation
Valerie Landry, Commissioner, Dept. of Labor
Kevin Concannon, Commissioner, Dept. Human Services
Paul Dionne, Exec. Director, Workers' Compensation Board
Al Iuppa, Dep. Superintendent, Bureau of Insurance
Dora Mills, MD, Director, Bureau of Health
Elizabeth Stowell, Chair, Commission on Safety and Health in the Maine Workplace

ATTACHMENT E

1996 Survey of Occupational Health Practices

Prepared by:

Karen Packard MS
Executive Director
Maine Institute for Occupational Health Education

and

Allison Hawkes MD
Director, Occupational Health Program
Maine Department of Human Services

Sponsored by:
Occupational Health and Safety Program, Maine Department of Human Services
and
Maine Institute for Occupational Health Education

Waterville, ME
March 24, 1996

INTRODUCTION

The Maine Institute for Occupational Health Education (MIOHE) is a non-profit educational institute designed to provide primary care providers with opportunities for clinical training on occupational medicine issues. MIOHE was established based on the results of a survey conducted in 1992 assessing the occupational health practices of Maine physicians. The Institute plans seminars and conferences on occupational health topics and serves as a center for networking occupational health resources.

The Occupational Health and Safety Program in the Maine Bureau of Health maintains an Occupational Disease Reporting System which mandates that any physician, chiropractor, nurse practitioner, physician assistant , or hospital diagnosing specified occupational diseases are required to report them to the Department of Human Services within 30 days (22 MSRS Chapter 259-A Subsection 1491-1495). Currently, minimal reporting takes place. The Bureau is interested in identifying factors contributing to this. Other services available through this program include training for health care providers on the recognition and case management of selected diseases, access to general and specific information on chemicals, referral to other state programs for information or follow-up services, and generation of statistical analyses and reports on certain topics.

MIOHE and the Bureau of Health have collaborated on this survey to identify occupational health practices of physicians, chiropractors, nurse practitioners, and physician assistants. The results from this survey will be used to help design a more effective occupational injury and illness surveillance system and identify the educational needs of providers.

A total of 2301 surveys were mailed, 609 were returned for an overall response rate of 26%. The length of time for entire survey process was approximately 3 months. Time constraints existed for both the Bureau of Health and MIOHE. The data was needed for strategic planning and grant proposals by the end of March.

RESEARCH QUESTIONS

1. Do health care providers have ongoing needs for education regarding occupational and environmental health?
2. What are the best methods for providing information on occupational and environmental resources?
3. How often are providers seeing work related injuries and illnesses in their practices?
4. Are providers taking occupational and environmental exposure histories? Under what conditions are they taken and how complete are they?
5. Are there variations in reporting by type of clinician?
6. How familiar are clinicians with MIOHE as a resource?
7. Do providers identify general barriers to reporting?
8. Is there a relationship between the barriers perceived and reporting behavior?
9. Do providers want technical assistance with treatment, diagnosis, or reporting?

SURVEY CHARACTERISTICS

A mailed self-administered questionnaire was selected as the most realistic and time efficient survey method for gathering data from busy medical professionals.

Survey Contents:

The survey contained 16 questions. Types of questions included 12 check off, multiple choice questions; two questions that required entering a number; one open ended question; and one single word entry question .

Questions requested information on type of medical provider and specialty, frequency with which they see injured/ ill workers, referrals, useful methods of providing them with information, familiarity with MIOHE, impact of 1993 WC changes on their practice, conditions and completeness of occupational history taking, frequency of diagnosis and reporting of ten selected occupational diseases, interest in technical assistance, and the county in which they practice. The two-sided questionnaire took less than 5 minutes to complete in pilot testing.

The 16 survey questions were constructed by a panel with experience in health research, occupational health, and medicine. The survey was pilot tested on providers from each of the professional disciplines to be surveyed. The survey was edited based on the comments from the pilot testing.

Cover letters and membership lists were obtained from the Maine Medical Association, Maine Osteopathic Association, Maine Academy of Family Physicians, Maine Chiropractic Association and the Downeast Association of Physician Assistants. Cover letters from the appropriate organization were included with the survey. Nurse practitioners and providers without an organizational affiliation received a cover letter from the Maine Institute for Occupational Health Education.

A return envelope without postage was provided. It was important to provide for anonymity in the survey, therefore no records were kept that would allow for individual follow-up. A four week period was allowed for surveys to be returned. The length of time for entire survey process was approximately 3 months.

METHODS

The sample consisted of providers mandated to report occupational injury/ illness under 22 MSRS Chapter 259-A Subsection 1491-1495, and includes allopathic and osteopathic physicians, chiropractors, physician assistants, and nurse practitioners. Licensing lists from the Maine Board of Medicine, Maine Board of Nursing, and Maine Board of Chiropractic Licensure and Examination were obtained. The Board of Medicine list was edited to omit retired, inactive, specialists in radiology, pediatrics, psychiatry, nuclear medicine, anesthesiology, neurology, vascular and thoracic surgery, urology, proctology, legal medicine, plastic surgery, and administration. The Maine Board of Nursing list was edited to include only adult and family nurse practitioners. All physician assistants and chiropractors with instate active licenses were included in the sample.

DATA ANALYSIS

Data processing and analysis was conducted by Al Leighton, Acting Director, Survey Research Center, Muskie Institute, University of Southern Maine, Portland, ME. All coding and data entry were double verified. Data was analyzed using SAS. Surveys with all data missing (ex: retired) were removed from the analysis, giving a final total of 599 surveys to analyze. Unless otherwise noted levels of significance are from Chi-square tests. Ten surveys were received after the deadline and were included in the response rate and comment sections only.

RESULTS AND DISCUSSION

When asked to describe their practice, 27% of the 599 survey respondents indicated their practice was general/ family practice. In a rural state such as Maine, it would be expected that primary care providers would be caring for a large portion of the work related injuries and illness. Internal medicine and surgical specialty providers each comprised 18% of the total respondents. Although chiropractors represented only 12.5% of the sample, they had the highest response rate (34.6%) of any provider type. Thirty percent of the subspecialties indicated under surgery and the "other" category described their practice as orthopedic. Figure 1 illustrates responding providers by type of practice.

Survey responses rates varied across provider groups from a high response rate of 34.6% for chiropractors to a low of 21.4% for nurse practitioners. Comments on the open ended question indicated that the chiropractors feel more of a negative impact from the present Workers' Compensation Laws. The lower response from the nurse practitioners may reflect the fact that no cover letter from a professional organization was included with the survey to that group, that the sample could not screened as thoroughly for those most likely to be treating work related injury/ illness, or that occupational injury/ illness is not as big an issue for nurse practitioners as the other providers surveyed.

Providers are frequently seeing work related injury/ illness in their practices. Seventy-eight percent of providers responding see patients for work related injury/ illness in their practice at least once a month, and 49.2% are seeing them at least twice a week, and 26.7% are seeing cases daily. Figure #2 illustrates the frequency of office visits for work related injury/ illness for all respondents. The type of provider seeing cases of work related injury/ illness at least 2-3 times a week varied significantly ($p=.002$) with 64% of DOs seeing cases this often, 63% of chiropractors, 59% of PAs, 45% of MDs, and 27% of nurse practitioners. Nurse practitioners reported seeing work related injuries significantly less often than other provider types. This may have been due in part to a sampling effect as family nurse practitioners may be seeing more pediatric patients than other providers.

The frequency with which a provider saw work related injury/ illness in practice was a relevant factor when compared with the other variables of history taking, treatment, impact of 1993 Workers' Compensation Law changes, and requests for technical assistance. Sixty-one percent of all respondents treat work related injury/ illness cases themselves with occasional consultation. Those respondents who reported seeing more than one case a week were more than twice as likely to report that they "treat it themselves with occasional consultation" as those who saw less than one case a week ($p=.000$). This group also asked key occupational / environmental history questions more frequently including: description of current job (58% vs. 41%, $p=.001$) and description of previous jobs (62% vs. 38%, $p=.021$). However, this group was less likely to ask about a patient's water supply than those respondents seeing one work related injury case a week or less (31% vs. 18%, $p=.001$). Table #1 summarizes occupational and environmental history taking information by frequency of office visits.

In looking at patient referrals, there were no differences in frequency of referral of patients to a medical or surgical specialty. However, seventy percent of respondents rarely or never refer to occupational medicine specialists as compared to 31% of respondents who refer to occupational medicine specialty at least sometimes (p=.000).

Providing information to busy practitioners in a predominantly rural state has been is an ongoing challenge. When asked about useful methods of providing information on occupational/ environmental medicine, 76% indicated a resource list by mail, 51% an in-state FAX number, 25% Internet/ WEB Site, and 21% e-mail. When asked the most useful method 58.6% indicated that a resource list by mail would be most useful. Figure 3 illustrates the most useful methods of receiving occupational and environmental medicine information. The number of providers using the Internet is impressive and could be considered an important future information and resource alternative.

Name recognition for the Maine Institute for Occupational Health Education was 28% overall with significant differences between provider groups (p=.000). Provider groups that most frequently recognized the name were chiropractors (49%) and physicians assistants (41%). Mailings from the Institute go to all in-state providers with active licenses in these two groups. Name recognition was the least with MDs (23%) and DOs (19%). Mailings to these to groups are limited to the Maine Academy of Family Physicians, the Maine Osteopathic Association, and physicians specializing in occupational medicine. Nurse practitioners recognized the name 28% of the time, and currently mailings go only to nurses belonging to the Maine Association for Occupational Health Nurses or those who have attended a conference in the past.

When asked what kind of impact the 1993 changes in the Workers' Compensation Law had on their practice 26% indicated that the changes made it harder to treat patients, 14% thought the changes made it easier to treat patients, and 48% indicated it made no difference. Those finding it harder to treat patients varied significantly across provider groups. Seventy-six percent of chiropractors find it more difficult to treat patients, compared to 33% of DOs, 17% of PAs, 15% of MDs, and 9% of nurse practitioners (p=.000). Chiropractors stated the 10 day rule made it more difficult for them to see patients early in the course of the injury. Seventy-eight of those indicating that the changes made it easier to treat patients had diagnosed at least 8 cases of work related injury or illness in the past year as compared to those who diagnosed less than eight cases (p=.000). It appears that the changes have made it easier for those treating the most patients. The 48% of those responding that the changes made no difference was put in perspective by a number of comments that respondents did not know about or had no information on the changes.

The open ended question inviting comments on the Workers' Compensation Law changes yielded 144 responses. Responses were grouped by positive, negative, informational, and other. Positive comments (16%) included less legal and attorney involvement, easier to get patients back to work, and a better M-1 form. Negative comments (48%) included more paperwork, more interference from insurers, patients

having no choice of provider in the first ten days, and slow reimbursement. Informational comments (12%) included lack of information on Workers' Compensation Law changes. Other comments (24%) included seeing patients infrequently for work related injury/ illness or not practicing prior to 1993.

When asked under what circumstances they obtained an occupational/ environmental history, 43% of respondents indicated they took history on "most patients, but the level of detail varies", and 40% obtain them when they "suspect an occupational or environmental related illness". Ten percent rarely take an occupational/ environmental history, 5% after a diagnosis has been made, and 2% take the history if other etiologies are ruled out.

The respondents who reported taking occupational/ environmental histories on "most patients", also reported diagnosing the most cases of work related injury/ illness. The respondents who reported less frequent occupational/ environmental history taking also diagnosed fewer patients (p=.006). Occupational medicine specialists reporting taking the histories on "most patients" 87% of the time, internal medicine specialists 51%, family practice and chiropractors 42%, and surgical specialists 39% (p=.000).

The more often a provider saw work related injury/ illness, the more likely they were to include current and previous job information in the history they obtained. Figure 4 summarizes the frequency with which respondents typically include the key items in an occupational/ environmental history. On the average, the five individual history elements related to occupation (current exposure, past exposure, health and safety practices at worksite, description of current job and past jobs),were included more frequently than the five related to environment (hobbies/ home exposures, pesticides, water supply, air pollution, and home insulation/ heating system). (61% vs. 33%). Generally, providers treating more cases asked about occupational factors more frequently; and providers treating fewer cases asked about environmental factors more frequently. Chiropractors asked exposures less frequently than other provider types. Since they treat musculoskeletal injuries almost exclusively, this seems appropriate. Figure #4 summarizes frequency with which respondents indicated asking the individual items included in an occupational/ environmental history.

General barriers to reporting work related injury/ illness to the Maine Bureau of Health identified by respondents centered primarily on "already short on time, reporting is a low priority" (59%) and "ambiguous reporting criteria"(54%). Potential barriers to reporting are summarized in Figure #5. The respondents reporting "short on time " varied significantly across provider types; with 67% of MDs, 58% of DOs, 51% of PAs, 41% of NPs, and 37% of chiropractors reporting this barrier (p=.000). This difference may be due to the fewer types of reportable injuries (agriculturally related injuries and CTS) that chiropractors would normally diagnose as compared to the additional eight poisoning or respiratory conditions the other provider types would be likely to diagnose. Of the respondents who did not report any cases of work related injury/ illness to the Occupational Health Program, 18% indicated they felt the reporting system caused

problems for workers compared with 4% of those who reported at least one case (p=.045).

A total of 6877 reportable cases of work related injury/ illness were diagnosed by respondents in the past year. Of these, respondents indicated that 730 cases were reported to the Maine Occupational Disease Reporting Program, only 10.6% of the total. This may be an over estimate given the number of comments from providers indicating that they did not recognize the Bureau of Health and the Workers' Compensation System as separate reporting systems. Figure 6 compares diagnosis with reports for the 10 reportable conditions.

Providers who see the most patients also diagnose the most cases for certain diseases. This varied by provider type as well. These analysis were performed by T-test comparing the means of how often the respondent saw visits (once a week or less vs. more than once a week). Only respondents diagnosing at least one case of the disease were included in the analysis for this procedure. DOs seeing more than one work related injury/ illness case per week were more likely to diagnose carpal tunnel syndrome than those seeing one case a week or less (p=.005). Similar comparisons of MDs showed more diagnosing of conditions with increased number of visits for the conditions of carbon monoxide (p=.017), agricultural injuries (p=.018), and carpal tunnel syndrome (p=.0001). Nurse practitioners seeing more than one visit per week diagnosed more carpal tunnel syndrome than those seeing one case a week or less (p=.044). Physician assistants seeing more than one visit per week diagnosed more agricultural injuries than those seeing one case a week or less (p=.023).

The average reporting percentage across the ten reportable conditions by provider type 10.2% for physicians, 14.2% for physician assistants, 6.8% for chiropractors, and 17% for nurse practitioners. It was noted that although other reportable conditions were diagnosed, the only disease reported by nurse practitioners was carpel tunnel syndrome; without these reports their reporting average was 0%. This raises this question of how many providers have any information on their work related injury/ illness reporting responsibilities. Comparisons of number of cases diagnosed with number of cases reported are illustrated in Figures 6A & 6B.

In fact, only 15% of respondents reported any cases, making direct analysis of comparisons between reporters and non-reporters difficult. In comparing the differences of the means, those respondents diagnosing one or more cases of any disease, the ones who saw one or fewer cases/ week were more likely to report cases diagnosed than respondents who saw more than one case/ week.

When asked if they would be interested in technical assistance with treatment, diagnosis, or reporting, many providers indicated that many would like additional information. When the requests for assistance were distributed across provider groups the only statistically significant difference was that more physician extenders (NPs and PAs) requested assistance than physicians with treatment. This is illustrated in Table #2 below.

Requested assistance with	Physicians	Chiropractors	Nurse Practitioners	Physician Assistants	P=
Treatment	56%	61%	74%	78%	0.039
Diagnosis	63%	68%	74%	69%	0.579
Reporting	76%	86%	78%	83%	0.352
Other	6%	7%	9%	6%	0.922
N= 349	233	57	23	36	

Help with reporting was requested by 79% of respondents. Those diagnosing 8 or more cases in the past year (84%)were significantly more likely to request reporting assistance than those diagnosing less than 8 cases (70%), (p=.003). When the physician category was separated by MDs and DOs, significant differences (p=.036) in requests for reporting assistance was noted: 73% MDs, 78% nurse practitioners, 83% PAs, 86% chiropractors, and 92% DOs.

Sixty-five percent of respondents requested technical assistance with diagnosis with no significant differences noted across provider types. Eighty-two percent of providers responding requested a sample occupational/ environmental history form to use in there practice. Providing this sample history may be an important first step in improving the thoroughness of history taking.

Given the large numbers of providers expressing an interest in occupational/ environmental medicine information, it appears that ongoing education efforts are still needed for providers of all types. Based on the data gathered in this survey, the areas the areas of history taking, diagnosis, treatment, and reporting would be high priorities.

CONCLUSIONS

Based on the information collected from the survey questions and accompanying comments, it clear that:

1. There are multiple State reporting systems (Workers' Compensation, Occupational Disease) with different forms, creating great confusion.
2. Many providers do not know they have reporting responsibilities for occupational injury/ illness beyond Workers' Compensation.
3. There is no comprehensive mechanism in place to disseminate information on reporting requirements.
4. Occupationally related illness and injury are only reported about 10% of the time.
5. The reporting process is too time consuming for providers.
6. Those who do not report feel the system causes problems for workers

Implications: The current system does not meet the data collection needs of the Bureau of Health or the informational needs of providers treating injured and ill workers in the State of Maine.

RECOMMENDATIONS

1. Develop a comprehensive system to disseminate information to all providers mandated to report occupational injury/ illness to the State. It would make the most sense to include all public health, infectious disease, and State reporting requirements in an information package tied to State licensure (upon issue and renewal) through the appropriate bureau of licensure. (The Maine Institute for Occupational Health Education would be interested in working with the Bureau of Health and professional associations to develop these materials.)

2. Revise the reporting criteria so that they are understandable to the providers using them.

3. Develop a brief, simple, standard report form that could file with one State agency and the information disseminated to other agencies as required. This could ideally be filed by computer, just as requests for medical payments are.

4. Provide feedback to providers in the form of a report, newsletter, or fact sheet on a regular basis. This will not only remind them that the system exists, but provide information and establish credibility that the State is doing something with the data they provide.

5. Capitalize on the opportunity to provide technical assistance with treatment and reporting.

6. Provide a brief summary of this reports findings and a sample environmental exposure/ occupational history form with the mailing of the new criteria document.

OVERVIEW OF SIGNIFICANT FINDINGS

MAINE INSTITUTE FOR OCCUPATIONAL HEALTH EDUCATION
1996 SURVEY OF OCCUPATIONAL HEALTH PRACTICES

OVERVIEW OF SIGNIFICANT FINDINGS

FREQUENCY OF WORK RELATED INJURIES:

By type of provider:

78% of respondents see work related injury/ illness at least occasionally (1-4 times/ month). p=.002

49% of all respondents see work related injury/ illness at least 2-3 times/ wk.
64% of DOs see work related injury/ illness at least 2-3 times/ wk.
45% of MDs see work related injury/ illness at least 2-3 times/ wk.
63% of Chiropractors see work related injury/ illness at least 2-3 times/ wk.
27% of Nurse practitioners see work related injury/ illness at least 2-3 times/ wk.
59% of Physician assistants see work related injury/ illness at least 2-3 times/ wk. p=.002

Those seeing frequent work related cases:

Respondents who saw work related injury/ illness more than once a week were twice as likely to report that they "treat it themselves with occasional consultation"as those who saw less than one case per week. p=.000

78% of respondents who see more than one case a week, indicated that the 1993 WComp law changes made it easier for them to treat pts., compared 22% of those who saw 1 case/ wk or less. p=.000
58% of respondents who see more than one case a week include description of current job in Hx, compared with 41% of those who see less than one case a week. p=.001
62% of respondents who see more than one case a week include description of previous job in Hx, compared with 38% of those who see less than one case a week. p=.021
59% of respondents who see cases daily ask about previous jobs in Hx, compared to 38% who occasionally see cases. p=.001
31% of respondents who see one case or less a week ask about water supply in Hx, compared to 18% who see more than one case a week. p=.001
15% of respondents who see cases daily ask about water supply in Hx, compared to 38% who rarely see cases. p=.003

60% of respondents who see more than one case a week want help with reporting, compared with 40% of those who see less than one case a week. p=.025

REFERRALS:

61% of all respondents frequently treat pts. themselves with occasional consult p=.038

70% of respondents rarely or never refer to occupational medicine specialty as compared to 31% of respondents who refer to occupational medicine specialty at least sometimes. p=.012

HEARD OF MIOHE:

19% of DOs had heard of MIOHE
23% of MDs had heard of MIOHE
49% of Chiropractors had heard of MIOHE
28% of Nurse practitioners had heard of MIOHE
41% of PAs had heard of MIOHE p=.000

WORKERS' COMP CHANGES AFFECTING PRACTICE:

76% of chiropractors find it harder to treat patients p=.000

Seventy-eight percent of those responding that the changes made it "easier" to treat patients had diagnosed more than 8 cases of work related injury/ illness in the past year compared to those who diagnosed less than 8 cases. (p=.000) It appears that the changes in the WC laws made it easier for those who diagnosed the most cases.

ELEMENTS OF HISTORY:

The respondents who reported taking histories on most patients also reported diagnosing the most cases. p=.006

The respondents who reported less frequent env/occ history taking also diagnosed fewer cases. p=.006

87% of Occupational medicine specialist take env/occ Hx on "most patients" . The next closest specialty taking a history on "most patients"was internal medicine with 51%, general/ family practice was third with 42%. (p=.000)

Current Exposures:

73% of respondents ask about current exposures in Hx
82% of DOs and NPs ask
78% of PAs ask
75% of MDs ask
43% of chiropractors ask
p=.000

Past exposures:

55% of providers ask about past exposures in Hx
63% of DOs ask
37% of Chiropractors ask (p=.014)

Health and safety practices at work:
45% of all respondents ask about health and safety practices at work
60% of chiropractors ask
64% of PAs ask
38% of MDs ask
p=.001

Description of previous jobs:
45% of respondents include description of previous jobs in Hx.
21% of NPs ask
p=.034
60% of respondents diagnosing 16 or more cases of work related injury/ illness per year included description of previous jobs in env/ occ Hx compared with 41.6% of those diagnosing less than 16 cases/ year. p=.002

Air pollution, indoor and outdoor:
13.3% of chiropractors included air pollution in Hx, compared to the average all providers, of 25%. p=.021
(probably appropriate for what they diagnose)

Home insulation, heating and cooling systems:
29% of respondents include in Hx.
10% of chiropractors ask (probably appropriate)
p=.010

BARRIERS TO REPORTING:

59% of respondents report already short on time, reporting a low priority
67% of MDs reported this barrier
37% of chiropractors reported this barrier
p=.000
18% of respondents who did not report any cases believe that the reporting system causes problems for workers, compared to 4% of those who reported at least one case. p=.045

IF HELP AVAILABLE, MORE LIKELY TO REPORT:

58% of MDs would be more likely to report if help was available
77% of DOs, DCs, and PAs would be more likely to report if help was available
89% of Nurse practitioners would be more likely to report if help was available
(Do NPs know about the reporting laws?)
p=.000

REPORTING:

Only 15% of respondents reported any cases, making direct analysis of comparisons between reporters and non-reporters difficult

By Diagnosis:

Total cases diagnosed 6877, total reported 730, percent of total diagnosed reported 10.6%.

Of those respondents diagnosing one or more cases of any disease, the ones who saw one or fewer cases/week reported more of the cases diagnosed than those who saw more than one case/ week.

By Provider Type:

10.2% Physicians
6.8% Chiropractors
17.0% Nurse practitioners (This drops to 0% without CTS reports!)
14.2% Physician assistants

Tables by diagnosis , reporting, disease, and provider available.

TECHNICAL ASSISTANCE:

Help with treatment:

60% of respondents requested help with treatment
56% of physicians requested help with treatment
61% of chiropractors requested help with treatment
74% of Nurse practitioners requested help with treatment
78% of physician assistants requested help with treatment.
p=.039

Help with reporting:

79% of respondents requested help with reporting.
92% of the DOs requested help with reporting
86% of the chiropractors requested help with reporting.
p=.036

Those diagnosing 8 or more cases wanted more help with reporting than those diagnosing less than 8 cases. p=.003

Want sample env/occ Hx form:

82% of respondents wanted a sample form
96% of DOs wanted a sample form
96% of Nurse practitioners wanted a sample form
90% of chiropractors wanted a sample form
76% of MDs wanted sample form
p=.000

TABLES AND FIGURES

INDEX OF TABLES AND FIGURES

TABLE 1. History taking practices and referral patterns in clinicians who frequently see patients for occupational/environmental related illnesses or injuries as compared with those clinicians who report fewer office visits by patients with these complaints

	Number of clinicians who reported characteristic (%)	
Characteristic (number of respondents who answered "yes" to question)	Less than frequent office visits (one or less patient per week)	Frequent office visits (more than one patient per week)
History		
Obtains an environmental/occupational history on most patients (n-223)	89 (40%)	134 (60%)
In the history includes the following items:		
• Current exposures to chemical physical, biologic or radiologic hazards (n=335)	151 (45%)	184 (55%)
• Past exposure to chemical, physical, biologic or radiologic hazards (n=256)	113 (44%)	143 (56%)
• Description of current job (n=413) *	169 (41%)	244 (59%)
• Health and safety practices at worksite (n=209)	88 (42%)	121 (58%)
• Description of previous jobs (n=210) *	80 (38%)	130 (62%)
• Hobbies, home exposures (n=281)	124 (44%)	157 (56%)
• Pesticide exposure (n=94)	46 (49%)	48 (51%)
• Water supply (n=110) *	63 (57%)	47 (43%)
• Air pollution, indoor and outdoor (n=116)	53 (46%)	63 (54%)
• Home insulating, heating and cooling system (n=132)	67 (51%)	65 (49%)
Treatment		
• Frequently treat the condition themselves with occasional consultation (n=311) *	100 (32%)	211 (68%)
• Frequently refer to a medical/surgical subspecialty (n=11)	5 (45%)	6 (55%)
• Have found that changes in worker's comp rules and regulations make it easier to treat patients (n=69) *	15 (22%)	54 (78%)

* observed differences are statistically significant ($p<0.05$)

ACKNOWLEDGMENTS

The authors would like to recognize and thank the following for their contributions with question development, survey construction, and technical assistance.

Geoffrey Beckett PA-C
John Bielecki MD
Greg Bogdan PhD
Carol Eckert MD
Helen King-Atallah COHN
Al Leighton
William Mathes DC
Lisa Miller MPH
Roy Miller MD
Jay Saulter DC

Down East Association of Physician Assistants
Maine Academy of Family Physicians
Maine Chiropractic Association
Maine Medical Association
Maine Osteopathic Association

INDEX

OVERVIEW OF FREQUENCIES

Maine Institute for Occupational Health Education
1996 Survey of Occupational Health Practices
Overview of Frequencies
(n=599 out of 2307 mailed, 26% Response Rate)

Provider type:	Response Rate:	N=
71.3% Physician	(22.6% DOs; 26.4% MDs)	DO=60, MD=367
12.5% Chiropractor	34.6%	79
6.5% Nurse Practitioner	21.4%	35
9.7% Physician Assistant	26.4%	58

Question #1: How would you describe your practice?

- 26.9% General/ family practice
- 18.2% Internal medicine
- 18.1% Surgery
- 12.5% Chiropractic
- 8.0% Emergency Medicine/ Urgent care
- 6.2% OB/GYN
- 6.0% Other
- 4.0% Occupational Medicine

Of the surgery/other subspecialty responses 30% were orthopedic.

Question #2: How often do you estimate you see visits for work related injury/ illness in your practice?

- 26.7% Regularly (daily)
- 22.5% Frequently (2-3 times a week)
- 29.2% Occasionally (1-4 times a month)
- 9.9% Rarely (once every 3-4 months)
- 11.7% Never

Question #3: If I determine an injury/ illness is work related, I do the following:

Treat it myself with occasional consultation.

- 61.1% Frequently
  - 63% of DOs
  - 57% of MDs
  - 79% of Chiropractors
  - 56% of Nurse Practitioners
  - 59% of Physician Assistants
- 22.8% Fairly often
- 12.2% Sometimes
- 3.1% Not very often
- 0.8% Never

Refer to medical surgical specialty.

2.9% Frequently
5.5% Fairly often
47.9% Sometimes
38.0% Not very often
5.8% Never

Refer to occupational medicine specialty.

4.9% Frequently
5.4% Fairly often
19.9% Sometimes
31.6% Not very often
38.1% Never

Comments: 6, see appendix

Question #4: Check which of the following would be useful for providing you with occupational / environmental medicine information. (Check all that apply.)

25% Internet/ WEB Site
21% e-mail address
51% In-state FAX number
76% Resource list by mail

Comments: 7, see appendix

Question #5: The most useful method for providing information from the list above is:

9.9% Internet/ WEB site
5.7% e-mail
25.7% In-state FAX number
58.6% Resource list by mail

Comments: 3, see appendix

Question #6: Prior to this survey have you ever heard of or seen materials from The Maine Institute for Occupational Health Education?

28.3% Yes

Question #7: If you knew that by reporting occupational injury/ illness to a central data bank that on-line help, CME, conferences, and technical assistance were available to you, would you be more likely to report the cases?

67% Yes

Comments: 15, see appendix

Question #8: How have the 1993 changes in the Workers' Compensation Rules and Regulations affected your ability to manage occupationally related cases?

25.8% Harder to treat
13.9% Easier to treat
48.8% No difference
11.5% Not applicable

Question #8 Comments: 146, see appendix

Question #9: Check the statement that best describes the conditions under which you obtain exposure and occupational histories.

42.8% Most patients, level of detail varies.
40.3% If I suspect an env/ occ. related illness.
5.4% After Dx of env/occ related illness is made.
2.1% If other etiologies for illness are ruled out.
9.4% Rarely obtain env/ occ. history.

Comments: 1, see appendix

Question #10: Check the items that you typically include in your env/ occ history:

72.4% Current exposures to chemical, physical, biologic, or radiologic hazards
55.4% Past exposure to above
89.0% Description of current job, including typical work day (job tasks, location, materials, agents used)
45.0% Health and safety practices at worksite
45.5% Description of previous jobs.
60.6% Hobbies, home exposures
20.5% Pesticide exposure
23.7% Water supply
25.0% Air pollution, indoor and outdoor
28.4% Home insulating, heating and cooling system

Comments: 2, see appendix

Question #11: Please put a check next to the statements you agree with:

22.8% Reluctant to report if no state agency follow-up
18.5% Reporting system causes legal/ economic problems for employers
22.1% Reporting system causes legal/ economic problems for workers
59.5% Already short on time, reporting is a low priority.
12.9% Reporting system breaches doctor-patient confidentiality
53.5% Reporting criteria are ambiguous
18.7% Did not check any statements

Comments: 18, see appendix

Question #12: In the past twelve months, please estimate how many patients you have diagnosed with an occupational disease?.
Comments: 7, see appendix

Question #13: In the past twelve months, please estimate how many patients you have reported to the Maine Occupational Disease Reporting Program.
Comments: 19, see appendix

Condition	Total diagnoses	Total reported	
Lead and other heavy metals	54	9	
Carbon monoxide poisoning	141	10	
Acute pesticide poisoning	24	3	
Hypersensitivity pneumonitis	160	7	
Asbestosis	132	2	
Occupational asthma	471	21	
Mesothelioma	40	3	
Silicosis	16	0	
Agriculturally related injuries	766	112	
Carpal Tunnel Syndrome	5073	563	
TOTALS	6877	730	(10.6%)

Question #14: Would you be interested in receiving technical assistance from the State's Maine Occupational Health Program to help with:

60.2% Treatment
65.0% Diagnosis
78.5% Reporting
6.0% Other

Percent indicates Yes responses.
Comments: 17, see appendix

Question #15: Would you be interested in receiving a sample of an occupational and exposure history form to use in your practice?

82.1% Yes

Comments: 3, see appendix

Question #16: Which county is your practice located in: (Actual number of responses.)

44 Androscoggin
29 Aroostook
137 Cumberland
12 Franklin
20 Hancock
66 Kennebec
21 Knox
16 Lincoln
13 Oxford
61 Penobscot
11 Piscataquis
3 Sagadahoc
23 Somerset
13 Waldo
14 Washington
36 York

(Actual number of responses)

Figure 1. Providers reporting type of practice.

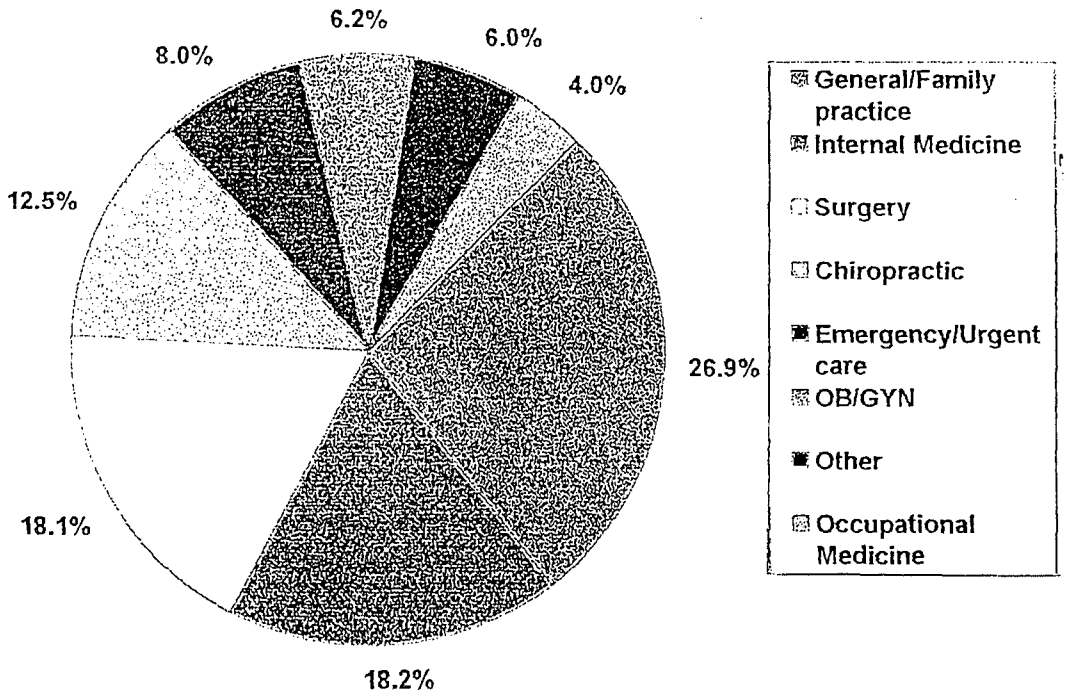


Figure 2. Percent of respondents reporting frequency of office visits for work related injury/illness (n=599)

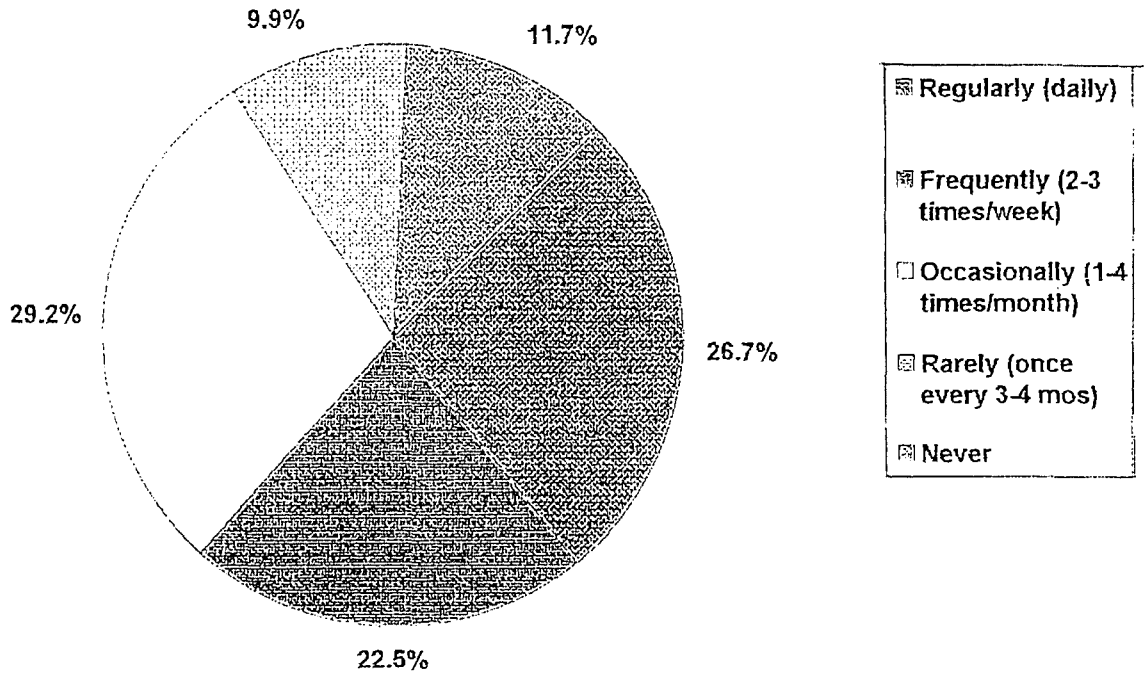


Figure 3. Preferred method of receiving information on occupational/environmental medicine, by percentage of respondents (n=599)

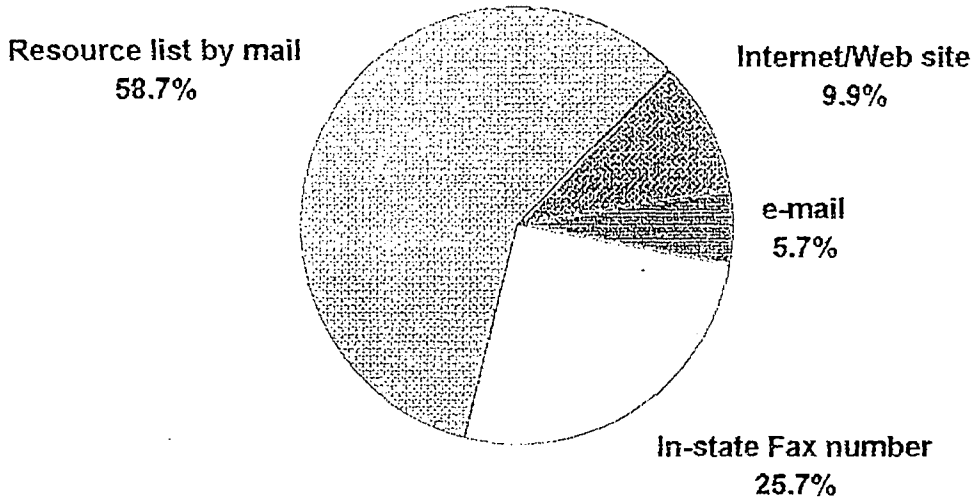
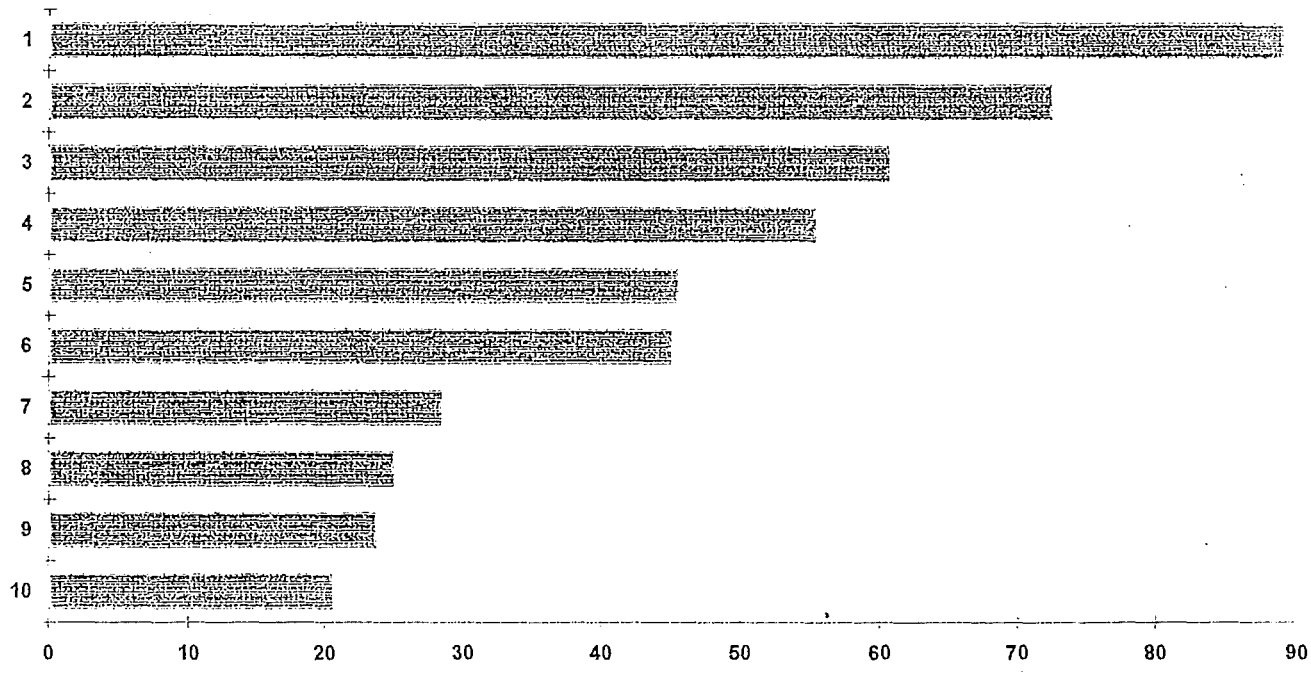


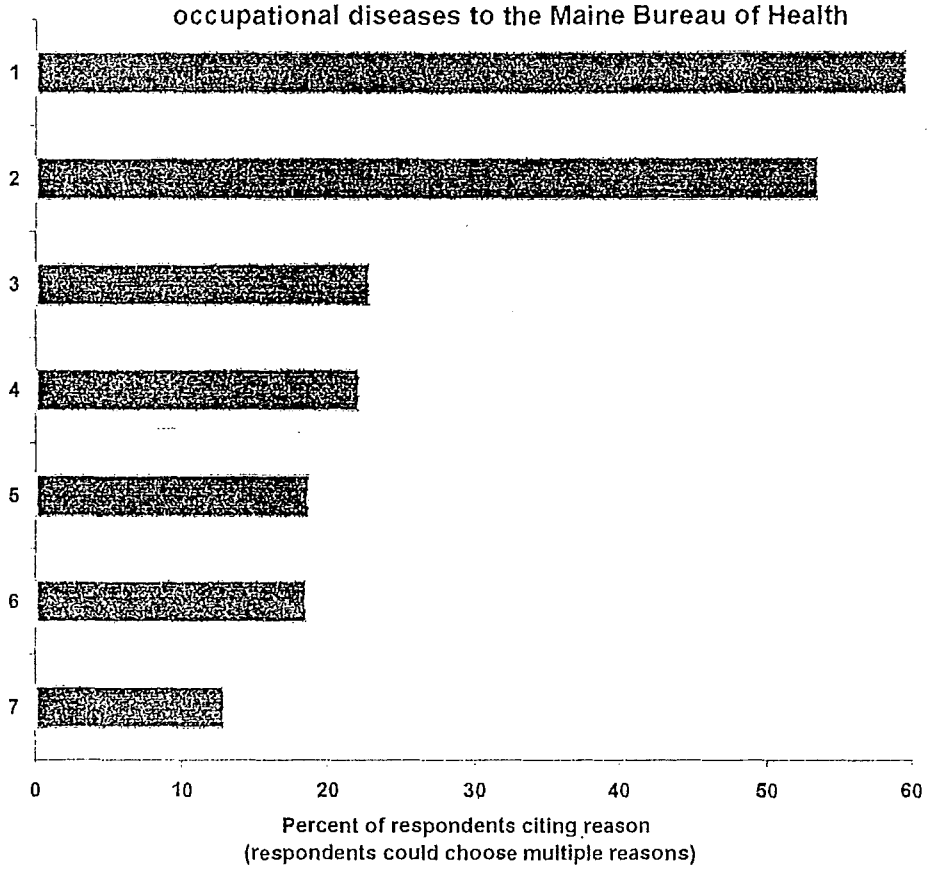
Figure 4. Items respondents typically include in an environmental/occupational history



KEY

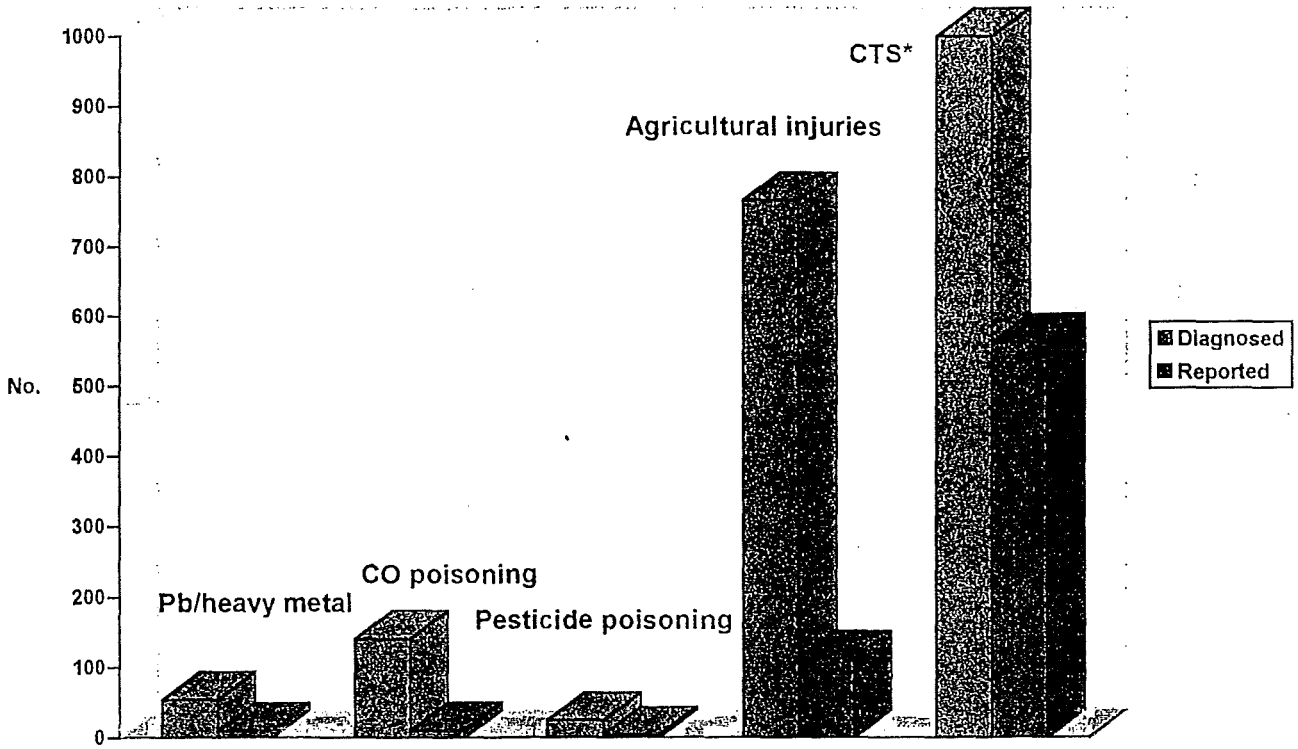
1 Description of current job, including typical work day (job tasks, location, materials, agents used)
2 Current exposures to chemical, physical, biologic, or radiologic hazards
3 Hobbies, home exposures
4 Past exposure to chemical, physical, biologic, or radiologic hazards
5 Description of previous jobs
6 Health and safety practices at worksite
7 Home insulating, heating and cooling system
8 Air pollution, indoor and outdoor
9 Water supply
10 Pesticide exposure

Figure 5. Potential barriers to clinicians for reporting occupational diseases to the Maine Bureau of Health



KEY
1 Already short on time, reporting is a low priority
2 Reporting criteria are ambiguous
3 Reluctant to report if no state agency follow-up
4 Reporting system causes legal/economic problems for workers
5 Did not check any statements
6 Reporting system causes legal/economic problems for employers
7 Reporting system breachers "doctor-patient" confidentiality

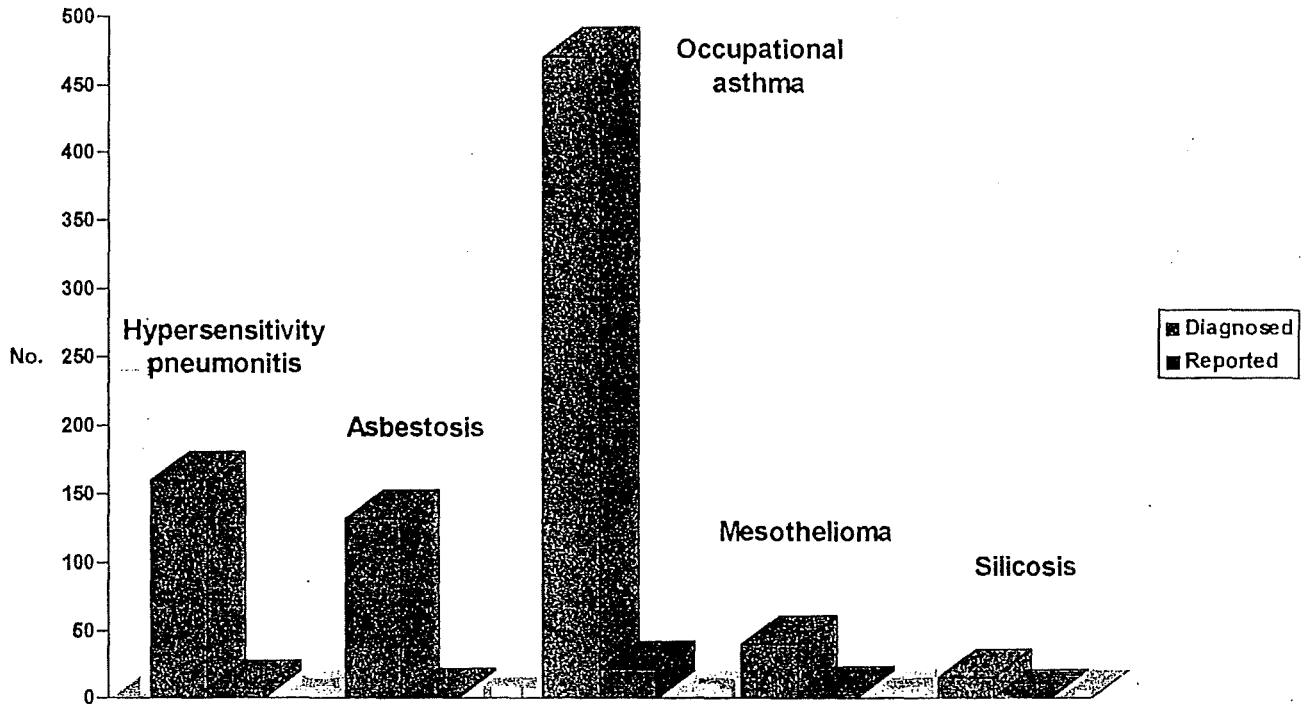
Figure 6a. Comparison of the number of injuries and poisonings diagnosed vs. the number reported to the Maine Bureau of Health (respondents self-reported)



Pb = lead; CO = carbon monoxide

* CTS = carpal tunnel syndrome (over 5000 cases were diagnosed)

Figure 6b. The number of pulmonary diseases diagnosed vs. the number reported to the Maine Bureau of Health
(respondents self-reported)



COMMENTS

COMMENTS FROM 1996 OCCUPATIONAL PRACTICES SURVEY

Comment Type Key
"o" = Other
"n" = Negative
"p" = Positive
"s" = Suggestion
"x" = Information

ID#	Q#		
1698	2	o	Less often in the past two years because of limited practice.
1575	3	o	Only see patients in consultation. They are sent to us.
1866	3	o	I am the consultant.
2107	3	o	Refer to occ med spec when available. ((Works multiple sites.))
1975	3	o	Usually, I am the surgical specialist the patient has been referred to.
1183	3	o	I am the specialist they refer pt. to
1833	3	o	I refer all work related disability to subspecialist if pt does not improve promptly. I do not want to be involve in paperwork and legal problems.
3033	4	o	A contact person at a library reference dept.
1836	4	o	Internet if I had a computer.
5105	4	o	Internet in future.
3022	4	o	I am not on the Internet!
2034	4	o	E-mail in future.
2091	4	o	Other - live person to talk with.

ID#	Q#		
1450	4	o	I don't have a FAX or computer.
2165	5	o	Most useful would be talking to someone.
2721	5	s	Library based lit. surveys with articles available?
1944	5	s	Phone number to call.
1316	6	o	What is this entity? OHES? NODRP? etc. I'm unfamiliar with organization.
2541	7	n	The info would be helpful, but not welcomed if used only as a "carrot".
3191	7	o	If system was secure.
1478	7	o	They are already reported by time I see them.
2393	7	o	Don't understand. Premium/ reward system?
2284	7	o	I report injuries identified as work related.
1391	7	o	Maybe but not if it added time to patient visit
1921	7	o	100% reporting to State of ME required now.
1061	7	o	It would depend on the value of these resourses vs. the problems in reporting.
3204	7	o	Report them anyway.
1583	7	o	Two separate issues.
4070	7	p	If we had on-line services available in my facility.
1396	7	s	Depends on content. Did not attend bad back seminar , might attend environmental exposure seminar.

ID#	Q#		
3031	7	x	The infromation would be helpful however. The occupational injuries I see are quite clear cut and are already reported to WC.((No reports #13))
4021	7	x	Do not have that kind of technical support.
2057	7	x	They are all reported on the M-1 already. ((13 DX, #13 no report))
1944	8	n	More paperwork.
2423	8	n	New changes have made it more difficult for the patient to receive both medical and legal help.
2721	8	n	One more set of rules of many.
1915	8	n	More paperwork.
3006	8	n	Patient access to legal opinion diminished. Some accounts that are WC and challenged are harder to receive payments on.
2107	8	n	Paperwork is complicated and often not available at treatment of Followup visits. Many diff Docs providing coverage of ER, used as walk-in clinic.
3022	8	n	In spite of research providing chiropractic is cost effective, employers continue to refer employees elsewhere.
1956	8	n	I have Occupational Health see most of my work related injuries as I find the system too flawed and self serving.
3021	8	n	More paperwork, more intervention.
1004	8	n	More paperwork
3000	8	n	More paperwork, difficulty communicating with employers and WC ins co. Workers do not follow through with proper TX, due to job loss risks or financial pressure.
2144	8	n	Reimbursement levels do not meet my costs of providing care or reports.
1558	8	n	Harder, due to increased paperwork.

ID#	Q#		
1036	8	n	Lower income
1159	8	n	The more reporting the harder it is to treat workers who are injured
1172	8	n	Patient care is now limited by insurers not employers
1173	8	n	System is now employer and insurance company protective
1180	8	n	Excessive paperwork/ forms often generated due to reports/ forms that would not be necessary most of the time
1357	8	n	The new form is too difficult-it's like the Post Office form which is an ED doc's nightmare
1373	8	n	A lot of pt. don't get the care since they are denied and don't have "easy" access to legal help. Most if not all have a very hard time from insurance carrier and employer.
1385	8	n	Too much paperwork
1391	8	n	Pain in rear form. Appears to be of limited utility.
1430	8	n	The present system is demeaning and contributes substantially to preventing workers from returning to work.
1833	8	n	Recently I ordered a CT scan of back. The insurance co. would not give permission for 2 mos. The pt had to wait 2 mos for a neurosurg. opinion.
1546	8	n	Unnecessary interference by case management personnel has delayed treatment of patients.
1913	8	n	Forms difficult to fill out sometimes.
1584	8	n	Insurance is so involved that managing care can be difficult because the insurance thinks they are the manager of care.
1658	8	n	More interference from assigned case managers, more inquieies from them
1665	8	n	Reimbursement for my work is too slow. I hate legal hassles.

ID#	Q#		
1669	8	n	New M-1 form not better.
1676	8	n	"Occupational Health" groups fiddle with problems they don't adequately know how to treat and refer often too late, so the problem is a bigger deal by the time I get it.
1682	8	n	More paperwork!!
1709	8	n	Workers' Comp laws are terrible! For example, I can't exclude someone from getting hired for construction work with a history of back problems, yet if their back hurts once employed, it's the employer's fault.
1716	8	n	There is a tendency for insurers to reject claims that are meritorious.
1775	8	n	The report forms is poorly designed.
1801	8	n	Harder since I'm still between pt and WC.
3033	8	n	Too much interference from employers.
1887	8	n	Many individuals with the worst injury and worst socioeconomic factors simply don't know their rights. Too much contraverting.
1468	8	n	Employer selected physicians make the pt-primary care relationship more difficult and confuse care for individual episodes. Either have all care done by one or the other.
5022	8	n	More waiting time to treat pts if case contraverted.
3084	8	n	Often DCs require referral or pt must go through company Drs; MDs are reluctant to send to DCs even with evidence of efficacy.
3195	8	n	Restricted pt access to chiro care inability to treat acute injuries within first 10 days.
3076	8	n	Pts. are intimidated by employers and ins. co., afraid to lose their jobs and get treatment. Often times they are not treated properly and cannot return to work and end up on Medicaid.

ID#	Q#		
3209	8	n	Many times ins. co's create more problems than they solve. Often pts who are MMI are referred for IME or denied further care. This raises costs, creates neg psychological impact. BAD NEWS FOR ALL.
3177	8	n	Workers aren't referred to DCs as frequently as they should be, Workers aren't aware they can choose DC after 10 days with MD.
3160	8	n	The additional paperwork is substantial. The patients are more confused and upset with running back to employer for permission etc.
5097	8	n	I don't like the WC form. ((No reports))
3086	8	n	Employers have forced pts. to seek other medical intervention early in the course of their treatment.
3113	8	n	Has increased the frustration of treating WC pts. Feel as though my office gets negative comments from claims adjusters.
3111	8	n	Many people refuse to go to designated 'sports clinic' and refuse to use the system due to prejudice against DCs Pts pay for own care, injuries not reported, when they do get DC care it 3-4 mos later.
3191	8	n	Some employers/ supervisors interfer with pt treatment, sending to personal Dr., and making Dx's etc.
3093	8	n	Restricted care has made the small percentage of difficult cases impossible to manage.
3074	8	n	Pts. sometimes have to wait 10 days before they see me.(Employer requests another provider initially.)
3071	8	n	Patients that would prefer being treated by a chiropractor are often told by their employers that they must treat with an MD or pay for chiro care themselves.
5004	8	n	There is still a loophole in the process. Ijury report form should require medical comment on fitness to work., employers acceptancr/ rejection and reason for, then be sent to WC for review and file.
3051	8	n	Pts may be directed to providers with the best marketing plan not the best treatment plan.
3052	8	n	Pts are less honest about work-related injuries, they ask that injury not be reported to employer because do not want company doc and are fearful of losing job.

ID#	Q#		
3098	8	n	Too much regulation.
5144	8	n	Too much paperwork!
3046	8	n	Pts are required to visit provider rec by employer and this is not in many cases inthe best interest of patient care, pt wish for Tx, or cost effective.
3059	8	n	Increased paperwork. Decreased referrals.
3043	8	n	Pts not able to have direct access to chiro care and limited visits. Also if pt wishes chiro care after 10 day wait, they are fearful of problems with employer.
3061	8	n	The "10 Day Rule" has prevented people from seeking chiropractic care.
3062	8	n	The first 2 weeks after an injury are the most important in its mgm't. If the individual is not directed to us in the first 10 days, they may not get off to an optimum start.
5134	8	n	Paperwork takes too long and is very repititious.
3036	8	n	Sometimes I don't get paid for treating injuries.
3090	8	n	Pts often want to come here right away, if their employer doesn't want them to, they have to wait 10 days, meanwhile the pts. condition worsens.
5019	8	n	More paperwork, more time.
4173	8	n	More paperwork to do.
1370	8	o	Almost all may cases are either noise related hearuing loss or facial injuries, fairly straight forward
1943	8	o	Guidelines for disability in pregnancy?
1946	8	o	Did not handles WC injuries before 1993.

ID#	Q#		
2034	8	o	Cases requiring longitudinal care are the most difficult and in the ER initial mgm't. 7 referral has not changed greatly. Referral back to PCP most frequent outcome.
4055	8	o	Was not in primary care in 1993.
1917	8	o	I have not been involved with a WC case directly.
1411	8	o	For some reason I seem to be outside the referral loop, despite a fundamental interest.
1864	8	o	No major difference noted.
4003	8	o	I am not well educated in occ health issues, but my clinic may be increasing its involvement in WC and preemp phys. in the future.
5040	8	o	I had to get used to doing the M-1 form.
1780	8	o	It's harder to get a WC determination, but easier to decide what is WC related. Seeing fewer people claiming WC injuries.
1974	8	o	I more frequently refer to occupational management at Franklin Memorial Hospital.
1704	8	o	I only see Workers' Comp cases if pre-approved payment from Workers' Comp. Generally this is consultation only
4070	8	o	Not practicing in Maine prior to 1995.
1685	8	o	Went into practice after 1993.
5036	8	o	I've only been in the civilian world since 1994.
4104	8	o	Just started this kind of practice this year (Occ Med)
4117	8	o	Did not practice before 1993.
1502	8	o	Don't know - most of work related injuries I see are referred to my hyperbaric medicine practice. In OB, significant problems are referred to orthopedic specialist.

ID#	Q#		
1518	8	o	Hernias are about only IA treated.
2091	8	o	Probably see fewer pts. because they are sent to company doc- doesn't affect those I do see.
5082	8	o	Did not practice before 1993.
2562	8	o	I try to refer to Occ.Health.
2448	8	o	I make out the M-1 form now. Occasionally refer, but often leave F/U prn, unless it definitly needs F/U.
2438	8	o	Don't see enough pts. who file WC to see a difference. Work for Univ. of ME all WC cases referred out.
1219	8	o	Not in state before 1993
2345	8	o	I've only been practicing since 1993.
2707	8	o	System already in place.
2722	8	o	I refer ".." to Workplace Health at MMMC.
2205	8	o	Form requirement does often cause me to effectively "upcode" a visit.
2150	8	o	N/App, work for Dept of Defense at Brunswick Naval Air Station
3025	8	o	Open practice in Oct. 1993.
1349	8	o	Virtually the only injury I see is hearing loss noise induced
1352	8	o	Practice just started - I may not have noticed a difference
1379	8	p	New forms are a great improvement.
1779	8	p	Paperwork reduced, but some employers still require too much paperwork.

ID#	Q#		
3074	8	p	It took time, but now I see M-1's are a good idea, communication is better.
1431	8	p	Better exchange of information has been a direct result.
1694	8	p	As a general rule the occupational injuries I see are hernias, which are covered by Workers' Comp.
2635	8	p	There are better incentives to get better.
2157	8	p	I find employers more amenable to light duty, although there is still some ignorance on the frontline of immediate supervisors.
3077	8	p	I feel changes are fair and practical but can sometimes penalize those with true chronic conditions resultant from occupational injury.
1874	8	p	The M-1's specificity and duplicity is helpful.
3019	8	p	I work with several companies as a preferred provider and I find it to be a very useful system.
1719	8	p	Ten day rule is a godsend to get patients back to work.
1091	8	p	Less attorney involvement which results in less coaching that turns employment issues into medical issues.
1591	8	p	Fewer depositions; dismayed that attorneys seen to expect a contingency % reward from pt disability awards.
1801	8	p	Easier since pt is made more in charge.
1583	8	p	Patients not free to direct their care initially, some delays before they reach specialty care.
3192	8	p	Decrease WC injury since 1993 because of new law
3116	8	p	Ultimately the 10 day rule has facilitated appropriate treatment of cases referred to me by employers and MDs, but it required working to estb. those patterns of referral.
1394	8	p	Maybe slightly easier in that patients are less able to "doctor shop"

ID#	Q#		
1921	8	p	More light duty programs available.
1398	8	p	The lawyers are generally out of the loop, and not encouraging patient to remain ill.
2239	8	p	It is very useful from an Emergency Medicine perspective to have a referral source.
1332	8	p	Less depositions
1591	8	p	M1 takes time, less adversarial
1836	8	s	Move "commissioners", quicker decisions. Diminish power of adjusters.
1175	8	s	The company Dr. has a vested interest in the co.The fam.Dr. has a vested interest in the pt. IME's are no good because they are being paid for a friendly opinion. The FP is the best way to deal with
5007	8	x	Not even aware of it.
1145	8	x	Don't know what the changes are
4059	8	x	Don't know much about the act.
2549	8	x	I didn't notice any change.
1814	8	x	Not very clear on what the "93 changes were relative to all the other changes we've seen.
2677	8	x	I am not sure as I do not have a copy of that reference.
2327	8	x	Don't know.
4042	8	x	Not sure what the changes are.
5049	8	x	Frankly, I'm not sure what these changes are.
1425	8	x	No known change

ID#	Q#		
1356	8	x	I am unaware of '93 changes.
5078	8	x	I'm not sure , I usually ignoreWC Board.
1335	8	x	Not aware of changes
5096	8	x	Unaware of the WC changes.
1386	8	x	Not clearly aware of changes.
1724	8	x	I don't know these rules.
2169	8	x	I need information about the changes.
1399	8	x	Don't know
1528	9	o	I'm trying to get better.
1833	10	o	I do what is appropriate. (Checked 2 out of 10 Hx components)
5007	10	x	Water supply sig. prob locally ((Waldo))
1833	11	n	Too much government.
1502	11	n	Another reason to get hauled into court and deal with lawyers.
1502	11	n	We already have toomuch paperwork.
2205	11	o	Criteria for determining if disease is really work related are worse.
5007	11	o	Have received little guidance.
2722	11	o	Ambiguous in terms of who does the reporting.

ID#	Q#		
3116	11	o	None
1399	11	o	Don't know.
5105	11	o	I am willing to improve this. I believe it is important but forget to do it.
1690	11	o	I just don't do it.
5099	11	o	None
1478	11	o	No need to report at my level. (Thoracic surgeon)
1704	11	p	But this is not a reason not to have a system. (Problem for employers.)
5049	11	x	Maybe I just don't know.
1801	11	x	I didn't know there was a list?!
2150	11	x	DOD and OSHA here at base does reporting.
1555	11	x	I didn't realize there was a reporting system.
5206	11	x	I see too many to report them all.
1911	12	o	consult.
2265	12	o	Most, greater than 95%, of work related visits to ER are for musculoskeletal injuries.
3022	12	o	Pts. usually come already diagnosed by company doctor.
5007	12	o	2 Dx Arsenic ((Waldo))
2245	12	o	High lead levels in pediatric population. ((No reports))

ID#	Q#		
1613	12	o	EMG testing upon referral.
3113	12	x	Silicosis not an occupational disease.
1913	13	n	I tell pts. to claim comp if they feel its in their interest. Many do not claim comp due to worries about discrimination.
2393	13	o	Recently reorganized our system.
1114	13	o	Seen by another physician & reported
1114	13	o	Usually seen by primary care before I get them
1940	13	o	None reported since nov 1994! Will remedy!
2722	13	x	Reported by personnel dept.
2448	13	x	Except M-1 Form. ((No reports))
2707	13	x	Referred to workplace Health Services, not reported directly.
2157	13	x	We do a fair amount of WC care in the ER. I am reluctant to add furthur paperwork/ reporting responsibilities to my existing work load.
2150	13	x	I report it to Occupational Medicine here at Brunswick Air Station.
2084	13	x	I file WC encounter forms though. ((9 DX, no reports))
3084	13	x	Pts from #12 these have been reported by 1st treating Drs.
1682	13	x	Reported via Workers' Comp.
1575	13	x	All seen in consultation.
5073	13	x	Most, I hope , done automatically by diagnosis.

ID#	Q#		
5045	13	x	All work related injuries have occupational health forms filed.
1911	13	x	Under care of other provider.
3033	13	x	Most of these were work injuries and reported to WC. ((10 DX, No reports #13))
4013	13	x	I file a form relative to each individual worksite.
1502	14	o	Something simple.
2721	14	o	Education re: latest way to approach CTS.
1613	14	o	Literature
2562	14	o	CME would be helpful to entire staff.
1450	14	o	Don't see that much
3113	14	o	deal with musculoskeletal injuries only
1391	14	o	We have occupational health program and staff locally.
1398	14	o	Statewide stats
3116	14	o	Evolving information systems and forming vertical networks.
2144	14	o	Coding for fair reimbursement.
1534	14	o	What kind of technical assistance?
1296	14	o	All of my occupational disease/ injury pts. are referred to the Occ.Health Clinic in Farmington
1061	14	s	Information on the ramifications of this reporting to all concerned and the process by which it happens- after reporting.

ID#	Q#		
4133	14	s	Standardized BRIEF forms.
5099	14	s	Education on how affects my practice as PA-C. Thank you.
5049	14	x	I would like to know how to obtain this help when I need it and reporting guidelines.
2058	14	x	Please contact the VA, not much call for this.
5000	15	o	We refer to CHP most times.
2677	15	p	Great idea.
1801	15	s	It should be provided to every MD who starts a practice in Maine. Not just occupational , but all public health laws.
2448	17	x	If you get poor return %, its probably because you did not stamp envelope. If you want someone's helpin questionnaire, you could at least pay for stamps!!
2491	17	x	This form does not address the needs of my specialty.

SURVEY

MAINE INSTITUTE FOR OCCUPATIONAL HEALTH EDUCATION
SURVEY OF OCCUPATIONAL HEALTH PRACTICES

Complete the following questions by checking the blanks next to the appropriate answer(s).
Please return in the enclosed return envelope by **February 29th.**

OFFICE USE ONLY
(1-4)__ __ __ __

1. How would you describe your practice? (Check one.)
   - ___ General/ Family Practice
   - ___ OB/GYN
   - ___ Emergency Medicine/ Urgent Care
   - ___ Occupational Medicine
   - ___ Internal Medicine
     Subspecialty _____
   - ___ Surgery
     Subspecialty _____
   - ___ Chiropractic
   - ___ Other _____

(5) 1 0
2
3
4
5
6
7
8

(6-7) __ __

2. How often would you estimate you see visits for work related injury and illness in your practice?
   - ___ Never
   - ___ Rarely (once every 3-4 months)
   - ___ Occasionally (1-4 times a month)
   - ___ Frequently (2-3 times a week)
   - ___ Regularly (daily)

(8) 1 0
2
3
4
5

If you answered "NEVER"; Stop here and return survey. Thank you.

3. If I determine an injury /illness is work related, I do the following:

	Frequently		Sometimes		Never	
Treat it myself with occasional consultation	5	4	3	2	1	(9) 5 4 3 2 1 0
Refer to medical/ surgical specialty	5	4	3	2	1	(10) 5 4 3 2 1 0
Refer to occupational medicine specialty	5	4	3	2	1	(11) 5 4 3 2 1 0

4. Check which of the following would be useful for providing you with occupational/ environmental medicine information.
   - ___ Internet/WEB site (12) 1 5 0
     (Bibliographies, resources, educational events, question and answer forum, contact people)
   - ___ E-Mail address for requesting such information (13) 1 5 0
   - ___ In-State FAX number for requesting such information (14) 1 5 0
   - ___ Resource list by mail (15) 1 5 0

5. Circle the MOST useful method for providing information in the list above. (16) __

6. Prior to this survey, have you ever heard of or seen materials from the Maine Institute for Occupational Health Education? (17) 1 5 0
   - ___ Yes
   - ___ No

7. If you knew by reporting occupational injury/ illness to a central data bank that on-line help, CME, conferences, and technical assistance were available to you, would you be more likely to report the cases? (18) 1 5 0
   - ___ Yes
   - ___ No

8. How have the 1993 changes in the Workers' Compensation Rules and Regulations affected your ability to manage occupationally related cases:
   - ___ Harder to treat patients (19) 1 0
   - ___ Easier to treat patients 2
   - ___ No difference 3
   - ___ Not applicable 4

Comments welcome: _____ (20) 1 5
_____
_____

9. Check the statement that best describes the conditions under which you obtain exposure and occupational histories:
   - ___ I obtain them on most patients, but the level of detail varies. (21) 1 0
   - ___ I obtain them on patients that I suspect may have environmental or occupationally related illness. 2
   - ___ I obtain them after a diagnosis of a known environmental or occupational related illness is made. 3
   - ___ I obtain them if other etiologies for an illness are ruled out. 4
   - ___ I rarely obtain an environmental exposure and occupational history. (Skip to Question #11) 5

-OVER-

ATTACHMENT F

Summary of Maine Law Review, Volume 34, No 1.1982

"What is Wrong With Maine's Occupational Disease Law"

This paper, written by Thomas R. Watson, Esquire takes us through the historical development of workers' compensation, the development of occupational disease coverage in Europe and the United States, the legislative history of Maine's Occupational Disease Law, which originated in 1937 and finally to recommendations for reform of the Occupational Disease Law.

ATTACHMENT F

MAINE LAW REVIEW

Volume 34, Number 1, 1982

What is Wrong with Maine's Occupational Disease Law?

Thomas R. Watson

WHAT IS WRONG WITH MAINE'S OCCUPATIONAL DISEASE LAW?

I. The Failure of Occupational Disease Coverage in Workers' Compensation Systems

By enacting occupational disease laws, state legislatures stepped ahead of limited medical knowledge concerning the etiology of disease. Responding to public and political pressures, legislatures placed the responsibility for adjudicating claims based on disease on administrators whose procedural and evidentiary systems were designed for claims due to injuries. But the causal connection of disease to employment is not so easily shown; until quite recently, diseases were not thought to support the common law causes of action that gave rise to the *quid pro quo* of workers' compensation systems.[1]

Some diseases, however, are easy to equate with a compensable injury; dermititis, for example, is often caused by contact with paint solvents. Once the substance is identified and the causal link to the employment proved, the compensation award is justifiable. Other diseases, however, are more etiologically problematic for the administrative fact finder to fit within the bounds of compensation law. Due in part to the restrictions in occupational disease laws, which were legislated in the era of fears of "insuring a burning house,"[2] and to the difficulties of etiology, most occupational disease claims today remain uncompensated.

According to national studies, occupational diseases may account for over 100,000 deaths a year.[3] The World Health Organization estimates that more than seventy-five percent of human cancers are caused or aggravated by exposures to environmental factors including stress, chemicals, and occupational hazards.[4] Yet one in every five individuals severely disabled due to an industrial disease receives no disability or income maintenance benefits.[5] For those who do receive income support, social security and welfare provide almost seventy percent of benefits received, or about $2.2 billion annually.[6] Workers' compensation benefits provide only about five percent of lost income from chronic occupational disease.[7] That figure

---

1. See text accompanying notes 59-64 *infra*.
2. See text accompanying notes 111 & 112 *infra*.
3. National Institute for Occupational Safety and Health, President's Report on Occupational Safety and Health 111, 128, Table 5-1 (1972).
4. American Lung Association, Occupational Lung Diseases, An Introduction 3 (1979).
5. Edes, *Compensation for Occupational Diseases*, 31 Lab. L.J. 595, 596 (1980). This article was adapted from a statement made before the U.S. Senate Committee on Labor and Human Resources, Aug. 26, 1980.
6. *Id.* at 597.
7. *Id.*

compares with almost sixty percent of lost income replaced by workers' compensation in cases of industrial injury victims.[8]

The disparity between compensation for injuries suffered on the job and compensation for occupational diseases does not end with a comparison of benefit levels. A recent study by Dr. Peter Barth[9] indicates that the workers' compensation system is not equipped administratively to deal with occupational disease claims litigation. Although the average work injury victim can often expect benefits within two months of disability, the occupational disease victim waits an average of one year.[10] Of those occupational disease sufferers awarded benefits, sixty percent face initial denial of their claims, compared with only ten percent of those bringing injury claims.[11] Dr. Barth's study also indicates that over half of the occupational disease awards nationwide are based on compromise and release agreements, which involve small lump-sum settlements. Only about sixteen percent of all injury claims are treated in this manner.[12] Thus, the disease victim who chooses to file a claim under the existing compensation laws faces the prospects of long delay, protracted and costly litigation, and a relatively small financial award. Clearly, in the *quid pro quo* of workers' compensation, which exchanged tort actions for speedy and certain remedies, the occupational disease sufferer does not share the benefit of the bargain.

Although it is impossible to gauge the effectiveness of Maine's law in achieving the goals of providing income maintenance and medical care to occupational disease victims,[13] some estimates can be made. According to one Maine Workers' Compensation Commissioner, the number of disease cases reaching adjudication is "less than one percent" of the total caseload;[14] another Commissioner estimates the number to be less than five percent.[15] The Commissioners interviewed perceived that the law is simply too restrictive and the issues

---

8. *Id.* at 596.

9. P. BARTH & H. HUNT, WORKERS' COMPENSATION AND WORK-RELATED ILLNESSES AND DISEASES (1980).

10. *Id.* at 135-87.

11. *Id.*

12. Statistical data similar to that in Dr. Barth's study are not available for Maine; consequently, no comparison is possible. Although the Workers' Compensation Commission is mandated to record and compile such data, the budget has never allowed for the task. ME. REV. STAT. ANN. tit. 39, § 108 (Supp. 1981-1982). *See, e.g.*, INDUSTRIAL ACCIDENT COMMISSION, REPORT OF THE INDUSTRIAL ACCIDENT COMMISSION FOR THE BIENNIAL PERIOD ENDING DEC. 31, 1960, at 1 (1961) (noting failure to comply with statistical report due to denial of budget request) (on file at State Law Library, Augusta, Maine)[hereinafter cited as INDUSTRIAL ACCIDENT COMMISSION].

13. *See* note 12 *supra*.

14. Telephonic interview with David Pomeroy, Workers' Compensation Commissioner (Feb. 23, 1981).

15. Telephonic interview with Ronald Russell, Workers' Compensation Commissioner (Feb. 19, 1981).

made too complex by problems of proof. As a result, if there is a possibility of a case being made under "gradual injury" theories,[16] rather than "disease" theory, the claimant is likely to drop his occupational disease claim and proceed under the more liberal Workers' Compensation Act.

This Comment first sketches the development of Workers' Compensation law and its extension of coverage to occupational disease. Following a brief legislative history of Maine's occupational disease law and a discussion of the law's application by the Law Court, this Comment demonstrates how and why the law fails to compensate adequately victims of industrial disease for disabilities related to employment.

II. Historical Development of Workers' Compensation

A. *The Common Law Underpinnings of Workers' Compensation*

The advent of industrialization necessitated a redefinition of the common law liability of an employer to an employee injured in the course of employment. The prevailing theory of limited liability rested on the economic maxim that the vast supply of work then available and the fluid mobility of labor meant that workers were free agents under no compulsion to enter into employment.[17] The employer was liable in tort only for a failure to exercise due care in providing for the employee's safety. This narrow duty of care was limited to specific requirements,[18] such as the duty to provide the following: 1) a safe workplace, 2) safe appliances and tools, 3) adequate warnings of dangers of which the employee may not reasonably be aware, 4) a sufficient number of co-workers, and 5) proper rules for the safe conduct of the work.

As a result of this narrow scope of employer liability and despite the lack of any real choice on the employee's part in entering the employment, the common law provided no remedy for an injury arising from the dangers normally incident to the employment. Further, when an injury was shown to have resulted from a breach of the employer's narrow duty of care, thus allowing a common law cause of action, the employee was faced with the "unholy trinity"[19] of employer defenses: contributory negligence, assumption of risk, and the fellow-servant rule.[20] The system worked effectively to shift

16. *See, e.g.*, Ross v. Oxford Paper Co., 363 A.2d 712 (Me. 1976) (finding compensable injury developing gradually over number of years from repetitive trauma to paper worker's hands).

17. W. Prosser, Handbook of the Law of Torts § 80, at 526 (4th ed. 1971).

18. *See* cases collected *id.* at 526 n.95, 527 n.96.

19. *Id.* at 526-27. The defenses were also known as the three "wicked step-sisters" of the common law.

20. Contributory negligence worked to bar plaintiff's recovery on the theory that defendant's negligent acts were not the proximate cause of the injury. The defense

the burden of a work-related injury from the employer to the injured worker, the party least able to bear the loss.[21] As a result, the great proportion of industrial injuries were not compensated under the common law in industrialized Europe and the United States.[22]

B. *Legislative Action Removes Industrial Injuries from Common Law*

The enactment of Germany's Workers' Compensation Act in 1884 was the first statutory attempt to shift the burden of workplace injuries to the employer.[23] Great Britain's Act followed in 1897[24] and by 1908 most of Europe had enacted similar legislation.[25] These acts represented a legislative recognition that industrial injuries should be treated as a cost of doing business; "[t]he cost of the product should bear the blood of the workman."[26]

The result of this legislation is the present system of no-fault compensation. The employer, usually through an insurance carrier, compensates an employee for injuries arising out of the business, without considering the negligence of either party. The compensation acts generally abolished the three common law defenses available to the employer;[27] the only issues that remained concerned whether the employee and the injury were covered by the statute and what amount of compensation was necessary.[28] As a *quid pro quo* for this more certain and speedy remedy for work-related injury, the employee forfeited his right to sue at common law and accepted a lower monetary award based solely upon wage loss, with no

---

thus revoked a worker's right to sue and the common law prohibited further investigation of the employee's relative fault. *See* Bohlen, *Contributory Negligence*, 21 HARV. L. REV. 233, 233 (1908).

The rule that an employer was not liable for injuries due to a fellow worker's negligence was first stated in an 1837 British case. Priestly v. Fowler, 150 Eng. Rep. 1030 (Ex. 1837). The rule was adopted in the United States shortly thereafter. *See* Farwell v. Boston & Worcester R.R., 45 Mass. (4 Met.) 49 (1842); W. PROSSER, *supra* note 17, § 80, at 528.

21. Mitchell, *Products Liability, Workmen's Compensation and the Industrial Accident*, 14 DUQ. L. REV. 349, 351 (1976).

22. W. PROSSER, *supra* note 17, § 80, at 530 n.32.

23. W. MALONE, M. PLANT, & J. LITTLE, THE EMPLOYMENT RELATION: CASES AND MATERIALS 35 (1974).

24. British Workmen's Compensation Act of 1897, 60 & 61 Vict., c. 37, §§ 1-10.

25. W. MALONE, M. PLANT, & J. LITTLE, *supra* note 23, at 35.

26. W. PROSSER, *supra* note 17, § 80, at 530. *See also* Bohlen, *A Problem in the Drafting of Workmen's Compensation Acts* (pts. 1-3), 25 HARV. L. REV. 328, 401, 517 (1912).

27. See notes 19 & 20 and accompanying text *supra*.

28. W. PROSSER, *supra* note 17, § 80, at 531. A ceiling was usually placed on the amount recoverable by fixing the recovery at a percentage of the jurisdiction's "average weekly wage" or some such figure. This amount was considered less than a potential jury award. Thus, the acts sought to compensate the injured employee for only a portion of the wages lost due to the injury and not for the injury itself.

compensation for pain and suffering. A provision that compensation under the act was the employee's exclusive remedy was generally included in the European acts and is present in all of the state compensation statutes.[29]

C. *Basic Features of Workers' Compensation*

The typical workers' compensation act combines the following features:[30]

1) the employee is entitled to certain benefits whenever he or she suffers a personal injury by accident *arising out of and in the course of employment*;[31]

2) negligence and fault are immaterial in the claim or employer's defense;[32]

3) coverage is limited to those having the status of "employee," as distinguished from independent contractors;[33]

4) benefits to the employee are usually limited to some percentage of his average weekly wage[34] and hospital or medical expenses[35] and death benefits are provided for surviving dependents;[36]

5) the employee, in exchange for these benefits, gives up his common law right to sue the employer for damages from any injury covered by the act;[37] and

6) the employer is required to secure his liability through insurance or other means,[38] thus shifting the burden of compensation to the consumer through higher prices.

The first acts were primarily concerned with reducing claims litigation; the break with tort law was intended to be complete. Thus, early attempts to equate the "arising out of and in the course of the employment" test with the tort concept of "proximately caused by the employment" were soon discontinued.[39] Presently, the statutory

---

29. 2 A. LARSON, THE LAW OF WORKMEN'S COMPENSATION § 65.10 (1976).

30. *See id.* § 1.10.

31. The criteria for compensation for injuries under the Maine Act were modified by 1973 Me. Laws, Pub. L., ch. 389 (current version at ME. REV. STAT. ANN. tit. 39, § 52 (Supp. 1981-1982)), which substituted "personal injury" for "personal injury by accident" as the new standard. All references to "accident" in the Act were eliminated by the 107th Legislature in 1975. 1975 Me. Laws, Pub. L., ch. 480. The Maine Supreme Judicial Court had pointed out that the earlier amendment did not specifically do so. Canning v. State Dep't of Transp., 347 A.2d 605 (Me. 1975).

32. ME. REV. STAT. ANN. tit. 39, § 3 (1978).

33. *Id.* § 2(5)(A)-(C) (1978 & Supp. 1981-1982).

34. In Maine this percentage is "⅔ his average gross weekly wages, earnings or salary" as computed by the statutory formulae. *Id.* § 2(2)(A)-(F).

35. *Id.* § 52.

36. *Id.* § 58 (Supp. 1981-1982).

37. *Id.* § 28 (1978).

38. *Id.* § 23 (1978 & Supp. 1981-1982).

39. *See, e.g.*, Madden's Case, 222 Mass. 487, 111 N.E. 379 (1916) (expression of the former Massachusetts rule).

right to benefits depends on one simple test: is there a work-connected injury? Larson states the rule: "Let the employer's conduct be flawless in its perfection, and let the employee's be abysmal in its clumsiness, rashness and ineptitude: if the accident arises out of and in the course of the employment, the employee receives his award."[40]

D. The coverage formula: "Arising out of . . ."

The coverage formula is the core of every workers' compensation act and the source of most litigation in the compensation field. The majority of the states and the federal Longshoremen's and Harbor Workers' Compensation Act[41] have adopted the formula of the British Compensation Act: a compensable injury is one "arising out of and in the course of the employment." Although some modifications have been made in state statutes,[42] Maine uses the standard "arising" language.[43] And as Larson notes, "[f]ew groups of statutory words in the history of law have had to bear the weight of such a mountain of interpretation as has been heaped upon this slender foundation."[44]

The phrase is normally applied in its two parts: "arising out of" refers to causal origin of the injury and "course of employment" refers to the time, place, and circumstances of the incident in relation to the work. In application, the phrase requires that both statutory requirements be met.[45] In *Gilbert v. Maheux*,[46] the Maine Supreme Judicial Court, sitting as the Law Court, provided a typical interpretation of the phrase and demonstrated how the test is met. The claimant in *Gilbert* sustained an injury while she was descending a flight of stairs in her employer's premises. Because the employee was living on the premises as a mere convenience and was on her way from her room to dinner when the incident occurred, the employer's appeal challenged whether either requirement of the test had been satisfied. The accident occurred, in time, when the employee was not actively employed and, in place, on the employer's premises, where she was permitted, but not required, to reside.[47] In denying the employer's appeal, the *Gilbert* court held that the commissioner's finding of fact that Mrs. Gilbert was "continuously on call" while present on the premises justified the conclusion that the injury was

---

40. 1 A. LARSON, *supra* note 29, § 2.10.
41. 33 U.S.C. §§ 901-950 (1970).
42. *See, e.g.*, W. VA. CODE § 23-4-1 (1966) (injuries "resulting from" employment).
43. ME. REV. STAT. ANN. tit. 39, § 51 (Supp. 1981-1982).
44. 1 A. LARSON, *supra* note 29, § 6.10.
45. Wolfe v. Shorey, 290 A.2d 892, 893 (Me. 1972); Paulauskis' Case, 126 Me. 32, 34, 135 A. 824, 825 (1927).
46. 391 A.2d 1203 (Me. 1978).
47. *Id.* at 1205.

compensable.[48] The court supported its opinion by citing similar cases from Massachusetts[49] and New Jersey,[50] which held that injuries incurred by the reasonable and proper use of employers' facilities were "incidental to employment and compensable."[51]

The standard of compensability adopted by the Maine court in its interpretation of the "arising out of" test is similar to Larson's "actual risk" doctrine.[52] An "actual risk" is one that is created by the employment, although not necessarily peculiar to it; the hazard may in fact be one that is also common to the public.[53] The important consideration is whether the injuries are "in a just sense related to the employment or have association with the work thereunder."[54] The "arising out of and in the course of employment" test provides the basic *causal connection* between injury and employment. Absent this connection, there can be no compensation.[55]

The issues in a typical workers' compensation claim begin with the injury itself. In many jurisdictions, the "arising out of" test is applied only after an additional requirement is met: the injury must occur by "accident." This term is now defined as an unexpected occurrence "traceable, within reasonable limits, to a definite time, place and occasion or cause."[56] The "by accident" requirement was

---

48. *Id.* at 1207.

49. *In re* Kilcoyne's Case, 352 Mass. 572, 227 N.E.2d 324 (1967). A male nurse was awarded compensation for an injury occurring on his day off while climbing steps to his home in the employer's residence. Although the employee was not required to live on the premises or to be on call during off-duty hours, the Massachusetts court found for the claimant. The court reasoned that the situation gave the employer an "advantage . . . [of] having an employee immediately available, although at his election, to fill in for absent employees." *Id.* at 575, 227 N.E.2d at 326.

50. Barbarise v. Overlook Hosp. Ass'n, 88 N.J. Super. 253, 211 A.2d 817 (1965). In a factual situation similar to *Kilcoyne's Case* and *Gilbert*, the New Jersey court based an award of compensation on the rationale that the residence facilities provided by the employer were mutually beneficial and "like parking lots provided for employees by the employer, [are] 'a part of the *locus* of employment.' " *Id.* at 261, 211 A.2d at 822.

51. 391 A.2d at 1208.

52. 1 A. LARSON, *supra* note 29, § 6.40.

53. *Id. See* Brown v. Palmer Constr. Co., 295 A.2d 263 (Me. 1972) (holding compensable injury resulting from faulty kitchen stove in premises near out of state job site). The *Brown* court ruled that the standard of compensability included risks that are not purely self-created but are created by, and are incidental to, the employment. *Id.* at 266. *Cf.* Barrett v. Herbert Eng'r, Inc., 371 A.2d 633 (Me. 1977) (denying employee's appeal for compensation for low back injury occurring while walking at normal gait to fetch tools).

54. Barrett v. Herbert Eng'r, Inc., 371 A.2d at 636.

55. *See, e.g.*, Ramsdell v. Naples, 393 A.2d 1352 (Me. 1978); Rioux v. Franklin County Memorial Hosp., 390 A.2d 1059 (Me. 1978); Oliver v. Wyandotte Indus. Corp., 360 A.2d 144 (Me. 1976).

56. Riesenfeld, *Contemporary Trends in Compensation for Industrial Accidents Here and Abroad*, 42 CALIF. L. REV. 531, 543 (1954) (citing Fenton v. J. Thorley & Co. [1903] A.C. 443, 448 and Eke v. Hart-Dyke, [1910] 2 K.B. 677, 682).

deleted from Maine's Act in a 1973 amendment;[57] the Act now requires only that the claim be based on disability resulting from a "personal injury."[58]

Application of the basic coverage formula to the typical work-related injury usually results in the speedy resolution promised by the *quid pro quo* of workers' compensation. Generally, only a few issues must be litigated because the employment relationship is known, the matter of fault is not relevant, the time and place of the incident are supported by witnesses, the causal link is apparent, and the employee's wage is on record. The modern claim for compensation based on industrial injury is generally an adequate substitute for its precursor, the action at common law. Claims for disability resulting from *disease* contracted on the job, however, do not share the same common law underpinnings. An occupational disease is a "stranger to the lexicon of the precompensation-era common law."[59]

III. The Development of Occupational Disease Coverage

A. *The Diseases of Man's Occupations*

Man has been aware of occupational diseases since antiquity. The ill effects of manual labor were described by Socrates:

> What are called the mechanical arts are held in utter disdain in our states. For they spoil the bodies of the workmen and the foremen, forcing them to sit still and live indoors, and in some cases to spend the day at the fire. The softening of the body involves a serious weakening of the mind.[60]

Diagnosis and documentation were more common in early history than attempts to treat ailing workers. Their plight was often obscured by the vast social gap between the laboring class and the aristocracy; the practice of medicine at the time was a luxury reserved for the very rich. Additionally, the causes of these diseases of the early laborers were not well understood. In Agricola's time, gases that accumulated in the mines were attributed to the breath of subterranean beasts. No one understood the gases' ill effects on the miners' bodies. Medicine and society were tardy in addressing occupational disease.

In 1713, Bernadino Ramazzini, now known as the father of occupational medicine, first described the conditions suffered by workers

---

57. 1973 Me. Laws, Pub. L., ch. 389 (current version at Me. Rev. Stat. Ann. tit. 39, § 52 (Supp. 1981-1982)).

58. Me. Rev. Stat. Ann. tit. 39, § 51 (Supp. 1981-1982).

59. 1B A. Larson, *supra* note 29, § 41.20. In holding Illinois's Occupational Disease Act unconstitutional, the Illinois Supreme Court noted: "This type of legislation was a complete stranger to the common law, and [this section] under consideration here has no common law origin or history." Boshuizen v. Thompson & Taylor Co., 360 Ill. 160, 163, 195 N.E. 625, 626-27 (1935).

60. Xenophon, Oeconomicus, iv. 204 (Loeb ed. E. Marchant trans. 1965).

who became ill as a result of their employment.[61] In his *Discourse on the Diseases of Workers,* Ramazzini brought the concept of industrial hygiene to Europe. He undertook not only to study the morbid conditions accompanying some occupations, but also to call attention to the practical applications of his knowledge. Ramazzini was keenly conscious of the value of the mechanical arts for economic development and, more broadly, for the progress of civilization as a whole.[62] His work contained a warning for society as it entered the age of industrial and economic expansion:

> [W]e must admit that the workers in certain arts and crafts sometimes derive from them grave injuries, so that where they hoped for a subsistence that would prolong their lives and feed their families, they are too often repaid with the most dangerous diseases and finally, . . . they desert their post among the living.[63]

Ramazzini's warning that workers must be protected from the hazards of the workplace was supported by the economics of mercantilism[64] as European countries competed to secure favorable balances of trade and foreign markets for newly developed manufacturing capacities. It was more than a century later, however, before the most progressive of European countries first recognized a social responsibility for dealing with the problems of industrial diseases.

B. The Development of Occupational Disease Coverage in Europe

The economic reality of occupational disease and its cost to the mercantile system became a concern to European governments late in the nineteenth century. The first nation to provide compensation for losses due to industrial disease was Switzerland.[65] The Swiss Federal Act of 1877 placed liability for such diseases on an equal footing with injuries: "The Federal Council shall also specify those industries the exercise of which demonstrably and exclusively gives rise to specific dangerous diseases, to which liability as defined for accidents shall extend."[66] Included in the Federal Act was a list, or "schedule," of forty-five substances that might cause specific industrial illnesses. The Swiss Act also extended compensation coverage for conditions "caused by work without the intervention of harmful substances."[67]

---

61. B. RAMAZZINI, DISEASES OF WORKERS 449 (W. Wright trans. 1964).
62. For the significance of the mechanical arts in early Europe, see E. JOHNSON, PREDECESSORS OF ADAM SMITH 259-77 (1937).
63. B. RAMAZZINI, *supra* note 61, at 7.
64. *See* E. HECKSCHER, MERCANTILISM (2d ed. 1955).
65. Chojnacki, *Occupational Disease Under the New York Workmen's Compensation Law,* 42 ST. JOHN'S L. REV. 473, 481 (1968).
66. *Id.* (quoting Switzerland, The Federal Act of 1877, § 5).
67. *Id.*

Switzerland's enactment of compensation legislation covering diseases was followed by Germany in 1883 and Austria in 1887. The British Workmen's Compensation Act of 1897,[68] however, took no notice of industrial disease and limited coverage to "personal injury by accident."[69] The British Act, however, was substantially amended in 1906 to include coverage for disability related to various diseases listed on the schedule annexed to the Act:

> [I]f the disease is due to the nature of any employment in which the workman was employed at any time within the twelve months previous to the date of the disablement . . . whether under one or more employers, he or his dependents shall be entitled to compensation under this Act as if the disease . . . were a personal injury by accident arising out of and in the course of that employment.[70]

The annexed schedule listing the covered diseases also had a corresponding list of industrial processes. This list provided a presumption of a causal relationship when the disease was contracted by a worker engaged in that process. The lists could be extended by administrative order to cover additional ailments and exposures.[71] The amended British Act was a "schedule" type statute, as opposed to later "general" coverage acts, which treat any occupational diseases causally related to the employment as "injuries" in the coverage formula. The British model was eventually adopted by most United States jurisdictions.

C. *Occupational Disease Coverage in the United States*

The British government's early recognition of occupational disease prompted demands for similar action in the United States. Yet the earliest of the states' workers' compensation acts excluded any mention of disease.[72] One commentator contends that the drafters of some early acts attempted to use language that would not alarm legislators but would leave the courts free to extend the prescribed coverage to victims of disease.[73] As early as 1914, the Massachusetts Supreme Court interpreted its workers' compensation act to include coverage of occupational disease because the statute provided compensation generally for "personal injury" and not specifically for "personal injury *by accident*."[74] Maine's early workers' compensa-

---

68. 60 & 61 Vict., c. 37, §§ 1-10 (1897).
69. *Id.* § 1(1) (1897).
70. Workmen's Compensation Act, 1906, 6 Edw. 7, c. 58, § 8(1).
71. *Id.* § 8(6).
72. P. BARTH & H. HUNT, *supra* note 9, at 2.
73. Kelley, *Statutes of Limitations in the Era of Compensation Systems: Workmen's Compensation Limitations Provisions for Accidental Injury Claims*, 1974 WASH. U.L.Q. 541, 554.
74. Sullivan's Case, 265 Mass. 497, 164 N.E. 457 (1929); Johnson's Case, 217 Mass. 388, 104 N.E. 735 (1914); Hurle's Case, 217 Mass. 223, 104 N.E. 336 (1914).

tion act required a "personal injury *by accident*," thus making coverage of disease more difficult. In *Bearor's Case*[75] and *Brodin's Case*,[76] liberal construction of the Maine Act resulted in compensation for skin infection and typhoid fever; the court reasoned that these were ailments caused at a single moment in time from a definite work-related infection. These instances of disease coverage, however, were exceptional.

Early resistance to statutory coverage for occupational disease was formidable. Maine's legislature wrestled with disease coverage for eight years before enacting limited coverage in 1945.[77] The New York legislature required a continuous five year effort to change that state's Act.[78] New York enacted its occupational disease statute[79] in 1920 and provided compensation to employees disabled by any of twenty-three scheduled diseases. The scheduled act also included a list of corresponding processes[80] and a statutory presumption of causation[81] similar to the British model.

During the years prior to World War II, state workers' compensation acts were selectively amended to cover certain diseases as public awareness and pressure overcame the legislative resistance to any industrial disease coverage. In the latter 1930's, considerable public attention was drawn to various respiratory diseases related to work, particularly silicosis.[82] For example, hundreds of workers were reported to have died from heavy doses of silica dust while tunnelling on a construction project at Gauley Bridge, West Virginia. The stories of workers buried in shallow mass graves hidden along the isolated road under construction resulted in well-publicized congressional hearings.[83]

Uncertainty concerning the legal status of compensation for occu-

---

75. 135 Me. 225, 193 A. 923 (1937).
76. 124 Me. 162, 126 A. 829 (1924).
77. See text accompanying notes 90-122 *infra*.
78. Chojnacki, *supra* note 65, at 487-88.
79. 1920 N.Y. Laws, ch. 538, §§ 37-496.
80. *Id.* § 49-a.
81. *Id.* § 49.
82. This disease is a form of pneumoconiosis resulting from inhalation of silica (quartz) dust. In advanced cases, pneumoconiosis is characterized by dense fibrosis and emphysema with impairment of respiratory function. TABER'S CYCLOPEDIC MEDICAL DICTIONARY S-46 (13th ed. 1977). Silicosis was of prime concern in New England, where granite quarry workers were particularly affected. See text accompanying notes 119-123 & 128 *infra*. The most striking example of resistance to silicosis coverage in the United States occurred in the granite works of Wisconsin. When that state enacted coverage for silicosis, the resulting insurance premiums soared higher than the payroll itself. The industry collapsed under the burden. 1B A. LARSON, *supra* note 29, § 41.81.
83. Precise estimates of the death toll at Gauley Bridge do not exist. The primary contractor claimed that 48 men died from various diseases during construction; Representative Marcantonio of New York charged that 476 workers had died and 1,500 more were dying from silicosis alone. N.Y. Times, Jan. 23, 1936, at 2, col. 3.

pational diseases may have encouraged the large number of civil suits begun following the Gauley Bridge revelations. By 1934, over $300 million in silicosis claims alone had been filed since 1930.[84] The employers in the silicosis suits who had traditionally resisted occupational disease coverage realized that the exclusive remedy of compensation might prevent the virtual collapse of some industries. The employers' change in position was communicated to legislators; during the 1930's, six states enacted coverage for silicosis.[85]

Incidents such as Gauley Bridge turned the 1930's into watershed years for workers' compensation laws and coverage for occupational disease. But the legislation passed in response to the employers' urging was not of the same benevolent nature as the early injury compensation acts. Rather, occupational disease statutes included severe eligibility requirements, statutes of limitation, and restrictions on available benefits. As a result, "[m]any of the apparent anomalies that exist today [in occupational disease coverage] can be traced to changes and reforms in this earlier decade."[86] Against this backdrop of economic depression and rising litigation, the Maine legislature first considered statutory coverage of occupational diseases.

IV. The Legislative History of Maine's Occupational Disease Law[87]

Although Maine's Occupational Disease Law was enacted in 1945,[88] it originated in the 88th Legislature of 1937. That body appointed a nine-member Recess Committee on Compensation for Occupational Diseases[89] to study the subject and recommend any needed legislation.

A. The Majority Report[90]

The Committee noted that compensation to an injured worker,

84. Because occupational diseases were not covered under many state acts, victims often instituted damage suits against the employers. *See* Solomons, *Workers' Compensation for Occupational Disease Victims: Federal Standards and Threshold Problems*, 41 Alb. L. Rev. 195, 198 n.16 (1977).

85. Trasko, *Socioeconomic Aspects of the Pneumoconioses*, 9 Archives Envt'l Health 521, 523 (1964).

86. P. Barth & H. Hunt, *supra* note 9, at 4.

87. Me. Rev. Stat. Ann. tit. 39, §§ 181-195 (1978).

88. 1945 Me. Laws, Pub. L., ch. 338 (current version at Me. Rev. Stat. Ann. tit. 39, §§ 181-195 (1978 & Supp. 1981-1982)).

89. 1937 Me. Laws, Resolves, ch. 132. The committee consisted of three members from the House, three from the Senate, and three non-members from labor and the employers, appointed by the Governor and Council. Legis. Rec., 88th Me. Legis., Reg. Sess., Senate 978-79 (April 15, 1937).

90. Recess Committee on Compensation for Occupational Diseases, Majority and Minority Reports to the Governor and Council, 88th Me. Legis., Reg. Sess. (1939) (on file in Maine State Law Library, Augusta, Maine) [hereinafter cited as Committee Report].

other than that provided at common law, had taken two paths: one leading to compensation by the community and the other, to compensation by industry itself. Once the injured worker's own funds run out, the ultimate burden must be borne by charity or the public treasury, unless the cost is more properly passed on to industry as a cost of doing business. As the Committee reasoned, "[d]isease like accident causes suffering. Disease like accident may have a causal connection with the man's job. . . . in both cases industry has hurt him, and in both cases industry unlike the workman can set aside reserve funds to meet the contingency."[91]

The Committee considered four possible objections to its "logical suggestion" that workers' compensation should extend to occupational diseases.[92] The most important objection concerned the difficulty in delimiting an extension of workers' compensation that would cover diseases. The problem of delimitation involves the desire to include truly industrially-related illnesses within the coverage, while not completely insuring the worker's health. The Committee noted: "Industry would be flattened out if it were called on to compensate every worker who gets a cold on the job. But how can we compensate for lead poisoning and not for pneumonia?"[93] The Committee's solution to this objection became the distinguishing feature of compensable and noncompensable illness: the word "occupational."

To be occupational, the suffering must be traceable not merely to the work but to a "series of exposures peculiar to that particular industry." The Committee's concern with the "peculiar to" limitation stemmed from a well known New York case of that time, *Goldberg v. 954 Marcy Corp.*[94] In *Goldberg*, a movie theatre cashier claimed that an electric heater in her ticket booth cycled from hot to cold throughout her shift, resulting in blotches and numbness of her lower extremities. While on her way to a doctor's appointment, her legs gave way and she fractured an ankle. She maintained that the weakened condition of her legs contributed to the fall.

Ms. Goldberg was awarded workers' compensation benefits predicated on both accidental injury and occupational disease claims. The feared absolute employers' liability for every employee ailment, real and imaginary, seemed to have occurred. The New York Court of Appeals, however, looked more closely at the occupational disease award and described the concept of compensable occupational disease:

> Thus an occupational disease is one which results from the nature

91. *Id.* at 8.
92. *Id.* at 8-9.
93. *Id.* at 9.
94. 276 N.Y. 313, 12 N.E.2d 311 (1938). This case is referred to in both the majority and minority discussions. COMMITTEE REPORT, *supra* note 98, at 12, 47-48.

> of the employment . . . not those conditions brought about by the failure of the employer to furnish a safe place to work, but conditions to which all employees of a class are subject, and which produce the disease as a natural incident of a particular occupation . . . . Such disease is not the equivalent of a disease resulting from the general risks and hazards common to every individual regardless of the employment in which he is engaged.[95]

The *Goldberg* court affirmed the award of compensation but based the decision solely on the accidental nature of the fall; the occupational disease grounds were rejected.

The Maine Committee postponed its definition of occupational disease to a later portion of the Report. The Committee reiterated, however, that when the two items of the *Goldberg* definition exist—a series of exposures and peculiarity to that industry—"a workman may be compensated even though his suffering is not from a condition commonly known as a 'disease.' 'Occupational' is more important than 'disease.' "[96]

The advocates of general and schedule coverage were distinctly divided concerning the *Goldberg* causal connection requirement. Representatives of labor, industry, and the insurers debated the merits of each other's plans.[97] The employers and occupational disease "experts" generally favored schedule coverage, limited to specific diseases brought on by specific industrial processes. Labor representatives, joined by members of the federal Department of Labor, argued for general coverage.[98]

The employers and insurers were genuinely apprehensive that unlimited general coverage would convince employees that "ills to which all flesh are heir" may be compensable.[99] The critics pointed out that under Illinois's non-schedule act, claims had been filed for rheumatic fever, arthritis, rheumatic heart, dysentary, varicose veins, flat feet, cerebral hemorrhage, and nerve disorders. Although most of these claims were disallowed, employers noted that such claims entailed costs to all parties and disappointment to the worker. The employers further warned that "there is always the possibility that an over-sympathetic administrator may cause confusion and set a dangerous precedent by granting such a claim."[100] The Committee minority joined the employers in this issue, arguing that

---

95. 276 N.Y. at 318-19, 12 N.E.2d at 313. The court found, however, that the fall itself was due to the weakness of her feet, which was, in turn, related to her employment. The court unanimously affirmed the award but solely on the theory of accidental injury.

96. COMMITTEE REPORT, *supra* note 90, at 9-10.

97. *Id.* at 4. The stenographic transcript of that hearing, annexed to the report, is not among materials held in the Maine State Law Library or Archives.

98. *Id.* at 12, 15.

99. *Id.* at 12.

100. *Id.*

general coverage would "[kick] the door wide open to any and all fancied or real ailments that are traceable in imagination, in theory or in fact to an occupation."[101]

The schedule-type coverage does obviate some of the difficult questions of fact and law that arise under occupational disease statutes and may prevent some potential abuses.[102] If the sufferer has a certain scheduled disease, he or she may not have to make a showing that it is peculiar to and characteristic of the industry in which he or she was exposed to the hazard. Under a general plan of coverage, the claimant has the burden of proving the occupational nature of the disease—an issue which may turn on the testimony of expensive experts, unless a precedent has been set for that particular disease. The advocates of schedule coverage feared that in instances of questionable causation and liability, attorneys for employees would gamble on a chance to recover compensation for their clients and the "racketeering" reported in connection with the Gauley Bridge episode would undoubtedly result.[103]

The Committee also heard extensive testimony, primarily from labor, in opposition to the schedule plan. The strong point of the labor argument concerned the unjust treatment of the occupational disease sufferer whose disease, whether by inadvertence or by lack of information, was omitted from the law. The Committee majority saw this potential injustice as counterbalancing the possible danger that commissioners and courts would over-liberalize the law and recommended that Maine's law be of the general coverage type.[104] The Committee majority stated its confidence in the Industrial Accident Commission's[105] ability to ensure that the new law's provisions would not be abused or converted to full-scale health insurance.

After recommending general coverage for Maine's law, the Committee relied on Maine case law for a definition of occupational disease: " 'Occupational disease' shall mean a disease or pathological condition normally peculiar to and gradually caused by hazards of the occupation in which the injured employee was regularly engaged at the time when he last became injuriously exposed to such

---

101. *Id.* at 47.

102. Generally, schedule acts also contain a list of industrial processes. Employment in these processes gives rise to a presumption of causal connection. See text accompanying notes 79-81, *supra*.

103. COMMITTEE REPORT, *supra* note 90, at 13. See also text accompanying note 83 *supra*.

104. COMMITTEE REPORT, *supra* note 90, at 15. The majority also argued that delegating the duty of adding new diseases to the schedule to an administrative board or the Industrial Accident Commission "would be unconstitutional in this state." *Id.*

105. In 1977, the name of the Industrial Accident Commission was changed to Workers' Compensation Commission. 1977 Me. Laws, Pub. L., ch. 612, § 1 (current version at ME. REV. STAT. ANN. tit. 39, § 2 (Supp. 1981-1982)).

hazards."[106] The Committee found its model definition in *Dillingham's Case*,[107] in which the Law Court denied compensation for an "accident" to a leather worker suffering from eczema of the hands. The condition was described by the court as arising from an "insensible progress of occupational disease, [and] was not as matter of law received by accident."[108] In *Dillingham*, the Law Court defined an occupational disease as "one normally peculiar to and gradually caused by the occupation in which the afflicted employee is or was regularly engaged, and to which everyone similarly working in the same industry is alike constantly exposed."[109]

The Committee added the words "pathological condition" to the definition to emphasize that industrial poisoning, though not strictly a disease, was covered, as under the schedule acts. The Committee noted: "[c]larity is what all parties seek. Controversy breeds from uncertainty and controversy leads to litigation with attendant expense, delay, disappointment and rancor."[110] Apparently, the Committee did not believe that the phrases "peculiar to," "gradually caused by," and "last . . . injuriously exposed" would be the subjects of litigation; these qualifications have, in fact, defeated the clarity "all parties seek."

B. The "Special Problem" of Dust Diseases

The sensational publicity surrounding the wave of dust disease claims and tort actions that followed Gauley Bridge strongly influenced the Committee's decision to include coverage for dust diseases under the new law.[111] Ironically, coverage of dust diseases under the

---

106. COMMITTEE REPORT, *supra* note 90, at 29 (Committee Bill § 59). *Compare* the definition enacted by 1945 Me. Laws, Pub. L., ch. 338, § 59:

> Whenever used in this law the term "occupational disease" shall be construed to mean only a disease set forth in section 69, [the schedule list], which is due to causes and conditions which are characteristic of and peculiar to a particular trade, occupation, process or employment and which arises out of and in the course of employment,

with the present definition found in ME. REV. STAT. ANN. tit. 39, § 183 (Supp. 1981-1982): "Whenever used in this law, the term 'occupational disease' shall be construed to mean only a disease which is due to causes and conditions which are characteristic of a particular trade, occupation, process or employment and which arises out of and in the course of employment."

107. 127 Me. 245, 142 A. 865 (1928).

108. *Id.* at 248, 142 A. at 866.

109. *Id.* at 247, 142 A. at 866.

110. COMMITTEE REPORT, *supra* note 90, at 17.

111. The Committee observed:

> Lurid articles on the dangers of dust disease in certain industries, and wholesale discharge of employes [sic] where the insurance rate has been increased to cover a suspected dust disease hazard, have tended to focus attention on the dust diseases. Employers and legislatures have been scared at the impending cloud of dust disease payments.

Id. at 18. There is no indication that the pun was intended.

statutes was not the result of legislative compassion, the urgings of labor activists, or the public outcry over incidents such as Gauley Bridge. Rather, coverage resulted from the fears of employers and insurers that large tort awards would be made unless the compensation system preempted those suits. As coverage and claims expanded, insurance premiums rose at an alarming rate in certain high-risk areas, such as the granite quarry regions of New England. Fear of "insuring a burning house" made it difficult to find carriers willing to underwrite policies for these industries. Consequently, many states that enacted dust disease coverage also enacted special provisions limiting recovery for those claims. The Recess Committee shared these apprehensions and reasoned that even though dust diseases should be included in the recommended bill, efforts must be made "to safeguard the employers and ease the load."[112]

The recommended bill included the following special provisions: 1) at least two years' exposure within the state out of a minimum of five years' exposure to the hazard itself,[113] 2) disability resulting within three years of the last exposure,[114] and 3) benefits limited to a strict maximum "during the early years of the act."[115] Although ostensibly included to cover the special problems of dust disease, these provisions applied with equal force to *all* claims for occupational disease under the new law. Thus, the anomalies between the subsequent Occupational Disease Law and its parent, the Workers' Compensation Act, can be directly traced to the concerns over the "special problems" of dust diseases.

C. *The Enactment of Maine's Occupational Disease Law*

Despite the Committee's efforts during the recess of 1938, the 89th Legislature ignored its recommendations. The recommended bill[116] was not passed in that session; the subject was not debated again for seven years. In 1945, the occupational disease bill was introduced in the 92d Legislature.[117] The bill was quite similar to that recommended by the Recess Committee in 1939. But opposition to such legislation must have remained strong in the state over the intervening war years. The new bill differed significantly from the first by providing for schedule, rather than general, coverage. The new bill contained a schedule of twenty-seven specific diseases and related processes, similar to the model of other scheduled acts. Addi-

112. *Id.* at 21.
113. *Id.* at 22.
114. *Id.*
115. *Id.* at 23.
116. "An Act Extending the Workmen's Compensation Act to Cover Occupational Diseases," H.P. 914, L.D. 305, 89th Me. Legis., Reg. Sess. (1939). *See* Legis. Rec., 89th Me. Legis., Reg. Sess., House 122 (Feb. 2, 1939).
117. H.P. 1238, L.D. 864, 92d Me. Legis., Reg. Sess. (1945).

tionally, however, the bill preceded its list of specific diseases with a "catch all" provision: "any and all occupational diseases. . . ."[118]

The catch-all provision at the beginning of the section was apparently too broad for the members of the Judiciary Committee to whom the bill was referred. The new draft[119] was reduced to a schedule of only thirteen specific diseases and specifically excluded coverage for dust diseases.[120] The bill was extensively debated.[121] Perhaps because of the relative prosperity of the war years or the fact that Maine was late among the states to enact disease coverage, the long-standing opposition to such coverage did not prevail. In 1945, Maine enacted its limited schedule-type Occupational Disease Law.[122]

D. The Liberalization Process: 1951-1975

Since 1945, Maine's occupational disease law has been liberalized through amendments, which broadened its coverage and reduced the claimant's burden of proof. This process began in 1951 with the enactment of coverage for silicosis.[123] Despite a paucity of legislative history concerning this amendment, the language of the section clearly reflects a legislative concern that such additions would "kick open the door" to expensive compensation claims. The silicosis amendment contained a negative presumption that "in the absence of conclusive evidence" supporting the claim, silicosis could not be presumed to have occurred occupationally. This conclusive evidence involved a showing that during the ten years immediately prior to the date of disability, the claimant was exposed to silica dust for five years; two years of the exposure had to occur in the State of Maine.[124] Partial disability due to silicosis was specifically noncompensable and compensable medical treatment was limited to $1000.[125] Interestingly, the silicosis coverage added to the law in 1951, including its restrictive eligibility standard and limited benefits available, is essentially the same as that recommended by the Recess Committee in 1938.[126] Presumably, the earlier fears of eco-

---

118. *Id.* § 69(28).

119. H.P. 1443, L.D. 1137, 92d Me. Legis., Reg. Sess. (1945).

120. "The quarry and granite industry of Maine, because of the excessive cost of insurance coverage, is not covered in this schedule for that reason and is purposely left out." Legis. Rec., 92d Me. Legis., Reg. Sess., House 977 (April 12, 1945)(remarks of Rep. Poulin).

121. *Id.* at 1089-95 (April 17, 1945); *id.*, Senate 1125-30 (April 18, 1945).

122. 1945 Me. Laws, Pub. L., ch. 338 (current version at ME. REV. STAT. ANN. tit. 39, §§ 181-195 (1978 & Supp. 1981-1982)).

123. 1951 Me. Laws, Pub. L., ch. 261, § 1 (current version at ME. REV. STAT. ANN. tit. 39, § 194 (1978)).

124. *Id.* § 2.

125. *Id.*

126. COMMITTEE REPORT, *supra* note 90, at 22-23.

nomic disaster and claimant-attorney racketeering had not lessened during the intervening years.

Compensation for partial disability due to silicosis, however, was added by a 1967 amendment.[127] This amendment also removed the word "conclusive" from the negative presumption and lowered the required time limits for exposure to the dust hazard to two years within ten years preceding the disability. Importantly, the 1967 amendment changed the overall scope of Maine's occupational disease law. The amendment changed the coverage to general by repealing the schedule[128] and made compensation benefits levels available for disease disability subject to the same criteria as those of the Workers' Compensation Act.[129] Coverage for asbestosis was also included in the 1967 amendment,[130] with eligibility standards and a negative presumption similar to the silicosis section.[131]

Yet the "liberalization" of the Law in 1967 was only illusory. The shift from scheduled to general coverage precluded claims for diseases actionable at common law, pursuant to the Act's exclusive remedy provision.[132] Moreover, the restrictive provision for asbestosis may have been enacted in response to a well-publicized study showing the prevalence of the disease among workers at the Bath Iron Works shipyard. For example, in *Davis v. Bath Iron Works Corp.*,[133] a claimant's common law action for damages due to asbestosis was barred by the Act's exclusive remedy provision, although he had left the employment thirteen months prior to the enactment of the 1967 amendment.[134]

---

127. 1967 Me. Laws, Pub. L., ch. 374, § 7 (current version at ME. REV. STAT. ANN. tit. 39, § 194 (1978)).

128. *Id.* §§ 3-5, 10.

129. Benefits levels are set according to the disability suffered by an employee under the Workers' Compensation Act. For total disability, the Act provides: "While the incapacity for work resulting from the injury is total, the employer shall pay the injured employee a weekly compensation equal to ⅔ his average gross weekly wages, earnings or salary. . . ." ME. REV. STAT. ANN. tit. 39, § 54 (Supp. 1981-1982).

For partial disability, the Act provides: "[A] weekly compensation equal to ⅔ the difference, due to the injury, between his average gross weekly wages, earnings or salary before the injury and the weekly wages, earnings or salary which he is able to earn thereafter. . . ." *Id.* § 55.

In the event of death, benefits are payable to surviving dependents. *Id.* § 58.

130. 1967 Me. Laws, Pub. L., ch. 374, § 8 (current version at ME. REV. STAT. ANN. tit. 39, § 194-A (1978)).

131. An asbestosis claimant had to show two years of exposure to the substance within the fifteen years immediately preceding disability. A silicosis claimant had to show exposure within the ten years preceding disability. *Id.*

132. This provision states: "An employee of an employer, who shall have secured the payment of compensation as provided in sections 21 to 27 shall be held to have waived his right of action at common law to recover damages for the injuries sustained by him . . . ." ME. REV. STAT. ANN. tit. 39, § 28 (1978).

133. 338 A.2d 146 (Me. 1975).

134. *Id.* at 148.

The impact of the 1967 amendment was further limited by the retention of a strict statute of limitation, which barred all claims in which the alleged disability arose more than two years after the "last injurious exposure" to the occupational hazard.[135] This provision provided a virtual bar to claims resulting from latent diseases, such as cancers induced by industrial hazards.[136]

The last major amendment to the Occupational Disease Law deleted the words "peculiar to" from the definition of occupational disease and raised from ten to fifteen the number of years preceding disability in which a silicosis victim could show two years of dust exposure.[137] Although there were no Law Court cases interpreting the "peculiar to" language in the former definition, it was apparently thought that the words were confusing and unnecessary. The amended definition still requires a compensable disease to be "characteristic of" the particular industry.

Thus, the "liberalization" process rendered the Occupational Disease Law a far more restrictive statute than the Workers' Compensation Act. The special limits imposed on dust diseases, the remaining confusion posed by the definition of compensable disease, and the strict eligibility requirements of the law restrict its utility in providing compensation for workers disabled by industrial disease. In this regard, Maine's statute reflects most of the problems common to occupational disease legislation in the United States today.

V. Entitlement to Compensation Under Maine's Occupational Disease Law

A. *Compensation for Injury or Disease*

An occupational disease claimant in Maine must adhere to the procedures and requirements of the Workers' Compensation Act[138] and to those peculiar to the Occupational Disease Law.[139] The two statutes were intended to complement each other and are used only when a compensable "injury" occurs. The Application section[140] of

---

135. 1967 Me. Laws, Pub. L., ch. 374, § 5 (current version at Me. Rev. Stat. Ann. tit. 39, § 189 (1978)). The 1967 amendment raised this limit from one year to two; in 1971, the limit was raised to the present three-year limit. 1971 Me. Laws, Pub. L., ch. 376 (current version at Me. Rev. Stat. Ann. tit. 39, § 189 (1978)).

136. See notes 238-242 and accompanying text *infra*.

137. 1975 Me. Laws, Pub. L., ch. 480, §§ 11-12 (current version at Me. Rev. Stat. Ann. tit. 39, § 194 (1978)).

138. Me. Rev. Stat. Ann. tit. 39, §§ 1-112 (1978).

139. *Id.* §§ 181-195.

140. The section provides:

> Except as otherwise specifically provided, incapacity to work or death of an employee arising out of and in the course of the employment, and resulting from an occupational disease, shall be treated as the happening of a personal injury arising out of and in the course of the employment, within the meaning of the Workers' Compensation Act, and all the provisions of

the Occupational Disease Law equates "incapacity to work or death" due to disease to "injury" under the Workers' Compensation Act. Thus, the threshold of compensability under both statutes is the "personal injury arising out of and in the course of the employment."[141] The statutes differ, however, in the definition of occupational disease; under the Occupational Disease Law, the disease must "arise out of and in the course of employment" *and* must be shown to be one "due to causes and conditions . . . characteristic of a particular trade. . . ."[142]

To understand the impact of statutory definitions of "disease," it is useful to analyze the various functions of such provisions. The definition of occupational disease should be a function of the purpose for which it was drafted.[143] Various definitions have been formulated for specific purposes, such as the denial of compensation because an injury is "not an accident but an occupational disease" in jurisdictions which did not recognize such a claim.[144] Similarly, exclusive coverage provisions of the compensation act were avoided in order to maintain a suit for damages relating to safe workplaces and conditions.[145]

Prior to the enactment of the Occupational Disease Law, the Law Court found compensable some disabilities arising from fact situations suggesting disease, rather than injury, as the underlying causa-

---

that Act shall apply to such occupational diseases. This law shall apply only to cases in which the last exposure to an occupational disease in an occupation subject to the hazards of such disease occurred in this State and subsequent to January 1, 1946.

Id. § 182.

141. Section 51 provides:

If an employee who has not given notice of his claim of common law or statutory rights of action, or who has given such notice and has waived the same, as provided in section 28 receives a personal injury arising out of and in the course of his employment or is disabled by occupational disease, he shall be paid compensation and furnished medical and other services by the employer who shall have assented to become subject to this Act.

Id. § 51. For a discussion of the "arising out of" test, see text accompanying notes 50-60 *supra*.

142. ME. REV. STAT. ANN. tit. 39, § 183 (1978). *See* COMMITTEE REPORT, *supra* note 90, at 22.

143. 1B A. LARSON, *supra* note 29, § 41.30.

144. *See* Dillingham's Case, 127 Me. 245, 142 A. 865 (1928).

145. An example of this stratagem is found in Perez v. Blumenthal Bros. Chocolate Co., 428 Pa. 225, 237 A.2d 227 (1968). In *Perez*, plaintiff suffered from pulmonary emphysema, which was either caused or aggravated by his employment. His common law action against the employer for failure to protect his health and safety and for exposing him to heavy dust and heat was successful. The verdict was upheld on appeal; the court ruled that because pulmonary emphysema was not peculiar to the industry or occupation and was common to the population, the suit was not barred by the Pennsylvania Act. *See also* Niles v. Marine Colloids, Inc., 249 A.2d 277 (Me. 1969)(similar facts and holding).

tive factor. In *Gagnon's Case*,[146] an employee was injured after slipping on a wet floor. Her compensation for this accident was reduced by the commission after it discovered that she had been suffering from Parkinson's disease[147] prior to her accident at work and that her present back disability was only 75 percent attributable to the compensable injury.[148] The Law Court reversed the decision reducing her benefits and stated:

> There is no evidence to indicate that her capacity to earn the wages which she was receiving at the time of the accident has been impaired because of the disease. Except for the accident she might still be able to earn the same wages. Assuming that she could, the appellees would be responsible for her total incapacity.[149]

In *Crowley's Case*,[150] a compensation award appeal was based on the contention that disease, rather than injury, underlay the disability. The employee had been initially incapacitated after an exposure to carbon monoxide gas. Medical examinations after the accident revealed that the claimant suffered from an arteriosclerotic condition and leukemia. The Law Court affirmed an award of total disability because the injury "brought about his incapacity or aggravated a preexisting condition. . . ."[151] Today, Ms. Gagnon and Mr. Crowley could still prevail under the Workers' Compensation Act. Clearly, they could not prevail on a claim for occupational disease under the present definition; their diseases are not "characteristic" of their trades.[152]

A statutory definition of disease also immunizes the employer from statutory or common law actions concerning a safe workplace. In *Davis v. Bath Iron Works Corp.*,[153] the Law Court reviewed the denial by a Superior Court justice of an employer's motion to dismiss. A worker alleged that his contraction of asbestosis was due to the employer's negligent failure to warn of the hazardous nature of

---

146. 144 Me. 131, 65 A.2d 6 (1949).

147. Parkinson's disease is a chronic nervous disease characterized by muscular weakness and rigidity and a peculiar gait. TABER'S CYCLOPEDIC MEDICAL DICTIONARY P-28 (13th ed. 1977).

148. 144 Me. at 132, 65 A.2d at 7.

149. *Id.* at 133, 65 A.2d at 8.

150. 130 Me. 1, 153 A. 184 (1931).

151. *Id.* at 5, 153 A. at 185.

152. *See* Brawn v. St. Regis Paper Co., 430 A.2d 843 (Me. 1981). In *Brawn*, the claimant was denied compensation under the Occupational Disease Law because his pulmonary emphysema, although aggravated by dust in his work environment, was not an "occupational disease" under section 183 of the Law. The Law Court noted that the Commission must find that the conditions of employment were at least one factor *causing* the disease. The court reasoned that "[s]uch causal contribution to the disease process is to be distinguished from a factual situation in which the conditions of employment merely aggravate, or worsen, the symptoms of a pre-existing disease." *Id.* at 845.

153. 338 A.2d 146 (Me. 1975).

the employment. The *Davis* court relied on *Niles v. Marine Colloids, Inc.*[154] and held that the employee was limited to his exclusive remedy under the Occupational Disease Law.[155] In *Niles*, the plaintiff sought damages at common law for having developed pulmonary emphysema during the course of his employment. Noting that emphysema was not a compensable occupational disease under the then existing scheduled act, the Law Court sustained the employee's appeal from a dismissal of his action below.[156]

Occupational disease definitions also limit benefits or force the application of unusual procedural rules in states where specific restrictions are placed on occupational disease claims as opposed to injury claims. Maine's definition of occupational disease apparently fits this latter category; the disease must meet the "arising out of test" and must be *characteristic* of the employment.

The definition of occupational disease is one of the few sections of the Maine law that has been construed by the Law Court.[157] In *Russell v. Camden Community Hospital*,[158] a nurse's aide claimed compensation after she became ill with tuberculosis. She alleged that she had contracted the disease as a result of intimate contact with a tuberculative patient under her care. The employer appealed an award by the commission and argued that the claimant's exposure to the disease was not within the sixty days required by the law[159]

---

154. 249 A.2d 277 (Me. 1969).

155. 338 A.2d at 148.

156. 249 A.2d at 279.

157. Since its enactment in 1945, only five cases under the Occupational Disease Law have reached the Law Court: Brawn v. St. Regis Paper Co., 430 A.2d 843 (Me. 1981)(aggravation, as opposed to causation, of pulmonary emphysema by conditions at paper mill not compensable under Occupational Disease Law); McKenzie v. C. F. Hathaway Co., 415 A.2d 252 (Me. 1980)(claimant's emphysema and chronic bronchitis not caused or aggravated by conditions at employer's plant); Russell v. Camden Community Hosp., 359 A.2d 607 (Me. 1976)(tuberculosis is disease characteristic of or peculiar to employment of nurse's aide claimant); Davis v. Bath Iron Works Corp., 338 A.2d 146 (Me. 1975)(asbestosis compensable notwithstanding provision not enacted until after employee ceased work); Niles v. Marine Colloids, Inc., 249 A.2d 277 (Me. 1969)(pulmonary emphysema not scheduled disease nor injury by accident; common law action could ensue).

158. 359 A.2d 607 (Me. 1976).

159. The employer based his first argument on section 186 of the Law, which provides:

> The date when an employee becomes incapacitated by an occupational disease from performing his work in the last occupation in which he was injuriously exposed to the hazards of such disease shall be taken as the date of the injury equivalent to the date of injury under the Workers' Compensation Act. Where compensation is payable for an occupational disease, the employer in whose employment the employee was last injuriously exposed to the hazards of such disease, and the insurance carrier, if any, on the risk when such employee was last so exposed under such employer, shall be liable therefor. The amount of the compensation shall be based upon the average wages of the employee when last so exposed under such employer,

and that tuberculosis was not a disease characteristic of or peculiar to her employment. The Law Court rejected both contentions and took the opportunity to discuss the definition of occupational disease:

> The requirement that the disease be 'characteristic of or peculiar to' the occupation of the claimant precludes coverage of diseases contracted merely because the employee was on the job. . . . To be within the purview of the Law, the disease must be so distinctively associated with the employee's occupation that there is a direct causal connection between the duties of the employment and the disease contracted.[160]

Finding that Ms. Russell contracted the disease as a direct result of providing care to a patient, the court affirmed the award because the requisite causal connection had been satisfied.[161] According to *Russell*, a compensable disease must not only meet the arising test but must also have an even closer connection, a "distinctive" association with the employment.

Maine's definition of occupational disease reflects a common element found in many state statutes containing detailed definitions:

> and notice of injury and claim for compensation shall be given and made to such employer. *The only employer and insurance carrier liable shall be the last employer in whose employment the employee was last injuriously exposed to the hazards of the disease during a period of 60 days or more, and the insurance carrier, if any, on the risk when the employee was last so exposed, under such employer.*

ME. REV. STAT. ANN. tit. 39, § 186 (1978)(emphasis added). The evidence showed that the claimant had been exposed to the patient for only twenty days; the employer contended that she did not meet the sixty day minimum exposure requirement. In a *per curiam* opinion, the court reasoned that because there are no cross-references to that requirement in other sections of the law (specifically, the silicosis and asbestosis provisions), the section is not a blanket exposure requirement. Rather, the section determines which employer, if more than one is involved, will be liable for compensation. The section also establishes guidelines for determining the amount of compensation due from the liable employer. 359 A.2d at 610.

An important omission from the *Russell* court's analysis, however, was consideration of the wording of section 195, which provides:

> Notwithstanding any of the provisions of this chapter, the employee need not be exposed to radioactive substances for a period of 60 days or more, *as otherwise stated under section 186*, and the time for filing claims shall not begin to run in cases of incapacity until the person claiming benefits knew, or by exercise of reasonable diligence should have known of the casual [sic] relationship between his employment and his incapacity, or after incapacity, whichever is later.

ME. REV. STAT. ANN. tit. 39, § 195 (1978)(emphasis added).

The reference to section 186, absent any mention of successive employers, would indicate that the legislature intended the section to mean exactly what the employer in *Russell* contended: a minimum exposure of sixty days to the hazard is necessary before causal connection could be shown.

160. 359 A.2d at 611-12.

161. *Id.* at 612.

the distinctive relation of the particular disease to the nature of the employment, as contrasted with disease that might be contracted as easily in other employments or everyday life, regardless of employment.[162] Although these statutes bar compensation for "ordinary diseases of life," the distinction is illusory. As Larson noted,

> [t]he infinite variety of conditions of other employments—ranging from accounting to lead mining and from baby-sitting to topping Douglas Fir trees—is just as great as the variety of conditions of non-employment life, and has no more of a common element than does "everyday life" to supply a measuring stick by which to judge what is "ordinary" and what is distinctively occupational in a particular employment.[163]

In cases involving exposures to chemicals, fumes, dust, or similar irritants and hazards, distinguishing ordinary diseases is not difficult. But the majority of controverted cases involve the problems of proof necessitated by the requirement that the disease be "characteristic" of the employment rather than problems concerning the definition itself. Applying the standard calling for a distinctive association or a distinction from "ordinary" diseases or conditions has resulted in conflicting and controversial decisions in other jurisdictions. For example, in *Carter v. International Detrola Corp.*,[164] a claimant developed a progressive swelling and stiffening of the arms and hands from repetitive motions required by her assembly line work. The Michigan court denied an award of compensation based on occupational disease because "[t]he resulting excessive movement of the scalenus anticus muscle is not so unique as to be 'characteristic of and peculiar to' the business of the employer."[165] Yet a few months later, the same court found compensable a gradually developing lumbosacral condition resulting from repetitive stooping or bending in *Underwood v. National Motor Casting Division, Campbell, Wyant & Cannon Foundry Co.*[166] The *Underwood* court found the disability arose from the nature of the employment, which "constituted causes and conditions which were characteristic of and peculiar to the defendant's business."[167] These Michigan cases are difficult to reconcile. They illustrate, however, the causation problems generated by the definition of occupational disease.

A compensable injury must "arise out of" and occur "in the course of" the employment. The Law Court has found this test satisfied

---

162. *See id.*

163. 1B A. LARSON, *supra* note 29, § 41.33, at 7-365.

164. 328 Mich. 367, 43 N.W.2d 890 (1950).

165. *Id.* at 370, 43 N.W.2d at 891; *cf.* Ross v. Oxford Paper Co., 363 A.2d 712 (Me. 1976)(onset of disability due to repetitive trauma to hands compensable as gradual injury).

166. 329 Mich. 273, 45 N.W.2d 286 (1951).

167. *Id.* at 276, 45 N.W.2d at 287.

when an injury results from a risk that is created by, and is incidental to, the employment rather than a self-created risk.[168] Thus, the court has found compensable injuries such as slipping in a puddle of coating fluid[169] or being involved in a traffic accident on a public street after leaving the employer's premises.[170] But it is difficult to see a distinction between the employment connection of a slip and fall at work and the noncompensable case of pneumonia "contracted merely because the employee was on the job."[171] Both incidents meet the "arising" test and both seem to fit either the "actual risk" or the "increased risk" doctrines of causal connection.[172] The extra burden of proving that a disease is "characteristic" of a particular employment is apparently the result of a legislative desire to preclude claims for the common cold and other "ordinary diseases of life." Yet if the basic purpose of compensation statutes is to replace partially wages lost due to an incapacity to work causally connected with the employment, this purpose is not served by this distinction. Any disability that incapacitates the employee for the three days required for compensation[173] and that meets the "arising" test of causation should be compensable, regardless of whether it results from industrial injury or occupational disease. The causation-plus burden imposed on the disease sufferer by Maine's definition of occupational disease is an arbitrary and unfair requirement, which can result in needless litigation and denial of claims. Both results are contrary to the purposes of workers' compensation.

B. The Claim Procedure

There are other features of the Occupational Disease Law that are not justified in light of workers' compensation law in general. These features include the exceptional *eligibility* requirements for disease claims, which are couched in terms of procedural requirements, and statutes of limitation.

To describe most effectively the procedural steps and potential pitfalls of Maine's Occupational Disease Law, a hypothetical claimant[174] is useful. At age fifty-seven, Richard Roe is dying. What began

---

168. Brown v. Palmer Constr. Co., 295 A.2d 263 (Me. 1972).

169. Soucy v. Fraser Paper, Ltd., 267 A.2d 919 (Me. 1970).

170. Oliver v. Wyandotte Indus. Corp., 360 A.2d 144 (Me. 1976).

171. Russell v. Camden Community Hosp., 359 A.2d at 611.

172. 1B A. LARSON, *supra* note 29, § 6.30; see text accompanying notes 52-59 *supra*.

173. Section 53 provides: "No compensation for incapacity to work shall be payable for the first 3 days of incapacity, except that firemen shall receive compensation from the date of incapacity. In case incapacity continues for more than 14 days, compensation shall be allowed from the date of incapacity." ME. REV. STAT. ANN. tit. 39, § 53 (1978).

174. The claimant, Richard Roe, is a hypothetical occupational disease victim constructed from pleadings in several products liability suits filed in the United

as general dyspnea, shortness of breath after exertion, has now been diagnosed as asbestosis,[175] an insidious and incurable form of pneumonoconiosis caused by the inhalation of asbestos fibers. Roe traces his exposure to asbestos to a job he held for eighteen months after graduating from high school. As an apprentice insulator, he traveled throughout New England and worked for various contractors doing "rip-outs" of old insulation blankets wrapped around boilers and pipes, mixing asbestos mortar, and cleaning up after the insulators. His exposure to airborne dust and asbestos fibers was extensive. After this short employment period, Roe worked in heavy construction. Today, many years after his exposure to asbestos, Roe's disease has intensified; he is physically incapacitated. Faced with unemployment and mounting medical bills, Roe files a claim for compensation benefits and alleges an occupational disease and disability.

The procedural path of his claim begins with the requirement of "notice" to his employer. All time periods regarding notice and the filing of an occupational disease claim are measured from the date of incapacity.[176] This date is defined as "[t]he date when the employee becomes incapacitated by an occupational disease from performing his work in the last occupation in which he was injuriously exposed to the hazards of such disease. . . ."[177] Roe is immediately presented with the first of many ambiguities in the Law: what is an injurious exposure? When were the first asbestos fibers inhaled? The last? Experts in the field cannot determine the point at which exposure to asbestos is "injurious." But the meaning of this ambiguous phrase will become crucial to Roe when he reaches a later procedural requirement. For the purposes of notice, however, it is reasonable to assume that "injurious exposure" means the employment in which the exposure occurred rather than the exact point in time.

States District Court for the District of Maine.

175. The pathogenesis of asbestosis was described by Dr. Merewhether, an early pioneer in the area of occupational disease:

> This disease, insidious at its onset, stealthily advances with but faint warnings of its progress; inexorably it cripples the essential tissues of the lungs, yet for a considerable period causes almost no inconvenience to the worker. As time goes on, however, the lungs find more and more difficulty in re-aerating the blood; and breathing is quickened on slight exertion. Still the worker is able to remain at work, but is aware of his undue shortness of breath on extra effort. Usually, however, he ascribes it to causes other than the dust. . . .
>
> As the disease progresses, if no acute illness has caused a fatal termination, a stage is reached when the shortness of breath is extreme. Even in its terminal stages, the disease, deceitful to the last, may masquerade as chronic bronchitis, pulmonary tuberculosis, bronchopneumonia, or the like.

Merewhether, *The Occurrence of Pulmonary Fibrosis and Other Pulmonary Affections in Asbestos Workers*, 12 J. INDUS. HYGIENE 198, 201-02 (1930).

176. *See* ME. REV. STAT. ANN. tit. 39, § 187 (1978).

177. *Id.* § 186; see note 159 *supra*.

The Law requires that Roe give the employer notice of an incapacity due to occupational disease within thirty days of the incapacity.

Although the question of the date of incapacity due to occupational disease has never been considered by the Law Court, it is possible that the date will be construed similarly to the notice requirements of section 63 of the Workers' Compensation Act.[178] Under most injury-type statutes requiring no "accident,"[179] there is almost complete agreement that the claim period runs from the time the compensable injury becomes apparent.[180] For example, *Ross v. Oxford Paper Co.*[181] involved the gradual onset of carpal tunnel syndrome in a paper worker's hands. The Law Court held that the compensable injury became apparent "when the disability manifested itself to such an extent that [the claimant] was compelled to cease work."[182] Carpal tunnel syndrome is a neurological condition of the hands and lower arms, which results from repetitive trauma to certain parts of the hand.[183] Because its onset is gradual and often unnoticed, the syndrome is similar in many respects to "disease." The *Ross* court's treatment of the date of injury as the date of incapacity is reasonably consistent with "date of incapacity" under the Occupational Disease Law.

The notice requirement of the Workers' Compensation Act provides a time extension for that period "during which the employee is unable by reason of physical or mental incapacity to give said notice, or fails to do so on account of mistake of fact. . . ."[184] Although this requirement has yet to be litigated, it appears reasonable to allow

---

178. Section 63 provides:

> No proceedings for compensation under this Act, except as provided, shall be maintained unless a notice of the injury shall have been given within 30 days after the date thereof. Such notice shall include the time, place and cause, and the nature of the injury, together with the name and address of the person injured. It shall be given by the person injured or by a person in his behalf; or, in the event of his death, by his legal representatives, or by a dependent or by a person in behalf of either.
>
> Such notice shall be given to the employer, or to one employer if there are more employers than one; or, if the employer is a corporation, to any official thereof; or to any employee designated by the employer as one to whom reports of accidents to employees should be made. It may be given to the general superintendent or to the foreman in charge of the particular work being done by the employee at the time of the injury.

ME. REV. STAT. ANN. tit. 39, § 63 (1978).

179. In 1973, Maine deleted the "by accident" provision from its Workers' Compensation Act. 1973 Me. Laws, Pub. L., ch. 389 (current version at ME. REV. STAT. ANN. tit. 39, § 52 (Supp. 1981)). For a discussion of this change, see Canning v. State Dep't of Transp., 347 A.2d 605 (Me. 1975).

180. 3 A. LARSON, *supra* note 29, § 78.42(a).

181. 363 A.2d 712 (Me. 1976).

182. *Id.* at 716.

183. *Id.* at 713.

184. ME. REV. STAT. ANN. tit. 39, § 64 (1978).

the occupational disease victim to rely on an extension of the period for similar cause. For the purposes of the hypothetical, Roe meets the notice requirement.

The second procedural issue involves filing. An employee's claim for compensation under the Workers' Compensation Act is barred, unless a petition or an agreement is filed within two years after the date of injury or within two years after the last voluntary payment made by the employer under the law.[185] Possibly to encourage the payment of voluntary benefits by employers to disease victims, the filing period to reopen a claim is shortened under the Occupational Disease Law to one year after the last payment.[186] Extensions of the two year after-injury filing period are available for any time an employee is physically or mentally incapacitated; in the case of mistake of fact, the extension is available "within a reasonable time."[187] There is an absolute bar to all claims arising ten years after the date of the last payment under the Act.[188] Because his date of "injury" is considered under the Occupational Disease Law as the date of "incapacity," Roe can presumably comply with the filing period, although it is unclear if the ten year bar would affect him as an absolute statute of limitation. The statutes of limitation presented by the notice and filing periods of the Workers' Compensation Act are similar in purpose to other statutes limiting stale claims. When applied to occupational disease claims, however, the statutes' rationale fails; the statutes tend to bar more "good" than "bad" claims.

Roe's third procedural step involves the eligibility period for occupational disease claims. When the Maine Occupational Disease Law was amended in 1967 to provide general coverage in place of the repealed schedule of diseases, section 189, which severely limits the scope of coverage, was retained.[189] Section 189 bars all claims for compensation due to incapacity not manifested within three years after the last injurious exposure to the industrial hazard. Under this provision, Roe's claim for disability due to asbestos exposure in his past employment is effectively barred, despite the fact that asbestosis, like many latent diseases, can take several years to progress to the point of incapacity.[190] Again, the ambiguous phrase "injurious

185. *Id.* § 95 (Supp. 1981-1982).
186. *See* note 176 *supra.*
187. *See* note 185 *supra.*
188. *Id.*
189. Section 189 provides:

> Compensation for partial or total incapacity or death from occupational disease shall be payable in the same manner and amounts as provided in sections 54, 55 and 58. Compensation shall not be payable for incapacity by reason of occupational disease unless such incapacity results within 3 years after the last injurious exposure to such disease in the employment.

ME. REV. STAT. ANN. tit. 39, § 189 (1978).
190. There is ample medical authority for the proposition that many latent degen-

exposure" is present; because Roe has not been exposed to asbestos at all during his intervening employment, the bar would apparently apply even if the term means the last exposure, "injurious" or not.

The Workers' Compensation Act has no similar provision, other than the ten-year statute of limitation imposed by section 95. As discussed below, a three-year limit on disease claims, compared with a ten-year limit for injury claims, suggests an equal protection argument for Roe. This issue has not yet been litigated in Maine.[191]

C. *Statutes of Limitation: Rationales*

Roe can definitively link his exposure to an industrial hazard in his employment and can prove the disabling consequences of that exposure. The absolute bar presented by statutes of limitation, such as section 189, is unjustified. In fact, the traditional rationales for such statutory limitations do not support their application in workers' compensation law in general.

Several rationales underlie statutes of limitation.[192] As a policy matter, such laws are defended as protection against stale claims after evidence has been lost, memories have faded, and witnesses have disappeared. This defense consists of two component arguments: 1) the statutes are based on the presumption that a "stale" claim is a "bad" one because the claim could have been successfully resisted had it not been for the passage of time; and 2) the limitation is one of "repose," based on the theory that courts have enough to do without adjudicating claims that arose years ago.

Neither argument, however, fits a workers' compensation claim. Claims do not become "stale" like beer or bread.[193] The adjective "stale" refers to a claim that is unenforceable or describes the purpose and result of the statute's application itself — an attempted cause of action that has failed. In either case, the word "stale" explains nothing about the justification of a time limitation. Essentially, the stale claim justification prohibits the successful assertion of claims after evidence to refute them has disappeared. The time bar presumes that the plaintiff's assertions are without merit because they could have been resisted successfully if evidence had

---

erative diseases can manifest themselves many years after initial exposure to the causative hazard. *See, e.g.*, NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH, A GUIDE TO THE WORK-RELATEDNESS OF DISEASE (1979); I. SELIKOFF & D. LEE, ASBESTOS AND DISEASE (1978) (cancers due to asbestos exposure manifest ten to forty years after exposure); Estep & Allan, *Radiation Injuries and Time Limitations in Workmen's Compensation Cases*, 62 MICH. L. REV. 259 (1963).

191. *See, e.g.*, Morgan v. Schuler's Restaurant, 64 Mich. App. 37, 234 N.W.2d 885 (1975). For a discussion of this case, see text accompanying notes 232-235 *infra*.

192. *See, e.g.*, Callahan, *Statutes of Limitation - Background*, 16 OHIO ST. L.J. 130 (1955); *Developments in the Law - Statutes of Limitations*, 63 HARV. L. REV. 1177 (1950).

193. Callahan, *supra* note 192, at 133.

been available.

But if it is presumed that a compensation claimant's allegations are meritorious, the defendant is not prejudiced by the elapsed time because the claim could not have been defended at any time. The workers' compensation claimant must still prove employment relation, injury, disability—the issues encompassed in the "arising out of and in the course of" test. Proof of employment relation would not be affected by the elapsed time. Similarly, the issue of disability would not be affected by time elapsed because it is the claimant's *present* condition that must be considered by the factfinder. Only causation will be clouded by defects in the evidence. Even if the fact finder errs in favor of the claim, the system's goal of shifting some of the burden of disability from the employee to the industry is attained.[194] Thus, if Roe's exposure is somehow documented, the elapsed time between that exposure and the eventual disability (the disease's latent period) becomes irrelevant to the issue of causation. Clearly, a meritorious claim should not be barred by such a "failure" of evidence.[195]

Statutes of limitation are also statutes of repose.[196] Of course, those seeking repose under statutes of limitation are defendants. The repose justification overlaps the goal of protecting defendants against the loss of evidence necessary to refute a claim. Again, if the claim is assumed to be without merit, the policies of protecting against missing evidence and of favoring a defendant's repose are quite similar. But if the claim is assumed to be meritorious, the statute of limitation should not enable a tort-feasor or an industry in which a worker is crippled to proceed, after a time, as though the incident never happened.[197]

The Workers' Compensation Act is consistently construed in favor of the injured worker.[198] Indeed, section 92 of the Act mandates such a construction.[199] In cases such as Roe's, the time that elapses between exposure and manifestation passes, necessarily, unnoticed. Application of a statute of limitation, such as that contained within

---

194. Kelley, *supra* note 73, at 626.

195. Justice Gray stated: "The statute of limitations was not enacted to protect persons from claims fictitious in their origin, but from ancient claims, whether well or ill founded, which may have been discharged, but the evidence of discharge may be lost." Shepherd v. Thompson, 122 U.S. 231, 236 (1887).

196. Guaranty Trust Co. v. United States, 304 U.S. 126, 136 (1938); Shepherd v. Thompson, 122 U.S. 231, 235 (1887); Bell v. Morrison, 26 U.S. (1 Pet.) 350, 360 (1828).

197. Callahan, *supra* note 192, at 135.

198. Gilbert v. Maheux, 391 A.2d 1203, 1205 (Me. 1978) (law must be construed liberally in favor of employee); *In re* Dudley, 256 A.2d 592, 594 (Me. 1969) (Act must be construed liberally in favor of workman).

199. ME. REV. STAT. ANN. tit. 39, § 92 (Supp. 1981-1982) reads in part: "[T]his Act [shall be construed] liberally and with a view to carry out its general purpose."

section 189 of the Law, serves no purpose other than the barring of an otherwise conpensable claim.

D. One More Anomaly: Apportionment of Disability

Even if Richard Roe can somehow overcome the bar of the two-year[200] and three-year[201] statutes of limitation, he faces yet another provision of the Occupational Disease Law that has no counterpart in the "injury" sections of the Workers' Compensation Act. Section 185 of the Law[202] provides that an award of compensation for occupational disease shall be reduced by that portion of the disability attributed to "any other disease or infirmity, not itself compensable." Essentially, this provision allows the employer to present evidence that the industrial hazard either combined with or aggravated a preexisting condition, such as the ill effects of cigarette smoking, and that the resulting disability must be apportioned between the two causes.

There are two forms of apportionment in the Workers' Compensation Act: apportionment between successive employers or carriers when the final disability is traceable to exposures or incidents under two or more of them[203] and apportionment between an employer and the Second Injury Fund when a preexisting permanent impairment covered by the Fund is involved.[204] Yet there is an important difference between apportionment in occupational disease cases and apportionment under the injury statute. Under the Workers' Compensation Act, the employee is assured full benefits from someone; the only issue concerns prospective payors. But in the occupational disease situation, the injured worker faces the possibility of having to bear personally a substantial portion of the final loss.

The apportionment section of the Occupational Disease Law presents two additional problems. First, in attempting to assign a portion of the final disability to preexisting noncompensable condi-

---

200. *Id.* § 95.
201. *Id.* § 189 (1978).
202. Section 185 provides:

> Where an occupational disease is aggravated by any other disease or infirmity, not itself compensable, or the death or incapacity from any other cause, not itself compensable is aggravated, prolonged, accelerated or in anywise contributed to by an occupational disease, the compensation payable shall be reduced and limited to such proportion only of the compensation that would be payable if the occupational disease were the sole cause of the incapacity or death as such occupational disease, as a causative factor, bears to all the causes of such incapacity or death, such reduction in compensation to be effected by reducing the number of weekly or monthly payments or the amounts of such payments, as under the circumstances of the particular case may be for the best interest of the claimant or claimants.

Id. § 185.
203. *Id.* § 104-B (Supp. 1981-1982).
204. *Id.* § 57.

tions, the fact finder faces a task of enormous complexity and uncertain results.[205] Second, apportionment of disability between compensable and noncompensable conditions is contrary to the theory of compensation law as it has evolved in Maine. Application of this apportionment in disease cases runs counter to the long-standing doctrine that an employer takes the employee as he or she finds the employee.[206]

Formulating a legal standard for apportionment of occupational diseases that have been aggravated or contributed to by noncompensable conditions is difficult. Expert medical opinions often conflict concerning whether a disability is due solely to occupational disease or is due to a combination of the work hazard and factors such as aging, personality, life-style, or genetic conditions.[207] Unlike attorneys, who are concerned primarily with the legal future of the resulting disability, physicians view disease or injury in terms of many past and present physiological factors. Disagreement among experts occurs concerning which factors are truly causative and which are merely correlative.[208] For example, a workers' compensation fact finder is frustrated by medical testimony suggesting that hypercholesterolemia is a risk factor for heart disease but that many people with elevated cholesterol have no evidence of heart disease.[209]

The Law Court has not yet formulated standards for apportionment in disease cases.[210] The difficulties of multiple causation have

---

205. There are at least three influential factors to be weighed carefully in this determination: the variability in the exact nature of the hazardous exposure, the physical state of the exposed worker, and the various environmental factors that accompanied the exposure. When the problem is a respiratory one, the most difficult etiological issues involve the multiple causality created by the victim's smoking habits.

For a discussion of the complexity of multifactorial causation, see P. BARTH & H. HUNT, *supra* note 9, at 70-74.

206. Bernier v. Coca-Cola Bottling Plants, Inc., 250 A.2d 820 (Me. 1969); 1 A. LARSON, *supra* note 29, § 12.20.

207. Ladov, Mulryan, & McCarthy, *Cumulative Injury or Disease Claims: An Attempt to Define Employer's Liability for Workers' Compensation*, 6 AM. J.L. & MED. 1, 12-13 (1980).

208. *Id.*

209. *Id.*

210. The Law Court has twice mentioned the apportionment provision in published opinions; in both cases, the court has reserved decision on the section's meaning. Brawn v. St. Regis Paper Co., 430 A.2d at 845 n.4 (because no occupational disease existed, court had no occasion to decide whether meaning of § 185 had been affected by changes in statutory definition of occupational disease in § 183); McKenzie v. C. F. Hathaway Co., 415 A.2d at 254 n.4 (noting that § 185 was enacted at time when "occupational disease" had different definition under § 183). The court is apparently signaling its willingness to interpret the apportionment section in light of both the different treatment of apportionment under the Workers' Compensation Act and the effect of deleting the "peculiar to" term from the statutory definition of occupational disease.

resulted in conflicting decisions in other jurisdictions with apportionment statutes. For example, California's apportionment provision[211] allows compensation "only for that proportion of the disability due to the aggravation [of a prior disease] . . . reasonably attributed to the injury." In *Southern California Edison Co. v. Industrial Accident Commission*,[212] the claimant suffered a head injury resulting in a detached retina, emotional disturbance, and heart attack. Evidence showed, however, that the heart attack was also caused in part by preexisting coronary arteriosclerosis. The court held that it was error not to apportion the resulting disability between the industrial accident and the non-industrial disease. But in *Pacific Employers Insurance Group v. Workmen's Compensation Appeals Board*,[213] a disability resulting in part from a preexisting and advanced lumbosacral disc disease was held to be fully compensable. In the latter decision, the court apparently determined that the disc disease was asymptomatic prior to the work injury and thus not a "disability" that required apportionment.[214]

California's requirement of a preexisting *disability*, as opposed to non-disabling conditions, before apportionment applies was further refined in *Zemke v. Workmen's Compensation Appeals Board*.[215] In *Zemke*, the claimant suffered from preexisting, non-disabling osteoarthritis, aggravated by a work-related back strain. Citing the lack of evidence that the employee would have suffered any disability from the arthritis had it not been for the accident and that any part of his present disability was the result of the natural progression of the arthritis, the California court held apportionment inapplicable.[216]

Importantly, California has construed its statute to mandate apportionment of the employee's *disability*, not apportionment among *causes* of his disability. This principle requires apportionment only in those situations in which the worker's disability would have occurred even without industrial aggravation, as part of the normal progress of the preexisting disease.[217] Thus, in *Pullman-Kellogg v.*

---

211. CAL. LAB. CODE § 4663 (West 1971).

212. 238 Cal. App. 2d 567, 48 Cal. Rptr. 46 (1965).

213. 247 Cal. App. 2d 102, 55 Cal. Rptr. 176 (1966).

214. *Id.* at 108, 55 Cal. Rptr. at 180. *See also* Gagnon's Case, 144 Me. 131, 65 A.2d 6 (1949). For a discussion of this case, see text accompanying notes 146-149 *supra*.

215. 68 Cal.2d 794, 441 P.2d 928, 69 Cal. Rptr. 88 (1968).

216. *Id.* at 799, 441 P.2d at 932, 69 Cal. Rptr. at 92. *See also* Berry v. Workmen's Compensation Appeals Bd., 68 Cal.2d 786, 441 P.2d 908, 69 Cal. Rptr. 68 (1968)(dormant fungus disease, which localized in knee by work accident and which resulted in disability, held not apportionable because fungus had not caused any disability prior to injury).

217. *See* Duthie v. Workmen's Compensation Appeals Bd., 86 Cal. App. 2d 271, 150 Cal. Rptr. 530 (1978); Hart v. Workmen's Compensation Appeals Bd., 82 Cal. App. 2d 642, 147 Cal. Rptr. 384 (1978).

Workmen's Compensation Appeals Board,[218] apportionment was held inapplicable in a case of lung disease exacerbated by a thirty-year smoking habit. Medical testimony indicated that 50 percent of the worker's present pathology was due to industrial exposure; the remainder was due to smoking. The court held that medical testimony alone was not a reasonable basis for apportionment absent other evidence that the employee would have suffered because of his smoking habit, regardless of exposure to damaging substances in his work.[219]

Although applied under a statute worded differently from Maine's,[220] the rule of *Pullman-Kellogg* places the burden on the party seeking apportionment to show that the non-compensable condition would have resulted in eventual disability, regardless of industrial conditions. That showing would be no more difficult than showing, under Maine law, the proportion of the final disability that was caused solely by the occupational disease.

Another problem inherent in the multi-causation requirement of Maine's apportionment statute arose under the similarly worded apportionment section of the Maryland statute.[221] In *Blake v. Bethlehem Steel Co.*,[222] an iron worker claimed compensation under the occupational disease law[223] when his non-industrial bronchitis was aggravated by fumes and dust caused by the open-hearth furnace he tended. The Maryland court rejected his claim. The court reasoned that allowing an ordinary disease to become occupational due to industrial aggravation "would virtually read out of the statute the requirement that in order to support a claim . . . there must be a finding that, in part at least, the disability is due to an occupational disease, and the claim can be allowed only for that part."[224] The *Blake* holding graphically demonstrates the inequity of treatment afforded disease sufferers compared with that afforded injury victims. If Mr. Blake had been burned by molten metal or if his lungs had been traumatically damaged by his inhalation of fumes, his injury would have been compensable. The reasoning that the apportionment section bars his claim completely because there was no preexisting occupational disease to aggravate and thus none to apportion is mystifying.

---

218. 26 Cal.3d 450, 605 P.2d 422, 161 Cal. Rptr. 783 (1980).

219. *Id.* at 455, 605 P.2d at 425, 161 Cal. Rptr. at 786.

220. The California apportionment statute reads: "In case of aggravation of any disease existing prior to a compensable injury, compensation shall be allowed only for the proportion of the disability due to the aggravation of such prior disease which is reasonably attributed to the injury." CAL. LAB. CODE § 4663 (West 1955). *See also* ME. REV. STAT. ANN. tit. 39, § 185 (1978). For the text of § 185, see note 202 *supra*.

221. MD. ANN. CODE art. 101, § 22(c) (1957).

222. 225 Md. 196, 170 A.2d 204 (1961).

223. MD. ANN. CODE art. 101, § 22 (1957).

224. 225 Md. at 200, 170 A.2d at 206.

Blake also suggests the second major problem presented by apportionment statutes. In jurisdictions such as Maine, where cumulative injuries are compensable, apportionment of disability in *any* form is anomalous. The axiom that an employer takes the employee as he or she finds the employee is consistently upheld in injury cases heard by the Law Court. In *Wadleigh v. Higgins,*[225] the claimant suffered from gout and osteoarthritis of the spinal column; both conditions were proved to have predated the back injury for which he claimed compensation. In awarding compensation for total disability, the court cited Maine authority,[226] which, in effect, "requires that an employer compensate an employee who is disabled as a result of the interaction between a work-related injury and a preexisting but non-disabling injury or disease to the full extent of his incapacity even though the injury would not have so extensively disabled a healthy individual."[227] Even in heart cases, which lie between injury and disease, the court has found compensable injuries aggravated by preexisting infirmity. In *Canning v. State Department of Transportation,*[228] for example, the claimant was found to have suffered a fully compensable attack of angina, although medical testimony showed that the condition was partially due to preexisting coronary insufficiency, diabetes, mild hypertension, and probable arteriosclerotic heart disease.

The test of compensability in cumulative injury cases and cases involving aggravation of preexisting conditions involves the existence of a "substantial causal relationship" between the injury and the employment.[229] Preexisting disease or infirmity does not disqualify a claim if the employment aggravated, accelerated, or combined with the disease or infirmity to produce the death or disability for which compensation is sought.[230] The test for both types of cases is the same: the industrial hazard or risk that accelerates or aggravates the underlying condition must "arise out of and in the course of the employment"; if the test is satisfied, the entire resulting incapacity is compensable.

Because the aggravation or cumulative injury rule is widely accepted in compensation cases involving "injuries," the distinction re-

---

225. 358 A.2d 531 (Me. 1976).

226. Kidder v. Coastal Constr. Co., 342 A.2d 729 (Me. 1975); Kidder v. Coastal Constr. Co., 309 A.2d 119 (Me. 1973); Soucy v. Fraser Paper Ltd., 267 A.2d 919 (Me. 1970); Bradbury v. General Foods Corp., 218 A.2d 673 (Me. 1966).

227. 358 A.2d at 533.

228. 347 A.2d 605 (Me. 1975).

229. Beaulieu v. Francis Bernard, Inc., 393 A.2d 163 (Me. 1978); Richardson v. Robbins Lumber, Inc., 379 A.2d 380 (Me. 1977).

230. Ferris' Case, 132 Me. 31, 165 A. 160 (1933)(death alleged to have resulted from infection contracted through skin scratch held compensable, although conflicting medical evidence did not rule out other causes; death need not be shown to have resulted from sole source but may be concurrent result of accident and disease).

quired by apportionment in occupational disease cases is difficult to justify. In Michigan, a similar distinction was challenged by an employer who alleged that apportionment of occupational disease awards, as mandated by that state's statute,[231] must also apply to injury awards; the employer contended that the distinction was a denial of equal protection of the law. The Michigan court, in *Morgan v. Schuler's Restaurant*,[232] noted that equal protection guarantees prohibit entirely arbitrary discrimination. In order to withstand the constitutional challenge, "[t]here must be a relation between the classification and the purposes of the act in which it is found."[233] In denying the employer's equal protection claim, the *Morgan* court found the requisite relation. The court stated:

> A possible rationale for the distinction is that occupational diseases, by their nature, are caused by harmful conditions characteristic of particular industries while single-event injuries can occur in any occupation. Thus, the Legislature, in formulating the classification, may have felt that employers who engage in particular industries which have inherent harmful conditions need preferential workmen's compensation treatment. If these employers were not given this preferential treatment, conceivably they would not enter such industries since their workmen's compensation insurance would undoubtedly cost more.[234]

The *Morgan* court was correct in refusing to apply apportionment of disability to single-event injuries but its rationale for treating occupational disease cases differently was weak. An important goal of workers' compensation laws is to encourage safety in the workplace.[235] The system approaches this goal by insurance rate adjustment among industries with differing accident records. If the *Morgan* court was correct in holding that apportionment of occupational disease disability can be justified in order to give preferential treatment to higher-risk industries, the decision was contrary to the safety-improvement goal of workers' compensation.

Additionally, apportionment of disability among compensable and non-compensable causes or conditions in either single-event injury or occupational disease cases contradicts the theory and purpose of compensation law in a second sense. The hypothetical asbestosis sufferer illustrates the concept. Under the apportionment provisions, Roe's employer can successfully reduce the award of compensation by providing medical evidence that smoking exacerbated the lung

---

231. MICH. COMP. LAWS ANN. § 418.431 (Supp. 1981).

232. 64 Mich. App. 37, 234 N.W.2d 885 (1975).

233. *Id.* at 42, 234 N.W.2d at 887 (quoting Fox v. Employment Sec. Comm'n, 379 Mich. 579, 588, 153 N.W.2d 644, 647 (1967)).

234. 64 Mich. App. at 42, 234 N.W.2d at 888.

235. *See* 3 NATIONAL COMMISSION OF STATE WORKMEN'S COMPENSATION LAWS, SUPPLEMENTAL STUDIES (1973).

damage done by the industrial hazard.[236] Thus, Roe's smoking habit is similar to the defense of contributory negligence, ostensibly precluded by the *quid pro quo* of compensation law. If, however, some apportionment is justified by the facts of a case, California's *Pullman-Kellogg* rule[237] provides a more equitable treatment of occupational disease. That rule requires the employer to show by medical evidence that the disability claimed would have manifested itself in the *absence* of contributing industrial hazards. This construction does not address the basic conflict presented by the difference in apportionment between disease cases and injury cases. The rule, however, places the burden of justifying apportionment on industry, the party better able to bear that burden.

E. The "Special Problem" of Dust Diseases

Roe's claim for compensation due to asbestosis is governed by a section devoted to that disease.[238] Maine's treatment of asbestosis and silicosis[239] contains a negative presumption that, in the absence of evidence supporting the claim, these diseases will be presumed to be non-occupational in nature, unless the claimant can show at least two years of exposure to the dust hazards within the last fifteen years immediately preceding the onset of disability. Because Roe worked in the asbestos environment for only eighteen months and regardless of the fact that asbestosis is a fibrotic lung condition caused only by the inhalation of that substance, his claim will be denied under the strict wording of this section. The requirement of two years of exposure, placed in the law to "ease the load in the early years"[240] of such claims, ignores modern etiological evidence that the diseases can result from heavy exposures over significantly shorter periods of time.[241]

A related problem faced by Roe and other dust disease claimants is the requirement to show *incapacity* prior to compensation. A degenerative disease such as asbestosis may restrict breathing capacity

---

236. There is little doubt about the symbiotic relationship between asbestos and cigarette smoking. *See* U.S. DEP'T OF HEALTH, EDUCATION & WELFARE, A GUIDE TO THE WORK-RELATEDNESS OF DISEASE 23 (1976).

237. See text accompanying notes 218-220 *supra*.

238. ME. REV. STAT. ANN. tit. 39, § 194-A (1978) provides:

> In the absence of evidence in favor of the claim, disability or death from asbestosis shall be presumed not to be due to the nature of any occupation, unless during the 15 years immediately preceding the date of disability the employee has been exposed to the inhalation of asbestos dust over a period of not less than 2 years. If the employee shall have been employed by the same employer during the whole of such 2-year period, his right to compensation against such employer shall not be affected by the fact that he had been employed during any part of such period outside of this State.

239. *Id.* § 194.

240. COMMITTEE REPORT, *supra* note 90, at 18, 22.

241. I. SELIKOFF & D. LEE, *supra* note 190, at 177.

long before the symptoms prevent the performance of work. If the employee is aware that he has contracted a dust disease but is not yet incapacitated, he faces the further dilemma of choosing to find other work away from the hazard or continuing on the job until disability occurs. If the employee chooses to find other employment, he or she may still be without remedy if the eventual incapacity does not manifest itself within three years after the last injurious exposure to the dust hazard.[242]

Claimants diagnosed with silicosis or asbestosis, which has resulted in an incapacity to work, should face only the burden of showing a causal relation between exposure and the occupation, regardless of the time elapsed between exposure and disability. This is the same burden placed on any other injury or disease claimant. Assigning an arbitrary minimum exposure time increases the claimant's burden beyond that necessitated by the "arising out of and in the course of employment" test of compensability. The sections dealing with dust diseases were included, in slightly different forms, in the bill first recommended by the Recess Committee in 1938. At that time, the special restrictions were included to "ease the load" on employers and to prevent the entire collapse of dust-hazard industries following the revelations and litigation of the 1930's. That justification has outlived its usefulness. Although far from comprehensive, medical knowledge of these diseases now permits identification of those claims that are truly employment-related.

VI. Reforms Necessary in Maine's Occupational Disease Law

Richard Roe will probably not prevail on his claim for compensation under the present Law. His case reflects the striking difference between the compensability of industrial injuries and most industrial diseases. If an injury on his construction job had aggravated a preexisting but non-compensable osteoarthritic condition in his back, his disability would be completely compensable under Maine law.[243] No showing that such an injury had a cause "characteristic" of his particular employment would be required. Further, the injury would not be subject to apportionment based on the underlying preexisting infirmity. The injury would be compensable even though disability might not occur until years later. But because his claim is for disease rather than injury, he must meet several requirements of the Occupational Disease Law that go beyond the test of "arising out of and in the course of the employment."

Early resistance to assigning liability to the employer for diseases contracted on the job was perhaps justified in an era of medical ignorance. But today, the etiology of many occupational diseases is no

242. Me. Rev. Stat. Ann. tit. 39, § 189 (1978).
243. *See, e.g.*, Wadleigh v. Higgins, 358 A.2d 531 (Me. 1976).

longer a mystery; causation can be clearly linked to industrial hazards. Yet the occupational disease laws imposed on an unreceptive workers' compensation system have not kept pace with advances in medicine and science. Early restrictions protecting employers from unfounded or excessively expensive claims still hamper the adjudication of otherwise compensable claims. The problem with Maine's Law today was well expressed in a conclusion stated by the Industrial Accident Commission more than twenty years ago: "The occupational disease amendment to the law is not at all satisfactory and workable and we submit that it does not afford the relief it intended."[244]

VII. Proposal For Reform

Many suggestions have been made for substantial reform of the current compensation systems. Compensation for work-related diseases and illnesses must be put on a basis comparable to that for work-related injuries.[245] The following discussion suggests a few reforms necessary in Maine's Law.

A. Definition of Occupational Disease

In 1967, the Maine Legislature repealed the specific disease schedule and substituted general coverage for occupational diseases. The amended Law, however, still requires that the claimant prove that his disease is one caused by conditions "characteristic" of the particular employment. The causation-plus burden imposed by this ambiguous term places the disease sufferer at a disadvantage, compared with victims of industrial injuries whose injury must meet only the "arising out of and in the course of" test. The extra burden is justified only because it insures that the *risk* of disease will be more prevalent on the job than away from it. Yet for industrial injuries, this requirement of "actual risk" is met by application of the traditional test.

The Law's definition of occupational disease should be reworded to delete the "characteristic" requirement. Alternatively, the definition should be deleted completely and a definition of "injury" should be drafted to encompass disease. The Model Act of the Council of State Governments provides a guide to this definition:

> 'Injury' means any harmful change in the human organism arising out of and in the course of employment, including damage to or the loss of a prosthetic appliance, but does not include any communicable disease unless the risk of contracting such diseases is in-

244. Industrial Accident Commission, *supra* note 12, at 1.

245. *See, e.g.*, U.S. Dep't of Labor, Analysis of Current Laws Reflecting Worker Benefits for Occupational Diseases (1979); Edes, *supra* note 5.

creased by the nature of the employment.[246]

Because of the problems of identifying actual work connection in the majority of cases, which may result from such frequent occurrences as the common cold or influenza, the inclusion of an "increased risk" factor for communicable disease is reasonable. But the causal connection of the disability to the employment should be tested against the same "arising out of and in the course of the employment" standard used for injury.

B. *Time Limitations*

The Law's requirement that a claim must be filed within three years after the "last injurious exposure" effectively bars compensation for diseases that develop slowly, regardless of a close relationship to the employment. For example, even if Roe attempts to comply with this requirement, the date of his last injurious exposure is unknown. Such an artificial and arbitrary provision has no place in workers' compensation law. If removal of the requirement places an undue burden on certain employers or carriers, an alternative exists in schemes such as the Second Injury Fund[247] or special funds such as that established under the federal Black Lung Act.[248] Similar criticism applies to the ten-year statute of limitation imposed by the Workers' Compensation Act. Both statutes penalize the unfortunate employee who has contracted a latent disease.

C. *Determining Proof of Causation*

The employee who can prevail under the restrictive definition of occupational disease and the impact of statutes of limitation still must show causal connection. Unlike the Workers' Compensation Act's treatment of injury claims, in which a liberal construction often favors the employee, the Occupational Disease Law shifts virtually the entire burden of proof to the disease victim. A fairer balance between employer and employee should be used. One step toward that balance is the use of a rebuttable presumption, such as that applied to firefighters;[249] a presumption of causation is established in certain high-risk industries absent evidence refuting the claim. Despite this precedent, Maine's provisions regarding dust diseases have the opposite effect. The provisions should be repealed. A provision allowing a claimant who can show a minimum exposure to

---

246. COUNCIL OF STATE GOVERNMENTS, WORKMEN'S COMPENSATION AND REHABILITATION LAW § 2(a) (rev. ed. 1974).

247. ME. REV. STAT. ANN. tit. 39, § 57 (Supp. 1981-1982).

248. 30 U.S.C. §§ 901-941, 951, 958 (1976)(amending 30 U.S.C. §§ 901-936, 951, 958 (1970)). For a discussion of the history and operation of this compensation program, see Solomons, *supra* note 84.

249. ME. REV. STAT. ANN. tit. 39, §§ 64-B-64-C (Supp. 1981-1982).

certain hazards a presumption that his disease *is* causally related should replace the repealed provisions. The use of presumptions by either side should be limited, however, to those categories of cases in which the presumptions are essential to achieve prompt resolution of the dispute and adequate compensation for the loss. Such categories include latent and degenerative diseases and those prevalent in industries utilizing carcenogenic substances.

D. The Effects of Prior Disability

Because an employer takes the employee as he or she finds the employee, aggravation of preexisting conditions or acceleration of disability by a prior infirmity is compensable under the Workers' Compensation Act. Reducing a disease claimant's award by apportionment among preexisting but non-compensable factors is anomalous. Apportionment resurrects the defense of contributory negligence and possibly offends constitutional guarantees of equal protection. As applied, the Law's apportionment provision, coupled with the bar to disabilities not arising within three years after the last exposure, can result in a shift of the burden of compensation to social security or welfare programs later in the worker's life. The apportionment provision should be repealed. Complicated cases involving preexisting, non-compensable disability should be referred to the Second Injury Fund or other programs designed to protect the employers, while spreading the cost of compensation throughout the industry.

VIII. Conclusion

Compensation for work-related injuries was created by social conscience and administered under recognized principles of common law. Compensation for work-related diseases, however, is an unwelcome and unwanted bastard-child of the same system. The two types of compensation can coexist and the system can provide a measure of assured relief for wages lost due to work-related disability. But this goal can be achieved only if the basic principles of workers' compensation law apply to occupational diseases as well as occupational injuries. Justifications for treating occupational diseases differently from other work-related disabilities have long outlived their rationales. The time for reform has come.

Thomas R. Watson

ATTACHMENT G

HealthWORKS
OCCUPATIONAL & SPORTS MEDICINE

12 Stillwater Avenue • Bangor, Maine 04401 • (207) 262-1830 • FAX (207) 262-1919

February 4, 1998

Heather Henderson
Legislative Analyst
Maine State Legislature-Office of Policy and Legal Analysis
13 State House Station
Augusta, Maine 04333

Dear Ms. Henderson:

Thank you for the opportunity to respond to the Joint Standing Committee on Labor's need to gather information and recommendations to improve the reporting of occupational diseases and exposures in Maine. This information is vital to the health and safety of our workers and should be available to our lawmakers when considering new or revised legislation.

Specifically, I agree that occupational diseases and exposures are significantly under-reported. This stems from the confusion over the definition of occupational disease and lack of understanding of the reporting system by many health care providers.

A good starting point is a unified definition for the Worker's Compensation Board, and the Bureau of Health. I would propose the use of O.S.H.A.'s broader definition with the following editing(in [ ]'s):

"An occupational illness [or disease] of an employee is an abnormal [health] condition or disorder, [excluding] one resulting from an occupational injury , caused by exposure to [specific] environmental factors [arising out of or in the course of] employment. It includes acute and chronic illnesses or diseases which may be caused by inhalation, absorption, ingestion or direct contact."

1

HealthWORKS
OCCUPATIONAL & SPORTS MEDICINE
Phone: (207) 262-1830
Fax: (207) 262-1919
CCurtis443@AOL.COM

Clinical Aspects of Occupational Medicine

Craig W. Curtis, M.D.
Medical Director
Certified Medical Review Officer

900 Broadway, Bangor, Maine 04401
. . . Keeping Maine healthy at work and play . . .

Harold R. Imbus, M.D., Sc.D.

Although occupational medicine is considered a type of preventive medicine, clinical problems arise frequently and are a major concern of most physicians working in this field. Occupational diseases, rehabilitation, and problems of placement of persons with disabilities have not received the same attention from researchers and clinicians as the nonoccupational problems. Therefore, the physician working in this field often has difficulty finding information, either because relatively little has been written or because of an absence locally of available literature. Consultants experienced in occupational problems often are not available. Furthermore, although clinical problems often are simple for the treating physician, for the patient work requirements, psychologic factors, and interpersonal relationships at work can magnify them to major proportions. For these reasons, occupational clinical conditions can prove to be difficult and even burdensome; however, they provide the conscientious physician with one of the finest challenges of medicine, namely, the opportunity to influence an individual's adjustment, rehabilitation, or longevity in his work.

OCCUPATIONAL DISEASES

Problems in Diagnosis

Occupational diseases are caused by a pathologic adaptation of the patient to his working environment; therefore, in order to properly diagnose occupational diseases (or any diseases caused by environment for that matter), the physician must evaluate both patient and environmental exposure. Very few occupational diseases present with specific pathognomonic, clinical, or laboratory findings. Thus, the anemia of benzene intoxication, the peripheral neuritis of acrylamide poisoning, the bronchitis of byssinosis, the fibrosis of asbestosis, the granuloma of berylliosis, the nodulation of silicosis cannot be adequately diagnosed as to etiologic agent from clinical and laboratory findings* alone. Only with knowledge of exposure, in addition to clinical factors, can an accurate diagnosis be made. Obtaining adequate environmental data and weighing their importance as causative factors can be an extremely difficult problem for the practicing physician, especially for one not experienced in this. Nevertheless, by applying the principles described herein, the practicing physician frequently can make an accurate diagnosis or, in more difficult cases, a preliminary evaluation prior to referral to other specialists.

The physician should recognize emotional factors that may complicate the diagnosis and treatment of the occupational disease patient. Depending on their feelings toward their employer, these patients may manifest hostility and anger, depression, concern for their ability to continue to work, or rejection. They may believe that they have given a great deal for an unappreciative and neglectful employer who is deserting them now that their work has caused them to become ill. The more enlightened and understanding the attitude of the employer, and the more positive the preexisting relationship has been, the less likely are these negative feelings to develop; nevertheless, they are quite common even in the best of circumstances. They may lead patients to arrive at illogical conclusions and to prediagnose their condition as occupational. These patients then may become quite suspicious of anyone who does not agree with their premises. These feelings are quite common, do not necessarily indicate malingering, and should not create a negative attitude on the part of the physician.

Unfortunately, in many cases there is a significant differential in indemnity between workers' compensation and other forms of insurance, resulting in a powerful economic motive for the disease to be designated occupational. Third parties, representing either the employee or the employer, may create pressure for their viewpoint, making objectivity even more difficult. Physicians should guard against a tendency toward bias. The personal physician of the employee may feel pressure to diagnose an occupational etiology, whereas the physician representing the employer may feel the opposite. The physician, although empathizing with either viewpoint, nevertheless must evaluate the facts as a true professional and detach himself from external pressures.

*Lung biopsy, which may be of value in the last three, is discussed in Chapters 13, 14, and 33.

from: Occupational Medicine 3rd Edition 1994 - Zenz

occupational medicine, quantitative aspects of safe exposure are expressed in the concept of the TLVs, a time-weighted average exposure. Details of TLVs are described elsewhere in this volume (see Chapter 70 and Appendix A). TLVs have recently come under criticism. The Occupational Safety and Health Administration (OSHA) has established permissible exposure levels (PELs) for 428 substances. Also, the National Institute for Occupational Safety and Health (NIOSH) has issued recommended exposure limits (RELs), which are often lower than the TLVs.

How can the physician not specializing in occupational medicine obtain the information described above? It can be difficult and time consuming; nevertheless, in most cases it is not impossible and can be most rewarding to the physician who is concerned with making adequate diagnoses. Information about occupational exposure generally is obtained from

1. The occupational history of the patient
2. Industrial hygiene data

Occupational history. If the history is to establish exposure or lack thereof to the suspected substance or agent, it can be quite detailed and time consuming; however, for the busy physician, this often is not practical. Time can be saved by focusing on the questions that will be the most productive of information.

The physician should determine where the patient works and how long he has worked in this place. This alone may be helpful, especially in small communities in which physicians have general knowledge of the industries in the area. If the physician is not familiar with the company or plant, he should determine what the product is. It is important to be as specific as possible concerning this. For example, in textiles, it will make a great deal of difference whether the product is cotton textiles or one of the various types of synthetics. If the company makes castings, are these from iron or brass? If it makes metal products, does it merely assemble the products or does it actually mold the metal in question? Other general information that is helpful is that concerning the company's safety and hygiene practices—whether the company has an occupational health program and whether the employees are given periodic health examinations and preplacement examinations.[5]

The above information is helpful in formulating a general impression of possible exposures. Questioning, then, should become more specific to determine exactly what the patient's job is. Often, he or she will use terminology to describe the job that may be quite foreign to the physician unless the physician has had experience with the particular type of trade in question. Although it often is quite difficult to determine specifically what the patient does without viewing the operation, much information often can be obtained directly from the patient by asking him or her to specifically describe the operation, perhaps to reproduce the motions involved and to describe the various materials with which he works. For example, if work is done on a conveyor line, the physician may assume that a great deal of lifting and pushing is involved. This may or may not be true. Specific questions concerning the size and approximate weight of objects, height above the floor level from which and to which they are lifted, distance carried, and the frequency of lifts will clarify these requirements.

Description of the job should include the materials with which the employee works. The employee may or may not know the materials with which he or she is in contact. Awareness on the part of the employee has increased with implementation of the Occupational Safety and Health Act, hazard communication standards requiring labeling of containers and informing employees of toxic substances with which they are working. Often, the patient will only know of a trade or slang name for the substance in question. If such is the case, the physician may be able to obtain more specific information from the employer or, short of this, have the employee bring the specific trade name and the address of the manufacturer. Under the new standard, manufacturers are required to supply information concerning the contents of their products on the request of medical and industrial hygiene personnel.

The patient should describe the form in which the substance is and the type of contact that he or she has. Is it a liquid? If so, does it emit vapors that were inhaled? Was there a skin contact? Is any protection provided, and, if so, how effective does the protection appear to the patient? For example, if he or she is wearing a respirator, does the company have a program for its maintenance? Has there been instruction in proper cleaning and maintenance or is this provided? Is he or she able to smell the chemical through the respirator? Does the respirator appear to have a proper fit?

The employee should be questioned as to whether other employees are experiencing a similar problem. It is important to determine how the symptoms relate to work. With an occupational disease characterized by acute symptoms, one would expect exacerbations of symptoms in relationship to work and some degree of remission during weekends and vacation periods. Some exposures cause symptoms so acute that they are noticed shortly after the beginning of the workday. Others may result in acute but delayed symptoms that occur after the employee is home at night. Byssinosis is characterized by the former, whereas exposure to Canadian red cedar and zinc fumes may result in the latter. When chronic disease is the result of the exposure, symptoms may appear gradually during a period of weeks, months, or even years after beginning employment. They may or may not improve with temporary removal from employment. In this respect, it is important to determine whether there were any similar symptoms prior to employment. Quite often, an occupational exposure will provide an aggravation to a preexisting condition or a latent but developing condition. This is especially true with exposure to pneumoconiosis-producing agents and pulmonary irritants when there is preexisting subclinical chronic obstructive lung disease.

Finally, the physician should determine what other stresses and exposures there are other than those that are job related. The role of smoking is a very important factor in many occupational lung diseases. Smoking provides an effect in addition to the occupational exposure. Noise exposure frequently results from hobbies or a second job. In my experience, many cases of noise-induced hearing loss have been seen in relatively young men who are coming into a noisy industry for the first time. Often, the history of noise exposure in the military service, on the farm, from driving heavy mechanical equipment, or from shooting rifles can be elicited in these cases.

Industrial hygiene data. The occupational history of the employee can give valuable insight into the possible etiology of his or her condition. However, a more precise indication of exposure can be obtained from industrial hygiene surveys of the work site. Details of this approach are provided in Chapter 70. The practicing physician should be familiar with ways to obtain these data. Large companies often have an industrial hygiene staff and, if so, they often are quite willing to supply the practicing physician with any necessary data.

More detailed information may be obtained through the company, which can supply the specific names and types of chemicals or physical agents and a rather detailed description of the process involved. Practicing physicians who are dealing with these types of cases will benefit themselves and their patients if they become familiar with the operations in the local industry. Many industries are happy to have physicians visit and learn op-

ten presents a most difficult problem for the patient. Needless to say, it is not something that should be undertaken lightly; indeed, permanent removal should be a last resort, especially in the more highly skilled jobs.

This question should be answered: Is this individual highly susceptible to the exposure, making it unlikely that reasonable controls will effect the desired relief? If so, attempts to keep the person in the same job are likely to be unsuccessful. However, quite frequently the exposure of the individual is unwarranted and unnecessary, and adjustments in the environmental situation can bring about relief. If an industry has employees who are unduly exposed to a harmful agent, it should make every effort to control this to the degree necessary to bring exposure to levels acceptable by current standards. The physician may be able to assist the patient by discussing this with company officials so that exposure can be minimized. It has been my experience that, frequently, relatively minor and inexpensive changes can bring about a greatly reduced exposure. Principles of control, namely, ventilation, enclosure, shielding, or work practices, are discussed in other chapters.

The physician should consider the seriousness of the condition in the light of the consideration of removal versus environmental control. Obviously, an irreversible condition that will progress even with minimal exposure warrants a change in job; however, self-limited or acute conditions that usually are completely reversible may be handled by limiting future exposure. In these cases, the employee usually can be assured that no permanent damage has occurred and that if the exposure is brought under reasonable control, recurrences of the condition should not occur.

Protective devices are another means of control of exposure. There are problems in their use, and they are not considered acceptable means of control of exposure over long periods of time when feasible engineering controls are available. Also, it is not infrequent that protective devices, especially negative-pressure respirators, are most difficult to wear for those who need them most. For example, an employee exhibiting chronic obstructive pulmonary disease would be most affected by exposure to dust and would be in most need of wearing a respiratory protective device. However, resistance to respiration is increased by the respirator, and since the employee already is impaired, additional resistance to respiration often is not well tolerated and is quite fatiguing. Some of the newer air-supplied respirators may be helpful in such cases.

The use of protective devices can be most helpful in selected cases, especially where short-term or intermittent usage is required because of heavy intermittent exposure. For example, I once was asked to evaluate a textile employee who had worked all his life with dyes. The employee was a heavy smoker and had developed emphysema and chronic bronchitis. He also had noticed in recent years that his respiratory symptoms had been increasingly aggravated by his work with dyes. The dust from these dyes would produce cough, sputum, and shortness of breath. He was advised by his family physician to obtain work elsewhere. Since the employee was highly skilled in this area, he would have had to take a significant reduction in wages, leave a job that he thoroughly enjoyed, and, in middle age, learn another skill. He questioned management about any possible way in which he might stay in his present job. Careful questioning by me revealed that it was only occasionally that he sustained exacerbations of his cough and dyspnea when exposed to dye dust. This was in relation to the use of several very dusty dyes of an irritating nature. He indicated that this particular mixing process occurred only once or twice a week. Since his obstructive lung disease was not advanced, he was able to wear respiratory protection during the limited periods when he was mixing the offending dyes. This protection, combined with measures taken to treat his underlying condition, namely, cessation of smoking and administration of antibiotics, enabled him to continue working on this job without difficulty.

The employee suffering from an occupational disease needs whatever reassurance the physician can give. Disease, of course, is always a threat to most individuals. Occupational disease not only threatens one's health but, in particular, offers an ominous threat to one's ability to continue to provide for one's self and family. Since it was the job that caused the sickness, the employee often will jump to the conclusion that he or she no longer can perform on that job. These negative thoughts and concerns interfere with recovery and rehabilitation. Utilizing the principles described above, it quite often is possible for the physician to reassure the employee that returning to work is possible. A great deal, of course, depends on the employer's attitude, willingness to control the environmental conditions that created the condition, and overall attitude toward the employee. When the practicing physician has developed the proper rapport with employer and employee, he or she is in a very strong position to intercede and to produce a favorable climate for recovery and rehabilitation. Every care must be taken that the physician and the patient do not come to premature conclusions concerning the patient's inability to continue working. Where it is obviously apparent that the patient cannot continue on his present job but will be fully capable of engaging in other types of activity, he should be strongly reassured of this at an early stage in the treatment.

MEDICAL PLACEMENT

Until the Americans with Disabilities Act (ADA) became effective (July 26, 1992), preplacement or preemployment examinations were common. Now the employer must make a job offer before an examination can be done. This is now called the employment entrance examination.

Employment Entrance Examination

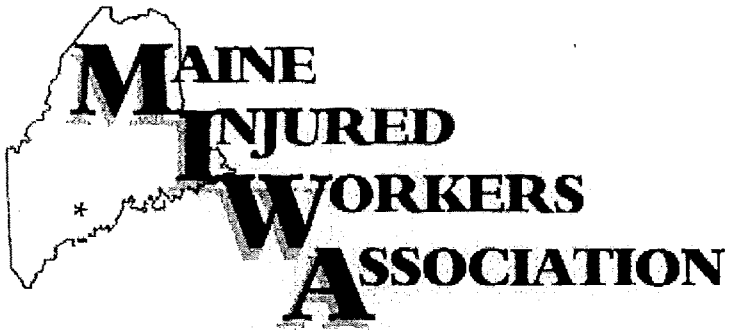
The value of this examination is as follows:

1. To determine the individual's physical and emotional capacity to perform a particular job.
2. To assess the individual's general health.
3. To establish a baseline record of physical condition for epidemiologic and medicolegal purposes.

Determination of Physical and Emotional Capacity for Job Performance

This has been and should continue to be the primary purpose of performing the employment entrance examination. Theoretically, individuals can be matched to specific jobs according to aptitude and physical and emotional capabilities. If the match is correct, presumably a happy, healthy employee will result; if incorrect, the employee will not succeed, because of a lack of physical or mental capacity, or perhaps some illness will develop or preexisting illnesses will worsen as a result of the work, or the employee will leave the job. Although there appears to be some theoretical basis and experience to substantiate the above, many previously held concepts are without adequate statistical validation. The necessity to evaluate physical capacity arose as a need to determine that individuals could do work that frequently re-

ATTACHMENT H



Chemically Injured Workers Ignored, Disenfranchised and Thrown Away....

OVERALL PROBLEM

For the last 10 years or more, the states and many employers have systematically bent, abused and eradicated state worker compensation laws. In addition, both the medical and legal systems are either unable to help chemically injured workers or they are part of the system that is abusing the worker's rights. In most states, laws have made it unprofitable for an attorneys and statue of limitations run out. Even more so in the case of chemical exposure. The erosion of the comp system's main purpose, which is to assist and rehabilitate workers who are hurt on the job, has been distorted so badly that now many workers cannot even get into the system to begin recovery. Chemically injured workers are the fastest growing disenfranchised group of injured workers in the United States.

These workers are the new poor and even homeless, some are on public assistance but many do not qualify for it. Most struggle to get SS disability and are left trying to piece their lives together in anyway they can. A once stable family then falls apart and the injured person is then burdened again by family stress. Blue collar or minimum wage workers are particularly venerable and terribly exploited in our society. The work environment does not have the security it once had and so therefore employees are losing benefits and the safety nets that once gave them an opportunity to protect themselves. People can be exposed to life threatening or debilitating toxic substances in manager

positions as well as janitorial positions. It isn't just the chemical factory worker that is susceptible to injury anymore. This cycle of abuse can only be stopped if workers know their rights before they are injured, have real medical, legal, technical and organize, change unfair laws and the institutions that are taking away or ignoring their rights.

Our Northeast Project

The Environmental Health Network, a national non-profit that assists communities and workers who have been harmed by toxic exposure, has been working with a fledgling struggling group of injured workers in Maine. The group's name is Maine Injured Workers Association. Our connection with them has been through 2 very persistent and wonderful men, Richard Pushard and Richard Bean. They both have brain damage from work place chemical exposure. In the last two years, Mr Bean and Mr. Pushard have exhausted personal income so they could uncover damning evidence against the state on corruption, have had numerous news paper articles in the newspaper and have begun to bring in other workers from across the state and even in other parts of New England. They are now ready to formally organize their group within Maine and perhaps widen it at some point to a regional group. Maine has some of the most damaging and distorted laws in the United States. Some are even being examined for their constitutionality. For example, by law a worker cannot get a medical expert to testify in his behalf if the expert is outside the state of Maine!!

Goals

EHN's goals are to travel to Maine for two large organizing and training sessions. To have at least 6 conference calls with leadership. To help the group formally organize, incorporate and get 501 C3 status. To do organizing outreach to get more workers involved. To help them formalize a web site to tell individuals stories and build a strong campaign to begin the process of institutional change within the state of Maine and perhaps regionally. By the end the year we are wanting to have a firmly established group that has growing membership, networking with other injured worker groups elsewhere and develop a campaign so that they can begin implementation at the end of the first and continued into the second year.

The training sessions will consist of developing a democratic well structured group, how to do outreach to current membership and new membership, fund raising, and campaign building. Our ultimate goal is to build in self-sufficiency of the group and to provide a model for other groups of workers through out the United States.

Why EHN? We have been working with Chemically Injured Workers for over 9 years. We have the technical expertise, networking capabilities, accumulate knowledge and dedication to help these workers become a force for justice. We collaborate with the Injured Workers Union in LA on some projects - such as our book - ***Job Damaged People: How to Survive and Change the Workers Comp System.***

Linda King - Director and Founder

To learn more about MIWA and listen to other injured workers' stories, check out our other pages above.

The E-mail address for the Maine Injured Workers Association is:
miwa@miwa.org

The mailing address for the Maine Injured Workers Association is:
P.O. Box 85
West Paris, Maine 04289-0085

The web site address for the Maine Injured Workers Association is:
http://www.miwa.org

For more imformation about The Environmental Network, visit their website at:
EHN33@aol.com

We have a new address to add to our site for resources, training and other types of work related injuries at the Canadian Injured Workers Alliance:
http://www.ciwa.ca

You are number  to arrive

This web site updated, Wednesday, December 2, 1998.

39A § 606. Date from which compensation is compute

The date ~~when an employee becomes incapacitated by an occupational disease from performing the employee's work in the last occupation in~~ on which the employee was last injuriously exposed to the hazards of the occupational disease is the date of the injury equivalent to the date of injury under the former Workers' Compensation Act or the Maine Workers' Compensation Act of 1992. When compensation is payable for an occupational disease, the employer in whose employment the employee was last injuriously exposed to the hazards of the occupational disease and the insurance carrier, if any, on the risk when the employee was last exposed under that employer, are liable. The amount of the compensation must be based on the average wages of the employee when last exposed under that employer and notice of injury and claim for compensation must be given to that employer. The only employer and insurance carrier liable are the last employer in whose employment the employee was last injuriously exposed to the hazards of the disease during a period of 60 days or more and the insurance carrier, if any, on the risk when the employee was last so exposed, under that employer. [1995, c. 462, Pt. A, §81 (amd).]

Section History:
1991, c. 885, § A8 (NEW).

1991, c. 885, § A9-11 (AFF).

1995, c. 462, § A81 (AMD).

39A § 607. Notice of incapacity; filing of claim

Sections 301 to 307 with reference to giving notice, making claims and filing petitions apply to cases under this chapter, except that, in cases under this chapter, ~~the date of incapacity defined in section 606 is equal to the date of injury in sections 301 to 307, and t~~ notice must be given within 90 days after the date of incapacity. The notice under section 301 must include the employee's name and address, the nature of the occupational disease, the date of incapacity, the name of the employer in whose employment the employee was last injuriously exposed for a period of 60 days to the hazards of the disease and the date when employment with that employer ceased. After compensation payments for an occupational disease have been legally discontinued, claim for further compensation for that occupational disease not due to further exposure to an occupational hazard tending to cause that disease are barred if not made within one year after the last previous payment. [1991, c. 885, Pt. A, §8 (new); §§9-11 (aff).]

Section History:
1991, c. 885, § A8 (NEW).

1991, c. 885, § A9-11 (AFF).

ATTACHMENT J

ENVIRONMENTAL MEDICINE

Integrating a Missing Element into Medical Education

Andrew M. Pope and David P. Rall, Editors

Committee on Curriculum Development in Environmental Medicine

Division of Health Promotion and Disease Prevention

INSTITUTE OF MEDICINE

NATIONAL ACADEMY PRESS

Washington, D.C. 1995

COMMITTEE ON CURRICULUM DEVELOPMENT IN ENVIRONMENTAL MEDICINE

DAVID P. RALL (Chair), Director (Retired), National Institute of Environmental Health Sciences, Washington, D.C.

M. BROWNELL ANDERSON, Assistant Vice President, Educational Programs, Association of American Medical Colleges, Washington, D.C.

ELIZABETH L. BOWEN, Assistant Professor, Department of Family Medicine and Medical Education, Morehouse School of Medicine, Atlanta

L. THOMPSON BOWLES, President, National Board of Medical Examiners, Philadelphia

EDDY A. BRESNITZ, Director, Division of Occupational and Environmental Health, and Professor and Chair of Community and Preventive Medicine, Medical College of Pennsylvania and Hahnemann University School of Medicine, Philadelphia

MARK R. CULLEN, Director, Occupational and Environmental Medicine Program, and Professor of Medicine and Public Health, Yale University, New Haven

RICHARD J. JACKSON,* Chief, Division of Communicable Disease Control, California Department of Health Services, Berkeley

KARL T. KELSEY, Associate Professor, Occupational Health Program, Harvard School of Public Health, Boston

HOWARD M. KIPEN, Associate Professor and Director of the Division of Occupational Medicine, Environmental/Occupational Health Sciences Institute, University of Medicine and Dentistry of New Jersey-Robert Wood Johnson Medical School, Piscataway, New Jersey

SHARON L. MORRIS,** Senior Lecturer and Director, Continuing Education, Department of Environmental Health, University of Washington, Seattle

KATHLEEN M. REST, Assistant Professor, Department of Family and Community Medicine, University of Massachusetts, Worcester

DONALD E. WILSON, Dean, University of Maryland School of Medicine, University of Maryland, Baltimore

Liaison to the Institute of Medicine's Board on Health Promotion and Disease Prevention

EULA BINGHAM, Professor of Environmental Health, College of Medicine, University of Cincinnati, Ohio

Study Staff

Andrew M. Pope, Study Director

Carrie E. Ingalls, Project Assistant

Michael A. Stoto, Director, Division of Health Promotion and Disease Prevention

Linda A. DePugh, Administrative Assistant

Judith Doody, Financial Associate

Laura Baird, Librarian

---

*Appointment ended September 1, 1994.

**Appointment ended May 1, 1994.

TABLE D-3: Selected Job Categories, Exposures, and Associated Work-Related Diseases and Conditions

Job Categories	Exposures	Work-Related Diseases and Conditions
Agricultural workers	Pesticides, infectious agents, gases, sunlight	Pesticide poisoning, "farmers' lung," skin cancer
Anesthetists	Anesthetic gases	Reproductive effects, cancer
Animal handlers	Infectious agents, allergens	Asthma
Automobile workers	Asbestos, plastics, lead, solvents	Asbestosis, dermatitis
Bakers	Flour	Asthma
Battery makers	Lead, arsenic	Lead poisoning, cancer
Butchers	Vinyl plastic fumes	"Meat wrappers' asthma"
Caisson workers	Pressurized work environments	"Caisson disease," "the bends"
Carpenters	Wood dust, wood preservatives, adhesives	Nasopharyngeal cancer, dermatitis
Cement workers	Cement dust, metals	Dermatitis, bronchitis
Ceramic workers	Talc, clays	Pneumoconiosis
Demolition workers	Asbestos, wood dust	Asbestosis
Drug manufacturers	Hormones, nitroglycerin, etc.	Reproductive effects
Dry cleaners	Solvents	Liver disease dermatitis
Dye workers	Dyestuffs, metals, solvents	Bladder cancer, dermatitis
Embalmers	Formaldehyde, infectious agents	Dermatitis
Felt makers	Mercury, polycyclic hydrocarbons	Mercuralism
Foundry workers	Silica, molten metals	Silicosis
Glass workers	Heat, solvents, metal powders	Cataracts
Hospital workers	Infectious agents, cleansers, radiation	Infections, accidents
Insulators	Asbestos, fibrous glass	Asbestosis, lung cancer, mesothelioma

Continued

TABLE D-3: Continued

Job Categories	Exposures	Work-Related Diseases and Conditions
Jack hammer operators	Vibration	Raynaud phenomenon
Lathe operators	Metal dusts, cutting oils	Lung disease, cancer
Laundry workers	Bleaches, soaps, alkalies	Dermatitis
Lead burners	Lead	Lead poisoning
Miners (coal, hard rock, metals, etc.)	Talc, radiation, metals, coal dust, silica	Pneumoconiosis, lung cancer
Natural gas workers	Polycyclic hydrocarbons	Lung cancer
Nuclear workers	Radiation, plutonium	Metal poisoning, cancer
Office workers	Poor lighting, poorly designed equipment	Joint problems, eye problems
Painters	Paints, solvents, spackling compounds	Neurologic problems
Paper makers	Acids, alkalies, solvents, metals	Lung disorders, dermatitis
Petroleum workers	Polycyclic hydrocarbons, catalysts, zeolites	Cancer, pneumoconiosis
Plumbers	Lead, solvents, asbestos	Lead poisoning
Railroad workers	Creosote, sunlight, oils, solvents	Cancer, dermatitis
Seamen	Sunlight, asbestos	Cancer, accidents
Smelter workers	Metals, heat, sulfur dioxide, arsenic	Cancer
Steel workers	Heat, metals, silica	Cataracts, heat stroke
Stone cutters	Silica	Silicosis
Textile workers	Cotton dust, fabrics, finishers, dyes, carbon disulfide	Byssinosis, dermatitis, psychosis
Varnish makers	Solvents, waxes	Dermatitis
Vineyard workers	Arsenic, pesticides	Cancer, dermatitis
Welders	Fumes, nonionizing radiation	Lead poisoning, cataracts

SOURCE: *Principles and Practice of Environmental Medicine*, A.B. Tarcher, ed., Plenum, New York, 1992.

REASON FOR REPORT CIRCLE ONE INITIAL PROGRESS FINAL	**M-1** **PRACTITIONER'S REPORT** **STATE OF MAINE** **WORKERS' COMPENSATION BOARD** **Office of Medical Rehabilitation Services**	**ATTACHMENT K** DC LIST OTHER _____

EMPLOYER NAME:	EMPLOYEE LAST NAME:	FIRST NAME:	M I.:
EMPLOYER MAILING ADDRESS & PHONE #:	ADDRESS - NUMBER AND STREET:		
INSURER NAME:	CITY: / STATE: / ZIP: / HOME PHONE:		
INSURER MAILING ADDRESS:	DATE OF INJURY:	SSN:	
PATIENT'S COMPLAINTS:			

ICD-9 CODE: _____

IN MY OPINION, THIS PROBLEM IS ☐ WORK RELATED ☐ NOT WORK RELATED ☐ IS NOT YET IDENTIFIED AS TO CAUSE

HAVE DIAGNOSTIC TESTS BEEN PERFORMED? ☐ YES ☐ NO RESULTS: _____

DATE OF THIS EXAMINATION: / / IS TREATMENT TO CONTINUE? ☐ YES ☐ NO

DATE PATIENT TO BE SEEN AGAIN: / / ESTIMATED LENGTH OF TREATMENT? _____

TREATMENT PLAN: _____

LIST ANY MEDICATION PRESCRIBED FOR THIS DIAGNOSIS/CONDITION THAT WOULD PREVENT YOUR PATIENT FROM DRIVING AND/OR WORKING SAFELY:

IF UNABLE TO WORK, ADVISE ESTIMATED DATE OF RETURN: / /

WORK CAPACITY: ☐ REGULAR DUTY ☐ MODIFIED DUTY ☐ NO WORK CAPACITY

RESTRICTIONS YES/NO	DESCRIBE:

SIGNATURE OF PRACTITIONER _____ PRINT NAME AND ADDRESS _____

TELEPHONE #: _____ NARRATIVES ATTACHED? ☐ YES ☐ NO

WCB M-1 (7/98) DISTRIBUTION: PRACTITIONER (1) EMPLOYEE (2) EMPLOYER (3) INS. COMPANY (4)

MAINE OCCUPATIONAL DISEASE REPORTING PROGRAM

ATTACHMENT K

Please complete this form on all patients with a reportable occupational disease.
Return form to: Occupational Health Program, Bureau of Health
SHS #11
151 Capitol St.
Augusta, ME 04333
For any questions: (207) 287-5378

NAME: _____
ADDRESS: _____
_____
PHONE # _____

PATIENT NAME (Last) (First) (Middle) (Maiden or aliases)

PATIENT'S ADDRESS AT DIAGNOSIS (Street, City, State, Zip Code)

RACE (check one)	SOCIAL SECURITY NUMBER	DATE OF BIRTH (Month, Day, Yr)	SEX (check one)
☐ Caucasian/White ☐ Black ☐ American Indian ☐ Asian ☐ Unknown ☐ Other _____		/ /	☐ 1 Male ☐ 2 Female

Does patient currently smoke? ☐ No ☐ Yes **If yes,** how many pack(s) a day? _____

Is there any reason we should not contact this patient directly? ☐ OK to contact patient
☐ Please do not contact the patient for the following reason(s):

PATIENT'S TELEPHONE NUMBER (including area code)

OCCUPATION	INDUSTRY
For fishers, please indicate the method of fishing employed, e.g. diving, trawling, digging, gillnetting, dredging, etc.	For fishers, please indicate the type of fish caught or harvested, e.g., scallops, lobster, haddock, etc.

NAME OF EMPLOYER and ADDRESS

TELEPHONE NUMBER OF EMPLOYER (including area code)

REPORTABLE DISEASE (please check) ☐ Diagnosed ☐ Suspected Date of Diagnosis _____
Date of Service _____

Check all that apply.

☐ Agriculturally -related injury (includes farming, logging, and fishing) . **Please describe how injury occurred, and the physical findings of the injury.**

☐ Asbestosis
☐ Byssinosis
☐ Carpal Tunnel Syndrome
☐ Heavy Metal Poisoning ☐ Lead (level) ____ ☐ Mercury (level) ____ ☐ Arsenic (level) ____ ☐ Cadmium (level) ____
☐ Hypersensitivity Pneumonitis (caused by _____)
☐ Mesothelioma
☐ Occupational Asthma (caused by NOISE INDUCED HEARING LOSS)
☐ Outbreaks (agent _____)
☐ Pesticide Poisoning (name of pesticide _____)
☐ Silicosis RADIATION EXPOSURE
☐ Solvent Toxicity (name of solvent _____)
☐ Toxic Gas Poisoning (name of gas _____)
☐ Other (please describe) _____

PLEASE CHECK ONE OF THE FOLLOWING: ☐ Work-Related ☐ Not Work-Related ☐ Suspect Work-Related ☐ Unknown

COMMENTS:

FORM COMPLETED BY: **DATE:**

CONFIDENTIAL INFORMATION

Please return white copy – retain yellow copy for your files

Executive Summary

A pregnant woman voices a concern to her obstetrician during a routine prenatal visit. It seems that several women in her neighborhood have recently had babies with a variety of birth defects. She worries that the recently discovered well-water contamination in her community may be responsible, and she wants to know what she should do.

* * *

A 24-year-old salesman consults his physician with a two-month history of fatigue, joint pain, and occasional gastrointestinal symptoms. Approximately three months ago, he bought an 80-year-old house and started renovating the interior.

* * *

The public is increasingly concerned about potential environmental health hazards and often wants answers to very concrete questions, such as: Is the water safe to drink? Could my miscarriage be due to my work environment? What is the likelihood of having a child with birth defects due to exposure during pregnancy to my computer's electromagnetic field? (see Box 1.) Are the pesticides used on fruit harmful? Is living close to power lines harmful? Patients ask their physicians these questions because, in general, they trust them and value their advice. Unfortunately, physicians often lack adequate, appropriate information and training with respect to environmental risks and health.

The integral relationship between the environment and health necessitates the active participation of knowledgeable physicians in both clinical and community contexts. In 1988, the Institute of Medicine (IOM) examined the role of primary care physicians in occupational and environmental medicine and called for enhanced physician training and education in this area. Noting that primary care physicians are often the health professionals of first contact for patients with environmentally related illnesses, the IOM suggested that, "as a minimum, all primary care physicians should be able to *identify possible occupationally or environmentally induced conditions* and *make the appropriate referrals for follow-up*" (Institute of Medicine, 1988:63).

Today's challenge is to help medical students develop the knowledge and skills they will need to deal effectively with environmental health issues in clinical care and public health contexts. Doing this within the confines of an already stressed and overcrowded

Box 1. Reproductive Hazards and VDT Exposure

A 31-year-old woman, gravida 1, para 0, presents to her obstetrician at six weeks' gestation with concerns about her home computer. She is a graduate student at the local university and is working on her thesis. This work requires that she use the computer for up to six hours per day. She has heard that there may be an association between electromagnetic fields from video display tubes (VDTs) and adverse pregnancy outcomes. She does not want to take any risks, but she hopes to finish her thesis before the child is born. She asks her physician's opinion of the literature on VDT exposure and birth defects.

There have been many reported clusters of women working with VDTs in office settings who gave birth to children with birth defects. Reported defects were widely heterogeneous, including clubfoot, congenital heart defects, neural tube defects, and cleft palate. In addition, clusters of prematurity and spontaneous abortion have been reported. VDTs emit nonionizing radiation: light, radiowaves, and microwave radiation. While there is some concern about the association of nonionizing radiation in the form of electromagnetic fields and the risk of hematologic tumors, brain tumors, and adverse reproductive outcomes, the evidence is still very mixed. The evidence of an association between electromagnetic fields and specific cancers (e.g., leukemia, brain tumors) is much stronger at this time than the evidence of an association between these fields and reproductive risk. Most physicians do not feel that VDTs pose a significant risk of adverse pregnancy outcomes.

Patients' concerns about potential occupational or environmental exposures during pregnancy must always be taken seriously. If the clinician does not know the medical literature on the exposure in question, it is imperative that he or she research the issue before simply reassuring the patient. Maternal exposures to many things clearly increase the risk of adverse pregnancy outcomes. Lead, solvents, ethylene oxide, glycol ethers, carbon monoxide, radiation, prolonged standing, and drugs such as thalidomide and alcohol are all clear examples of reproductive hazards. Caution and awareness of the possibility of new reproductive hazards is important to prevent unnecessary reproductive tragedies.

Adapted from Bentur and Koren (1991), Paul and Himmelstein (1988).
See also case study number 53 in Appendix C for more information on reproductive and developmental hazards.

four-year undergraduate medical curriculum that has been described by some as unresponsive to societal changes and needs (Abrahamson, 1978; Marston and Jones, 1992; Pew Health Professions Commission, 1991) and reinforcing and expanding this knowledge and these skills during postgraduate residency training is a formidable challenge.

Although efforts at curriculum reform have failed in the past, medical education may be embarking upon a new era. There are renewed calls for change; those calls and the

current evolutionary changes that are occurring in the health care system could be important driving forces for curricular change. Such change could include the integration and enhancement of environmental health in the curriculum.

To help prepare physicians for the emerging awareness of environmental health issues and their roles in addressing them, principles and concepts of environmental health must be taught and continually reinforced throughout undergraduate and postgraduate medical education and training. The committee believes, however, that specifying what should be taught is not as useful as describing what students should know and be able to do at the end of their training. With such competency-based objectives in mind, the committee recommends that all graduating medical students have the knowledge and skills listed below.

1. **Graduating medical students should understand the influence of the environment and environmental agents on human health based on knowledge of relevant epidemiologic, toxicologic, and exposure factors.**
2. **Graduating medical students should be able to recognize the signs, symptoms, diseases, and sources of exposure relating to common environmental agents and conditions.**
3. **Graduating medical students should be able to elicit an appropriately detailed environmental exposure history, including a work history, from all patients.**
4. **Graduating medical students should be able to identify and access the informational, clinical, and other resources available to help address patient and community environmental health problems and concerns.**
5. **Graduating medical students should be able to discuss environmental risks with their patients and provide understandable information about risk-reduction strategies in ways that exhibit sensitivity to patients' health beliefs and concerns.**
6. **Graduating medical students should be able to understand the ethical and legal responsibilities of seeing patients with environmental and occupational health problems or concerns.**

Consensus on the goals and content of a curriculum, such as the six competency-based learning objectives above, is a necessary but insufficient prerequisite for training medical students and residents in environmental medicine. Reasoned arguments for such a curriculum cannot alone ensure that it will be implemented. Other factors that affect the extent, quality, and success of implementation efforts include the availability of faculty time in an already overcrowded curriculum; support for teaching and curricular innovation; competing faculty and community concerns or interests; and budgetary constraints. Any strategy for implementing changes in the curriculum must be sensitive to these factors and include action at many levels.

At the medical school level, there is a need for knowledgeable and enthusiastic teachers, exciting teaching materials and methods, and creative and judicious use of

curricular time. This will require that administrators who recognize the importance of the curriculum support ongoing faculty development and provide adequate rewards for the teaching faculty. All this may necessitate activities at many other levels, for example, expanded initiatives by federal agencies, residency review committees, and professional organizations. Practice barriers, such as lack of reimbursement for preventive services, will also require attention.

With these many counterpressures and demanding complexities in mind, we present a practical and simple approach to integrating environmental medicine into the medical curriculum. Rather than defining and carving out new blocks or courses in an already crowded curriculum, the committee favors an integrative approach to enhancing the environmental and occupational health content in undergraduate medical education. This is not only the most expeditious approach to achieving the competency-based objectives, but it seems to be the most appropriate as well given the pervasive and fundamental nature of environmental effects on health. Integration also highlights the relevance of environmental and occupational medicine to basic science and clinical studies and provides a vehicle for enhancing faculty awareness of those issues. As described in this report, instructors should be able to integrate environmental medicine into existing medical school courses and clerkships fairly easily.

To ensure the progressive enhancement of competency in environmental medicine in medical education and practice, the committee makes recommendations for the continued funding and expansion of programs that currently support research and training, such as Academic Awards and Center Grants. This enhancement should build on the success of current programs and include adequate funding to support reasonable progress in curriculum development, faculty development, and continuing education. In addition to the current activities, the committee recommends that consideration be given to establishing (1) a database of curricular materials for faculty and students, and (2) a speakers bureau in environmental medicine. Information about these activities and resources should be disseminated with vigor to help ensure the integration of environmental medicine into medical education and practice.

To facilitate integration and enhancement of environmental medicine in medical education, the report includes four appendixes that provide 55 case studies and other detailed information on available educational resources and teaching aids. Of particular utility will be the indexes in Appendix C, which guide the reader to cases in environmental medicine based on: (1) chemical agents and conditions, (2) medical school courses and clerkships/clinical rotations, (3) sentinel pathophysiological conditions, and (4) clinical signs, symptoms, and presenting complaints. The appendixes and case studies can and should be used to facilitate the integration of environmental medicine into both education and practice.

ATSDR June 1990

Case Studies in Environmental Medicine

10

Cadmium Toxicity

Environmental ALERT . . .

- ☑ *Prevention is the key to managing cadmium exposure; no effective treatment for cadmium toxicity exists.*
- ☑ *Nutritional deficiencies can increase the risk of cadmium toxicity.*
- ☑ *Cadmium affects primarily the renal and skeletal systems.*

This monograph is one in a series of self-instructional publications designed to increase the primary care provider's knowledge of hazardous substances in the environment and to aid in the evaluation of potentially exposed patients. The Agency for Toxic Substances and Disease Registry (ATSDR) and the Centers for Disease Control (CDC) designate this continuing medical education activity for 1 credit hour in Category 1 of the Physician's Recognition Award of the American Medical Association and 0.1 continuing education units for other health professionals. See pages 21 to 23 for further information.

Guest Contributor: Emily E. Grum, MD
Guest Editor: Eddy A. Bresnitz, MD, MS

Peer Reviewers: Charles Becker, MD; Jonathan Borak, MD; Joseph Cannella, MD; Bernard Goldstein, MD; Alan Hall, MD; Richard J. Jackson, MD, MPH; Jonathan Rodnick, MD; Robert Wheater, MS; Brian Wummer, MD

U.S. DEPARTMENT OF HEALTH & HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry

Case Study

Low back pain and waddling gait in a 60-year-old woman

A 60-year-old woman comes to your office with complaints of low back pain, which is causing progressive difficulty in walking. The pain has gradually increased since the onset of menopause 5 years ago. This discomfort is especially noticeable after prolonged sitting.

Social history reveals that the patient has been a housewife since her marriage 38 years ago. Her husband, who is in good health, owns and operates a small retail shop in their home. The patient has been making jewelry for sale in her husband's shop and as a hobby for about 35 years. They have two adult sons who are in good health.

The patient denies a personal or family history of kidney disease, hypertension, diabetes mellitus, or cardiovascular disease; she also denies history of back trauma or weight loss. She has smoked one to two packs of cigarettes a day for the past 40 years. She does not take estrogens, calcium supplements, vitamins, or other medications.

On examination you find a thin female with a slightly stooped posture and a waddling gait. Blood pressure is 120/70. Her teeth have a yellow discoloration above the crown, and her fingernails are stained with nicotine. She is anosmic on cranial nerve examination. Results of cardiovascular and abdominal examination are normal. The lower lumbar spine is tender to percussion, but the patient does not complain of pain on straight leg raising. Her deep tendon reflexes are intact, and the remainder of the physical examination, including neurologic testing, is normal. Sensation and strength are normal in legs and feet. Range of motion is normal in hips and knees.

Initial laboratory data include a urinalysis showing 3+ proteinuria and glycosuria. BUN, creatinine, and albumin levels are normal. Roentgenograms of the pelvis and lumbosacral spine reveal pseudofractures and other evidence of severe osteomalacia and mild osteoporosis. There are no osteolytic or osteoblastic lesions.

(a) What should be included on the patient's problem list?

(b) What additional information would be helpful in diagnosing this woman's condition?

(c) What further tests, if any, would you recommend?

(d) What treatment would be appropriate for this patient?

Answers to the Pretest are included in Challenge answers (6) through (9) on page 19.

ATSDR

Exposure Pathways

- **In the general population, exposure to cadmium occurs primarily by eating crops grown in contaminated soil and seafood.**
- **Airborne cadmium sources include combustion of fossil fuels, incineration of municipal waste, and smelter emissions.**

Pure cadmium is a silver-white, lustrous metal, but cadmium in this form is not common in the environment. It is most often encountered in the earth's crust combined with chlorine (cadmium chloride), oxygen (cadmium oxide), and sulfur (cadmium sulfide). Cadmium oxide also exists as small particles in air (fume), the result of smelting, soldering, or other high-temperature industrial processes. Most cadmium used in the United States is obtained as a byproduct of the smelting of zinc, lead, or copper ores. Cadmium is used mainly in metal plating; in producing pigments, batteries, and plastics; and as a neutron absorbant in nuclear reactors.

Foods are the most important source of cadmium exposure for the general population. Low levels of cadmium are found in basic foodstuffs, especially grains, cereals, and leafy vegetables, which readily absorb naturally occurring cadmium or cadmium in soil contaminated by sewage sludge, fertilizers, and polluted groundwater. In 1946, the inhabitants of the Jintzu River basin in Japan were afflicted with a disease characterized by pain and bone fractures (called itai-itai or ouch-ouch disease), which was caused by high levels of cadmium in water and rice, the result of using water contaminated by discharges from a local zinc-mining operation. Cadmium bioaccumulates in the food chain; consequently, ingestion of animal internal organs, such as liver and kidneys, and some types of fish and shellfish may result in increased exposure.

The greatest sources of airborne cadmium are burning fossil fuels such as coal or oil, and incineration of municipal waste such as plastics and nickel-cadmium batteries. Cadmium may also escape into the air from zinc, lead, or copper smelters, and from iron and steel production facilities. Like most plants, tobacco contains cadmium, which is inhaled in cigarette smoke.

Cadmium concentrations in drinking water supplies are typically less than 1 microgram per liter (µg/L) or 1 part per billion (ppb). Groundwater seldom contains high levels of cadmium unless it is contaminated by mining or industrial wastewater, or seepage from hazardous waste sites. Soft or acidic water tends to dissolve cadmium and lead from water lines; cadmium levels are increased in water stagnating in household pipes. These sources have not caused clinical cadmium poisoning, but even low levels of contamination presumably contribute to the body's accumulation of cadmium.

Cadmium is a component of *chuifong tokwan*, a pharmaceutical compound manufactured in Asia and sold illegally in the United States as a "miracle herb." Some artists' paints contain a yellow pigment made from cadmium sulfide. Cadmium at one time was a leachable component of the alloy used in ice cube trays.

Who's at Risk

Background levels of cadmium in food, water, and ambient air are not a health concern for the general North American population. Typical dietary intake is about 30 micrograms of cadmium per day (30 µg/day), a rate roughly ten times lower than that required to cause critical renal effects. Acute cadmium toxicity is rare because very high levels are seldom encountered in the workplace today, and low doses are not acutely toxic. An acute oral dose of 50 µg/kilogram (kg) body weight (about 3500 µg in an adult) is considered the minimal amount capable of causing gastric irritation. Chronic exposures, however, can be a major concern because cadmium has a tendency to accumulate in the body.

Persons in the United States at greatest risk of cadmium exposure are 500,000 workers, including the following:

- Alloy makers
- Aluminum solder makers
- Ammunition makers
- Auto mechanics
- Battery makers
- Bearing makers
- Braziers and solderers
- Cable, trolley wire makers
- Cadmium platers
- Cadmium vapor lamp makers
- Ceramics, pottery makers
- Copper-cadmium alloy makers
- Dental amalgam makers
- Electric instrument makers
- Electrical condenser makers
- Electroplaters
- Engravers
- Glass makers
- Incandescent lamp makers
- Jewelers
- Lithographers
- Lithopone makers
- Mining and refining workers
- Paint makers
- Paint sprayers
- Pesticide makers
- Pharmaceutical workers
- Photoelectric cell makers
- Pigment makers
- Plastic products makers
- Sculptors, metal
- Smelterers
- Solder makers
- Textile printers
- Welders, cadmium alloy and cadmium-plate

- **Workers in industries producing or using cadmium have the greatest potential for cadmium exposure; hobbyists such as jewelry fabricators and artists may also be at increased risk.**
- **Cigarette smoke may add to the body's cadmium burden.**
- **Cadmium absorption may be increased in nutritionally deficient persons.**

Hobbyists may also encounter cadmium in their pursuits. For example, cadmium is present in many gold and silver solders used in fabricating jewelry and in the metal dust produced in grinding or engraving cadmium-plated surfaces. The likelihood of cadmium inhalation is increased in poorly ventilated work areas, and cadmium ingestion is increased by eating and smoking in these areas.

Cadmium air levels are usually thousands of times greater in the workplace than in the general environment. For example, the permissible exposure limit (PEL) of cadmium fume or cadmium oxide in the workplace is 100 micrograms per cubic meter of air (100 $\mu g/m^3$), whereas concentrations of cadmium in ambient air rarely exceed 0.0025 $\mu g/m^3$ in nonindustrialized areas and 0.040 $\mu g/m^3$ in urban areas. The U.S. Environmental Protection Agency (EPA) has estimated that 24-hour, lifelong inhalation of air containing 1 $\mu g/m^3$ cadmium is associated with a lung cancer risk of, at most, 2 additional cases in 1000 persons exposed.

Each cigarette contains 2 µg of cadmium, with 50% absorbed from the lungs during active cigarette smoking. Persons who smoke one pack per day typically have cadmium blood and body burdens approximately twice as high as those of nonsmokers.

Nutritional factors affect the amount of cadmium absorbed. Persons with low calcium, protein, or iron reserves absorb cadmium more efficiently and may be at increased risk of developing toxicity. Age and gender may also play a role. Iron-deficient neonates absorb greater amounts of cadmium than iron-deficient adults; females absorb more than males. Iron deficiency, resulting in increased cadmium absorption, may have contributed to the high incidence of itai-itai disease in multiparous Japanese women.

Challenge

(1) *Additional information for the case study: The patient maintains a jewelry fabricating and engraving area in her home basement where she uses abrasive grinders, engraving equipment, soldering tools, and various raw materials. She does not use a dust mask but does wear a face shield when operating the grinder. The work area is dusty, with only two small windows near the top of one wall capable of providing ventilation; there is no local or general mechanical exhaust system. She admits to smoking and eating in the work area. The patient and her husband also tend a small garden in the backyard in which they grow vegetables for the table. A nearby wastewater treatment plant provides free fertilizer, which her husband applies to the garden every few weeks. The garden is irrigated with water from a municipal well.*

What are the potential sources of cadmium exposure for this patient?

(2) *Why is the patient described in the case study at increased risk of cadmium toxicity?*

(3) *Is the patient's husband also at increased risk? Explain.*

Biologic Fate

Respiratory absorption of cadmium in humans is estimated to be from 30% to 60% of an inhaled dose, depending on particle size. Only the smallest particles penetrate to the alveoli, the major site of absorption. As a result, cadmium particles in fumes and cigarette smoke, which are smaller, are more completely absorbed than most cadmium particles of industrial origin.

In humans, no more than 5% of ingested cadmium is absorbed from the gut into the blood or lymphatic fluid. Although some nutritional factors increase this absorption, zinc and chromium can decrease cadmium uptake. Absorption through the skin is not a significant route of cadmium entry.

- ❑ **Cadmium has no known beneficial function in the human body.**
- ❑ **Cadmium is transported in the blood bound to metallothionein.**
- ❑ **The greatest cadmium concentrations are found in the kidneys and the liver.**

- **Urinary cadmium excretion is slow; however, it constitutes the major mechanism of elimination. Cadmium biologic half-life may be up to 30 years.**

Once absorbed, cadmium is distributed by the blood. Lymphocytes synthesize metallothionein, a metal-binding protein, which concentrates cadmium three-thousandfold. Cadmium does not undergo metabolic conversion *in vivo*.

Cadmium is eliminated from the body primarily in urine. The rate of excretion is low, probably because cadmium remains tightly bound to metallothionein, which is almost completely reabsorbed from the glomerular filtrate. Because excretion is slow, cadmium accumulation can be significant. Whereas cadmium concentration in blood reflects recent exposure, urinary cadmium concentration more closely reflects total body burden. However, when renal damage from cadmium exposure occurs, the excretion rate increases sharply, and urinary cadmium levels no longer reflect body burden.

The total cadmium body burden at birth is less than 1 µg, which gradually increases with age to about 30 milligrams (mg). The highest cadmium concentration is found in the kidneys, especially the renal cortex, followed by the liver, pancreas, and adrenals. In the kidney, cadmium concentration steadily increases over time, then declines at 50 to 60 years of age. In the liver, however, cadmium concentration increases continuously with age. The kidneys and liver together total about 50% of the body accumulation in humans.

Both the liver and kidneys store cadmium as a metallothionein complex, which serves not only to transport cadmium but also acts as a defense mechanism against the toxicity of the unbound cadmium ion. Ironically, it is the cadmium-metallothionein complex that accumulates in the kidneys and is partially responsible for cadmium's toxic renal effects. Cadmium does not accumulate in bone, and the blood-brain barrier appears to limit its uptake into the central nervous system. The placenta acts only as a partial barrier to fetal exposure.

The biologic half-life of cadmium in the body is estimated to be 30 years. This long half-life is due to the body's inability to deal with increasing cadmium intake by homeostatic control mechanisms; humans do not have an effective cadmium elimination pathway. Cadmium has no known biologic function in humans, and bioaccumulation appears to be a byproduct of increasing industrialization. Any excessive accumulation in the body should be regarded as potentially toxic.

(4) Could diet play a role in the condition of the patient described in the case study?

Physiologic Effects

The mechanisms of cadmium toxicity are not fully understood but may involve binding of the metal to key cellular sulfhydryl groups, competition with other metals (zinc and selenium) for inclusion in metalloenzymes, and competition with calcium for binding sites on regulatory proteins such as calmodulin. The route and extent of cadmium exposure will influence the presentation of toxic effects.

Cadmium primarily affects the kidneys and skeletal system.

Renal Effects

Nephrotoxicity may be caused by either chronic inhalation or chronic ingestion of the metal. Data from human studies suggest a latency period of approximately 10 years before clinical onset of renal damage, depending on intensity of exposure. Proteinuria appears to be irreversible, and continued exposure can lead to progressive renal dysfunction.

Cadmium toxicity may cause both tubular and glomerular damage with resultant proteinuria.

Typically the proximal renal tubules are affected, resulting in a Fanconi-like syndrome with urinary excretion of low molecular weight proteins such as β_2-microglobulin, lysozyme, and retinol-binding protein. Glucosuria, aminoaciduria, increased excretion of calcium and phosphate, and decreased renal concentrating capacity also occur. Disturbances in calcium and phosphorus metabolism may subsequently lead to formation of kidney stones and demineralization of bones.

Tubular proteinuria may be accompanied by glomerular dysfunction with increased urinary excretion of high molecular weight proteins such as albumin, transferrin, and immunoglobulin G (IgG). An increased renal excretion of enzymes may also occur.

Skeletal Effects

- **Bone changes appear to be secondary to renal tubular dysfunction.**

Bone lesions usually occur late in severe chronic cadmium poisoning and include pseudofractures and other effects of osteomalacia and osteoporosis. Pseudofractures are spontaneous fractures that follow the distribution of stress in normal skeleton or occur at sites where major arteries cross the bone and cause mechanical stress through pulsation. Such fractures may have contributed to the waddling gait seen in Japanese patients with itai-itai disease.

Skeletal effects appear to be secondary to increased urinary calcium and phosphorus losses. These effects are compounded by inhibition of renal hydroxylation of vitamin D, which eventually leads to a deficiency of its active form. Some investigators believe cadmium also exerts an inhibitory effect on calcium absorption from the gastrointestinal tract.

Respiratory Effects

- **Acute cadmium inhalation may mimic metal fume fever.**
- **Chronic cadmium inhalation may result in impairment of pulmonary function with a reduction in ventilatory capacity.**

Acute cadmium oxide inhalation exposure occurs rarely, but has been reported to cause chemical pneumonitis and metal fume fever (a transient and generally benign syndrome of fever, malaise, and chest tightness). Studies have associated chronic cadmium inhalation with pulmonary function impairment, notably mild emphysema and pulmonary fibrosis with reduced ventilatory capacity. However, study limitations, such as small sample size, lack of a suitable cohort, and failure to control for the confounding effects of cigarette smoking, have raised questions about these findings. In one study of workers making copper-cadmium alloy, the largest reductions in forced expiratory volume in 1 second (FEV_1), its ratio to forced vital capacity (FEV_1/FVC%), and gas transfer were noted in those cadmium workers with the highest liver cadmium levels and the highest cumulative cadmium exposures. Pulmonary changes appear to occur after renal damage and are rarely seen today.

Carcinogenic Effects

- **Cadmium's carcinogenic effects have been demonstrated in experimental animals; evidence in humans is less conclusive.**

Inhalation of cadmium chloride and intratracheal instillation of high doses of cadmium sulfide are associated with an increased frequency of lung tumors in rats. Inhalation of various cadmium compounds did not produce increased incidence of lung tumors in hamsters or mice, however.

Epidemiologic studies of workers suggest a possible association between cadmium inhalation and the development of lung, prostatic, and testicular cancer. Many of these studies failed to control for smoking or exposure to other chemicals, however, and only small numbers of persons were evaluated. No clinical or experimental evidence indicates that ingesting cadmium in food or drinking water causes cancer. This is also true in Japan, where oral intake of cadmium tends to be high. Despite the uncertainty regarding the carcinogenicity of cadmium in humans, EPA and the International Agency for Research on Cancer have classified cadmium as a probable human carcinogen when inhaled.

Developmental Effects

No conclusive evidence of cadmium-induced teratogenicity in either experimental animals or humans has been reported. In a Swedish epidemiologic study of pregnant women exposed to high cadmium concentrations in the workplace, an increased incidence of infants with low birth weight was reported.

❑ **No evidence of teratogenic effects in cadmium-exposed humans has been reported.**

Other Effects

Chronic cadmium exposure has been reported to cause mild anemia, anosmia, yellowing of teeth, and, occasionally, liver damage. There is no conclusive evidence that cadmium alone causes hypertension. However, cadmium-induced renal dysfunction can eventually manifest in hypertension.

Challenge

(5) Could cadmium intoxication explain the problem list and initial laboratory findings for the patient described in the case study? Explain.

ATSDR

Clinical Evaluation

History and Physical Examination

- **Concomitant exposure to other heavy metals should be assessed.**

Detailed questioning about occupations and hobbies is the key to including chronic cadmium poisoning in the differential diagnosis. Inhalation exposure most often occurs among workers and hobbyists when cadmium fumes are produced by high-temperature processes such as welding, smelting, and soldering, and where cadmium dust results from grinding.

In the general population, ingestion of cadmium-contaminated food is more likely to occur than inhalation of cadmium particles. Today, acute cadmium ingestion is unlikely to be a clinically significant source of exposure in North America. Chronic ingestion, however, is still possible in certain populations, for example, children with pica who ingest contaminated soil.

Signs and Symptoms

Adverse effects of excessive cadmium exposure may include the following:

Acute Exposure
- Gastroenteritis (ingestion only)
- Bronchitis (inhalation only)*
- Interstitial pneumonitis (inhalation only)
- Pulmonary edema (inhalation only)

Chronic Exposure
- Proteinuria
- Osteomalacia (itai-itai disease)
- Pulmonary fibrosis (inhalation only)*
- Liver damage (rare)
- Hypertension
- Lung cancer*
- Prostatic cancer*
- Mild anemia
- Yellow discoloration of front teeth near gum line
- Anosmia

* Evidence of human health effects is inconclusive.

Acute Exposure

Most acute cadmium inhalation exposures involve initial symptoms and physical findings relating to the respiratory system. The first symptom, usually throat irritation, may not be severe enough to prompt the worker to leave the area. Symptoms, which may be delayed by hours or days, include pleuritic chest pain, dyspnea, cyanosis, fever, tachycardia, and nausea. Depending on the extent of exposure, noncardiogenic pulmonary edema may appear and progress to death.

- **Acute inhalation of cadmium may cause symptoms similar to those of metal fume fever.**

In the past, acute cadmium intoxication occurred after ingestion of acidic foods or beverages stored in cadmium-plated containers, with symptoms of severe nausea, vomiting, salivation, abdominal cramps, and diarrhea. Acute renal failure, cardiopulmonary depression, and shock due to fluid loss have also occurred. In humans, single lethal oral doses of cadmium have ranged from 350 to 8900 mg. An ingestion of 150 grams (g) of cadmium chloride was reported to produce facial edema, vomiting, hypotension, metabolic acidosis, pulmonary edema, oliguria, respiratory arrest, and, finally, death after 30 hours.

- **Acute oral ingestion results in severe gastroenteritis.**

Chronic Exposure

Effects of chronic cadmium exposure are dose-dependent. Low-level chronic exposure produces few early physical findings. Severe chronic exposure leads to manifestations of renal tubular dysfunction, especially in postmenopausal, multiparous females. This group typically has calcium and vitamin deficiencies that can increase the gastrointestinal absorption of cadmium. Other symptoms include low back pain and bone pain secondary to pseudo- and pathologic fractures. Chronic cadmium intoxication may also play a role in the development of hypertension, although the association is weak. Anosmia and yellow discoloration of teeth near the gum line may be noted.

- **Mild anemia and yellow discoloration of teeth may occur.**
- **Chronic exposure may result in back pain and renal dysfunction.**

Laboratory Evaluation

Initial laboratory evaluation should focus on the kidneys. Screening tests include measures of renal dysfunction such as BUN, serum and urinary creatinine, serum and urinary protein, and glucose. Complete blood count, liver function tests, and chest

X ray (if cadmium inhalation is suspected) should be performed. Specialized laboratory tests include direct measurement of cadmium levels and more sophisticated renal function tests.

Direct Biologic Indicators

The best screening and diagnostic test for chronic cadmium exposure is a 24-hour urinary cadmium level, normalized to creatinine excretion.

Urine cadmium. With low to moderate chronic exposure, urinary cadmium reflects the total body burden. The average daily excretion of cadmium in persons with no known cadmium exposure is usually below 1 µg/L, or 1 µg/g creatinine, increasing with age and smoking. When all cadmium-binding sites in the kidney become saturated, however, renal dysfunction results and the direct relationship to body burden is lost. The amount of cadmium excreted then increases dramatically, reflecting recent exposure rather than total body burden. When urinary cadmium levels are less than 10 µg/g creatinine, renal dysfunction is considered unlikely.

Serum cadmium. Serum cadmium levels reflect recent exposure and generally are not useful for evaluating chronically exposed patients. Normal serum concentrations of cadmium in nonexposed persons range from 0.05 to 0.3 micrograms per deciliter (µg/dL). Occupationally exposed persons may have levels ranging from 1 to 10 µg/dL. A blood level of 5 µg/dL or higher is considered toxic.

Cadmium in hair. Studies of exposed workers have not found a quantitative relationship between hair cadmium levels and body burden. Because of the potential for sample contamination, hair levels are not reliable either as a predictor of toxicity or as an indicator of occupational exposure.

Indirect Biologic Indicators

Urinary metallothionein and β_2-microglobulin excretion can be correlated with long-term cadmium exposure.

The tests that follow have been used to determine renal damage in persons exposed to high cadmium levels. They may have little relevance in evaluating persons exposed to lower environmental levels, however.

Urinary β_2-microglobulin. This low molecular weight protein is found in increased amounts in the urine of patients with long-term cadmium exposure and is considered a more sensitive indicator of cadmium exposure than total proteinuria. However, other renal diseases, such as chronic pyelonephritis, also cause

increased β_2-microglobulin excretion. Excretion of β_2-microglobulin increases with age and cadmium exposure, but has been reported to average about 200 µg/g creatinine in unexposed persons.

Urinary metallothionein. Metallothionein is a low molecular weight protein synthesized in response to the presence of divalent metals such as cadmium, zinc, and copper. The protein is formed primarily in the lymphocytes, kidney, liver, and intestine. Its function appears to be the binding of metal ions, thus rendering them less toxic. Once metallothionein binds to cadmium, the complex preferentially accumulates in the kidney. Urinary levels of metallothionein correlate well with urinary cadmium levels and can reflect total cadmium body burden; however, urinary concentration of the cadmium-metallothionein complex increases significantly once renal dysfunction has developed.

Urinary retinol-binding protein. Retinol-binding protein is another low molecular weight protein appearing in the urine after chronic cadmium exposure. It is excreted when tubular reabsorption decreases due to any cause and, therefore, is nonspecific and can be used only as a supportive test in cases of suspected cadmium exposure.

(6) If you suspect cadmium poisoning, what other questions could help gauge the extent of exposure to the patient described in the case study?

(7) What tests would be helpful in further evaluating the patient or in supporting a diagnosis of cadmium toxicity?

(8) Assuming the patient described in the case study has cadmium toxicity, what would be a likely urinary cadmium level?

ATSDR

Treatment and Management

One exposed person often signals potential or actual exposure of others, with the possibility of a common exposure source. Such sources include the workplace, drinking water supply, community irrigation, proximity to a smelter, and so on. Public health authorities should be notified whenever cadmium toxicity is suspected in a patient so that case-finding may be initiated and preventive measures taken.

Acute Exposure

There is no specific antidote for cadmium poisoning.

There is no effective treatment for cadmium poisoning. Standard chelation therapy using ethylenediaminetetraacetic acid (EDTA), British anti-Lewisite (BAL or dimercaprol), or dimercaptosuccinic acid (DMSA) has generally not proven effective. BAL is contraindicated because it may increase nephrotoxicity. Treatment remains supportive, including fluid replacement, supplemental oxygen, and mechanical ventilation, if necessary. In cases of ingestion, gastric decontamination by emesis or gastric lavage may be beneficial soon after exposure. Administration of activated charcoal has not been proven effective.

Chronic Exposure

Prevention of further exposure is the most important step in management of patients with symptoms suggestive of cadmium intoxication.

The mainstay of therapy in chronic poisoning involves removing the patient from further exposure. In the workplace, engineering controls, improved ventilation, and personal hygiene are the first line of defense. In addition, patient and worker education is vital in encouraging preventive behavior and in assisting early detection of cadmium toxicity. Respiratory protection should be worn in occupational or hobby settings where airborne concentrations may exceed allowable limits. Smoking, eating, and drinking in the work area should be discouraged.

(9) What treatment will you recommend for the patient described in the case study?

(10) Should the patient's neighbors be evaluated for cadmium or other heavy-metal exposure? Explain.

Standards and Regulations

With increasing evidence of its toxicity, both national and international agencies have sought to regulate cadmium exposure. These efforts encompass workplace and environmental guidelines or regulations for air emissions, drinking water, food, industrial discharges, and hazardous waste concentrations. Table 1 summarizes standards, regulations, and guidelines for cadmium.

Table 1. Standards and regulations for cadmium

Agency*	Focus	Level	Comments
ACGIH	Air -Workplace		
	cadmium dust	0.05 mg/m^3	Advisory; TWA†
	cadmium fume	0.05 mg/m^3	15-minute ceiling limit
NIOSH	Air -Workplace	N/A	Advisory; lowest possible limit based on carcinogenic risk
OSHA	Air -Workplace		
	cadmium dust	0.2 mg/m^3	Regulation; PEL§
	cadmium fume	0.1 mg/m^3	
EPA	Air	N/A	Under review
	Water	0.01 ppm	Regulation; maximum contaminant level in drinking water; suggested revision to 0.005 ppm
WHO	Food	0.4-0.5 mg	Advisory; provisional tolerable weekly intake for adults

* ACGIH = American Conference of Governmental Industrial Hygienists; EPA = Environmental Protection Agency; NIOSH = National Institute for Occupational Safety and Health; OSHA = Occupational Safety and Health Administration; WHO = World Health Organization

† TWA (Time-Weighted Average) = time-weighted average concentration for a normal 8-hour workday and 40-hour workweek to which nearly all workers may be repeatedly exposed

§ PEL (Permissible Exposure Limit) = highest level averaged over a normal workday, to which a worker may be exposed.

Workplace

Air

The PEL for airborne cadmium in the workplace has been set by the Occupational Safety and Health Administration (OSHA) at 0.2 mg/m^3 as an 8-hour time-weighted average (TWA) for cadmium dust, and 0.1 mg/m^3 for cadmium fume (cadmium oxide). A 15-minute ceiling concentration of 0.6 mg/m^3 for cadmium dust

❑ OSHA has proposed lowering cadmium workplace exposures by 99%.

and 0.3 mg/m³ for cadmium fume (cadmium oxide) has been mandated. OSHA's proposed 1990 ruling seeks to reduce permissible cadmium workplace exposures by 99%.

The National Institute for Occupational Safety and Health (NIOSH) recommends that cadmium be regarded as a potential carcinogen based both on epidemiologic studies of lung cancer among workers and laboratory studies.

Environment

Air

- **No EPA air standard for cadmium currently exists.**

Cadmium levels in the ambient atmosphere are generally low. Typically, cadmium concentrations range from 1 to 5 nanograms per cubic meter (ng/m³) in sparsely populated rural areas and from 5 to 40 ng/m³ in urban air. In the vicinity of active zinc or lead smelters, cadmium values of 300 to 700 ng/m³ have been measured at distances of 0.5 to 1 kilometer from the smelter. Near incinerators, average cadmium air levels have been estimated to be 7 ng/m³. EPA is seeking classification of cadmium as a hazardous air pollutant; however, no ambient air standard for cadmium currently exists.

Water

- **EPA has proposed lowering the regulated level of cadmium in drinking water.**

EPA has established a maximum contaminant level (MCL) for cadmium in drinking water of 0.010 mg/L (0.01 ppm) and is currently seeking its revision to 0.005 mg/L (0.005 ppm). EPA and some states regulate the amount of cadmium discharged in industrial wastewaters.

Food

- **Dietary cadmium is not regulated.**

Average daily dietary cadmium intake is 10 to 50 µg. The World Health Organization has recommended a provisional tolerable weekly intake of 400 to 500 µg cadmium for adults. Nevertheless, the exact amount of cadmium in the average American diet is difficult to control. For this reason, efforts have been directed toward reducing cadmium discharged into waterways and deposited on soil, which could eventually enter the food chain.

Soil

- **EPA regulates application of solid waste to topsoil.**

A 1979 report noted that topsoils in the United States contain an average cadmium level of about 260 µg/kg. Levels in soil near sources of contamination may greatly exceed this value. Crops grown in contaminated soil are capable of translocating the metal and present a likelihood of exposure to consumers. Currently, there is no effective way to decontaminate soil. EPA regulation for application of solid waste to topsoil used in crop production for human consumption is 0.5 kg of solid waste per hectare annually.

Suggested Reading List

General

Baker EL Jr, Peterson WA, Holtz JL, Coleman C, Landrigan PJ. Subacute cadmium intoxication in jewelry workers: an evaluation of diagnostic procedures. Arch Environ Health 1979;34(3):173-7.

Stoeppler M, Piscator M, eds. Cadmium. New York: Springer-Verlag, 1985 (Environmental toxin series, vol. 2).

Carcinogenicity

International Agency for Research on Cancer. Overall evaluations of carcinogenicity: an updating of IARC monographs. Lyon: IARC, 1987:139-142. (IARC monographs on the evaluation of carcinogenic risk of chemicals to man; Suppl 7).

Kazantzis G. The mutagenic and carcinogenic effects of cadmium: an update. Toxicological and environmental chemistry 1987;15:83-100.

Respiratory Effects

Barnhart S, Rosenstock L. Cadmium chemical pneumonitis. Chest 1984;86:789-91.

Davison AG, Fayers PM, Taylor AJ, et al. Cadmium fume inhalation and emphysema. Lancet 1988;1(8587): 663-7.

Skeletal Effects

Nogawa K, Tsuritani I, Kido T, Honda R, Yamada Y, Ishizaki M. Mechanism for bone disease found in inhabitants environmentally exposed to cadmium: decreased serum 1*a*, 25-dihydroxyvitamin D level. Int Arch Occup Environ Health 1987;59:21-30.

Renal Effects

Kido T, Honda R, Tsuritani I, et al. Progress of renal dysfunction in inhabitants environmentally exposed to cadmium. Arch Environ Health 1988;43:213-17.

Shaikh ZA, Tohyama C, Nolan CV. Occupational exposure to cadmium: effect on metallothionein and other biological indices of exposure and renal function. Arch Toxicol 1987;59:360-4.

Food and Diet

Flanagan PR, McLellan JS, Haist J, Cherian G, Chamberlain MJ, Valberg LS. Increased dietary cadmium absorption in mice and human subjects with iron deficiency. Gastroenterology 1978;74:841-6.

Sherlock JC. Cadmium in foods and the diet. Experientia 1984;40:152-6.

Laboratory Evaluation

Shaikh ZA, Smith LM. Biological indicators of cadmium exposure and toxicity. Experientia 1984; 40:36-43.

Thun MJ, Clarkson TW. Spectrum of tests available to evaluate occupationally induced renal disease. J Occup Med 1986;28:1026-33.

Human Health Effects

Bernard A, Lauwerys R. Cadmium in human population. Experientia 1984;40:143-52.

Hallenbeck WH. Human health effects of exposure to cadmium. Experientia 1984; 40:136-42.

Yost KJ. Cadmium, the environment and human health: an overview. Experientia 1984;40:157-64.

ATSDR

Related Government Documents

Agency for Toxic Substances and Disease Registry. Toxicological profile for cadmium. Atlanta: US Department of Health and Human Services, Public Health Service, 1989. NTIS report no. PB/89/194476/AS.
Environmental Protection Agency. Health effects assessment for cadmium. Washington, DC: Office of Health and Environmental Assessment. EPA report no. 540/1-86-038.
Environmental Protection Agency. Cadmium contamination of the environment: an assessment of nationwide risk. Washington, DC: Environmental Protection Agency. EPA report no. 440/4-85-023.

Answers to Pretest and Challenge Questions

Pretest is found on page 1. Challenge questions begin on page 5.

(1) Potential sources of cadmium are as follows:
   (a) cadmium fume (cadmium oxide) generated by use of gold and silver solders during jewelry fabrication
   (b) cadmium dust produced in smoothing jewelry with abrasive grinding or in engraving cadmium-plated surfaces
   (c) food and cigarettes in the workplace contaminated by cadmium-containing particulates and dust
   (d) cigarette smoke
   (e) food grown in soil contaminated with cadmium-containing fertilizer obtained from the wastewater treatment plant

(2) Risk factors are due to not only increased opportunity for cadmium exposure, but age and nutritional status as well. The patient's hobby, jewelry fabrication, may provide low-to-moderate chronic cadmium exposure. Lack of respiratory protection, poor ventilation, and poor hygiene in the work area increase the amount of her exposure. The patient also inhales approximately 2 μg cadmium with each cigarette smoked. The amount of cadmium ingested from the vegetables grown in her garden is unknown, but sludges from wastewater treatment plants have been found to contain significant levels of cadmium. Factors that may enhance cadmium absorption from the gut are age and certain dietary deficiencies.

(3) Yes, the patient's husband also may be at increased risk of cadmium toxicity because of increased opportunity for exposure, although his risk is probably less than his wife's. The husband is exposed to cadmium by eating food from the contaminated garden and by inhaling tobacco smoke from cigarettes, even more so if he smokes. In the basement work area, he may encounter cadmium fumes and dust as a result of his wife's hobby. He also may be exposed to the cadmium on his wife's clothing and skin if she does not shower and change clothes before leaving the work area.

(4) Yes, diet could play an important role in the patient's condition, both for what it contributes and for what it does not include. For example, the homegrown vegetables from the garden, particularly leafy vegetables, and animal liver or kidney and shellfish could be contributing to her cadmium burden. If her diet is deficient in iron, calcium, or protein she may be absorbing cadmium more efficiently.

(5) The patient's problem list includes the following:
   back pain
   severe osteomalacia and mild osteoporosis
   pseudofractures
   yellow discoloration of the teeth
   proteinuria and glycosuria

All of these are consistent with chronic cadmium toxicity. The patient is also a smoker. Chronic cadmium exposure primarily affects the kidneys and skeleton. Renal dysfunction in this patient is indicated by the laboratory findings. The stooped posture, waddling gait, lumbar pain, and pain induced by spinal percussion are the result of skeletal changes and deformities.

(6) Most of your questions will probably center on the patient's hobby, as this is the greatest potential source of cadmium exposure. Typical questions would include the following:
   (a) What types of materials and metals are used in making jewelry? What are the ingredients of all composite products?
   (b) On a weekly basis, how many hours are spent fabricating jewelry in the basement?
   (c) What type of face shield is used? Why is respiratory protection not used during grinding and soldering operations?
   (d) Is the work area kept clean and free of dust? How?
   (e) Does she wash her hands before eating in the work area and are attempts made to keep food and cigarettes from becoming contaminated by dust and particulates?
   (f) Does she shower and change her clothes before leaving the work area?

   It is also important to investigate smoking habits.

(7) The most useful diagnostic test for cadmium exposure is a 24-hour urinary cadmium excretion standardized for creatinine. β_2-microglobulin levels, in conjunction with cadmium excretion, will aid in evaluating subclinical renal dysfunction. The following tests also may be helpful in evaluating the patient: urinary protein and glucose, LDH, SGPT or ALT, and SGOT or AST. A chest X ray and pulmonary function test should be obtained if cadmium inhalation is a factor.

(8) The patient is experiencing renal dysfunction, as evidenced by the 3+ level of proteinuria and glycosuria. When proximal tubular damage occurs, cadmium excretion can result from two sources; breakdown of the tubular epithelium and decreased reabsorption. Under these conditions, urinary cadmium levels are likely to be markedly increased and no longer reflect body burden. Exposed workers can excrete several hundred micrograms of cadmium per gram of creatinine; urinary cadmium levels in an unexposed population are typically between 1 and 10 µg cadmium/g creatinine. The patient therefore would be expected to have a urinary cadmium level of several hundred micrograms of cadmium per gram of creatinine, depending on her most recent exposure.

(9) There is no effective treatment for cadmium toxicity; chelation therapy has no role in cadmium poisoning. Removal from the source of exposure and patient education to significantly reduce exposure are important, particularly before the condition has progressed to irreversible renal dysfunction. Supportive measures to alleviate symptoms should be provided.

(10) The neighbors should be evaluated and educated. Even if they do not use the fertilizer from the wastewater treatment plant or water from the same irrigation source, runoff from the patient's land may contaminate their soil or well water. Consultation with the local or state health department is advisable if a potential public health hazard exists.

TSDR October 1992
Case Studies in Environmental Medicine

11

Benzene Toxicity

Environmental ALERT . . .

- ☑ *Benzene is an important commercial commodity and has become widespread in the environments of developed countries.*
- ☑ *In the United States, gasoline contains up to 2% benzene by volume; in other countries, the benzene concentration in gasoline may be as high as 5%.*
- ☑ *Benzene in the workplace has been associated with aplastic anemia and leukemia and may also cause nonhematologic cancers.*

This monograph is one in a series of self-instructional publications designed to increase the primary care provider's knowledge of hazardous substances in the environment and to aid in the evaluation of potentially exposed patients. *See page 19 for more information about continuing medical education credits and continuing education units.*

Guest Contributors: James E. Cone, MD, MPH, and Kate Gelperin, MD, MPH
Guest Editor: Bernard Goldstein, MD

Peer Reviewers: John Ambre, MD, PhD; Charles Becker, MD; Jonathan Borak, MD; Joseph Cannella, MD; Alan Hall, MD; Richard J. Jackson, MD, MPH; Howard Kipen, MD; Jonathan Rodnick, MD; Brian Wummer, MD

U.S. DEPARTMENT OF HEALTH & HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry

Case Study

A 50-year-old diesel mechanic with recurring nosebleeds, fatigue, and weight loss

A 50-year-old man is prompted to visit your office because of a nosebleed that has been recurring for 2 days. He says that this is the third episode of nosebleeds in the last 6 months. He expresses concern that he becomes easily fatigued at work, and 2 months ago he began noticing bruises on his arms and legs, although he does not recall the causes. He has lost more than 12 pounds in the last 2 years, which he attributes to loss of appetite.

History of previous illness includes a fractured arm in childhood. He has had three bad colds in the past 2 years that lasted for more than a week and included coughing and breathing difficulty. The patient occasionally drinks beer; he quit smoking cigarettes 4 years ago. He does not have allergies and is taking no medications at this time.

On examination, you find a muscular man with somewhat pale and dry skin. Conjunctivae are pale. Numerous ecchymoses and petechiae are noted on arms and legs. Many seem to be old with incomplete healing. BP is 138/84; HR is 94. Temperature is normal. His throat is moderately inflamed, and prominent cervical nodes are palpable. Examination is otherwise within normal limits.

On further questioning, you learn that the patient is a diesel mechanic and has worked on trucks for the same employer for the previous 12 years. He and his wife divorced 8 years ago; his wife became nervous and withdrawn after two miscarriages, which led to marital stress. He has lived in his home for the past 16 years. He has a daughter, age 16, who lives with his ex-wife.

Laboratory studies reveal the following: glucose, BUN, and bilirubin within normal limits; Hgb 10.2 g/dL (normal 14.0-18.0); Hct 32.6% (44.8-52.0); RBC 3.32 mil/mm^3 (4.3-6.0); MCV 98 fl (80-100); MCH 31 pg (26-31); MCHC 31% (31-36); WBC 1500 mm^3 (5000-10,000); segs 60% (40-60); bands 1% (0-5); lymphs 31% (20-40); monos 8% (4-8); platelets 50,000/mm^3 (150,000-400,000). A chest X ray is negative except for some suggestion of hyperlucency; ECG is normal.

(a) What is the problem list for this patient? What is the differential diagnosis?

(b) What additional testing would you recommend?

(c) What measures would you take to manage the case and treat this patient?

Answers to the Pretest can be found in Challenge answers (3) through (7) on pages 17 and 18.

1

ATSDR

Exposure Pathways

- **Benzene is commonly used as a solvent and as a raw material in chemical syntheses.**
- **Benzene is added to unleaded motor fuels for its antiknock characteristics.**
- **Because benzene plays such a vital role in many industrial processes and is a component of gasoline, it is widespread in the environment.**

Benzene (C_6H_6) is the first member of a series of aromatic hydrocarbons recovered from refinery streams during catalytic reformation and other petroleum processes. It is a clear, colorless, highly flammable liquid at room temperature. Its vapor is heavier than air and can travel to a source of ignition and flash back. It has a pleasant odor detectable at concentrations greater than 4 parts per million (ppm). (The workplace permissible exposure level [PEL] is 1 ppm). Common synonyms for benzene include benzol, cyclohexatriene, phenyl hydride, and coal tar naphtha.

Benzene is one of the world's major commodity chemicals. Its primary use (85% of production) is as an intermediate in the production of other chemicals, predominantly styrene (for styrofoam and other plastics), cumene (for various resins), and cyclohexane (for nylon and other synthetic fibers). Benzene is an important raw material for the manufacture of synthetic rubbers, gums, lubricants, dyes, pharmaceuticals, and agricultural chemicals.

Benzene is a natural component of crude and refined petroleum. The mandatory decrease of lead alkyls in gasoline has led to an increase in the aromatic hydrocarbon content of gasoline to maintain high octane levels and antiknock properties. In the United States, gasoline typically contains less than 2% benzene by volume, but in other countries the benzene concentration may be as high as 5%.

Because of its lipophilic nature, benzene is an excellent solvent. Its use in paints, thinners, inks, adhesives, and rubbers, however, is decreasing and now accounts for less than 2% of current benzene production. Benzene was also an important component of many industrial cleaning and degreasing formulations but now is replaced mostly by toluene, chlorinated solvents, or mineral spirits. Although benzene is no longer added in significant quantities to most commercial products, traces of it may still be present as a contaminant.

Because of its many uses, benzene is widespread in the environment. It is a component of both indoor and outdoor air pollution. Benzene levels measured in ambient air have ranged from less than 0.001 ppm in pristine rural areas to more than 0.1 ppm in urban areas. Sources of benzene in air are usually associated with chemical manufacturing or gasoline, including gasoline bulk-loading and discharging facilities and combustion engines (such as in automobiles, lawn mowers, and snow blowers). In almost all cases, benzene levels inside residences or offices are higher than levels outside. Benzene levels are also usually higher in homes with attached garages and those occupied by smokers. In the fall

and winter when buildings are less-well ventilated, benzene levels are even higher. The Environmental Protection Agency (EPA) classifies benzene as a Group A* carcinogen and has estimated that a lifetime exposure to 0.004 ppm benzene in air will result in, at most, 1 additional case of leukemia in 10,000 people exposed. (EPA risk estimates assume there is no threshold for benzene's carcinogenic effects.)

Leakage from underground storage tanks and seepage from landfills or improper disposal of hazardous wastes has resulted in benzene contamination of groundwater used for drinking. Effluent from industries is also a source of ground-water contamination. EPA's Office of Drinking Water has estimated that lifetime exposure to a benzene concentration of 68 parts per billion (ppb) in drinking water would correspond to, at most, 1 additional cancer case in 10,000 people exposed. (The current EPA maximum contaminant level [MCL] for benzene in drinking water is 5 ppb.) In addition to being ingested, benzene in water can also be absorbed through wet skin and inhaled as it volatilizes during showering or laundering.

Persons who smoke one pack of cigarettes a day inhale a daily dose of approximately 1 milligram of benzene, which is about one-thirtieth of the daily amount inhaled by a worker exposed at the currently permissible workplace level.

* Group A consists of agents for which sufficient evidence supports a causal association between exposure and cancer in humans and in experimental animals.

Challenge

(1) Later, the patient in the case study tells you that his well water has always tasted "funny" and smells like "solvent." You learn that a chemical plant was adjacent to his property until 9 years ago when the company moved to another location. You are concerned about your patient's description of his drinking water, and you request that the state health department investigate the problem. The investigator contacts the chemical company that owns the abandoned site and learns that benzene is stored at the site in tanks that are above and below ground. Laboratory analyses of the patient's well water reveal an average concentration of 20 ppm benzene and traces of 1,1,1-trichloroethane and toluene.

What areas will you explore in your questioning to gauge the extent of the patient's exposure to benzene?

ATSDR

Who's at Risk

- Two to three million U.S. workers are at risk of benzene exposure.
- Alcohol and other drugs that induce the mixed function oxidase (MFO) enzymes may potentiate the effects of benzene.

Workers employed in industries using or producing benzene have the greatest likelihood of exposure. The National Institute for Occupational Safety and Health (NIOSH) estimates that approximately two to three million workers in the United States may be exposed to benzene during refining operations; gasoline storage, shipment, and retail operations; chemical manufacturing; plastics and rubber manufacturing; shoe manufacturing; printing; and activities in chemical laboratories. A review of benzene exposure in the U.S. petroleum industry from 1978 to 1983 indicated that 87% of exposures were below an 8-hour time-weighted average (TWA) of 1 ppm and 98% were below 10 ppm.

In 1980, an estimated 37 million people in this country were exposed to benzene vapors at self-service gasoline stations. During gasoline pumping, atmospheric benzene levels up to 6.6 ppm have been measured, with a 6-hour TWA of 0.1 ppm. This risk has been lowered by installing vapor recapture devices on delivery hoses, which, if used properly, significantly reduce exposure. Catalytic converters have significantly reduced the benzene in automobile emissions.

Benzene is converted to toxic metabolites mostly by mixed-function oxidases (MFO) in the liver and bone marrow. MFO-inducing drugs (e.g., phenobarbital, alcohol) and certain chemicals (e.g., chlordane, parathion) may increase the rate at which toxic metabolites of benzene are formed. Theoretically, persons with rapidly synthesizing marrows—the fetus, infants and children, persons with hemolytic anemia or with agranulocytosis—are at increased risk.

Challenge

(2) Does the patient in the case study have any risk factors for the adverse effects of benzene? Is anyone else in the case at risk of benzene exposure or its adverse effects?

Biologic Fate

Benzene is absorbed rapidly by inhalation and ingestion, and slowly through intact skin. After a 4-hour exposure to approximately 50 ppm benzene in air, human volunteers absorbed about 50% of the amount inhaled.

Distribution of benzene to tissues is dependent on relative perfusion rates. In humans, approximately half of an inhaled dose is distributed to the liver and bone marrow. Benzene accumulation is slow in fat, but the total potential uptake is great because of benzene's high lipid solubility.

Absorbed benzene is metabolized primarily in the liver. Benzene metabolism initially involves oxidation, with phenol as the major metabolite. Further metabolic products formed by the introduction of hydroxyl groups on the aromatic ring include hydroquinone, catechol, and 1,2,4-trihydroxybenzene. These hydroxylated metabolites can be further oxidized to their corresponding quinones or semiquinones. Urinary excretion of small amounts of muconic acid, a straight-chain dicarboxylic acid, indicates that the benzene ring also is opened during metabolism.

Bone marrow is the main target organ of benzene toxicity. It contains the MFO enzymes necessary to metabolize benzene, and although benzene metabolism in bone marrow is not clearly understood, one or more benzene metabolites are suspected as responsible for the hematotoxicity. The metabolites may bind covalently to cellular macromolecules (e.g., proteins, DNA, and RNA), causing disruption of cell growth and replication. The rate of benzene metabolism in bone marrow is lower than that in the liver.

Approximately 50% of absorbed benzene is excreted unchanged via the lungs over a 36-hour period, depending on exercise level and amount of body fat. Respiratory elimination is triphasic, with approximate half-lives of 1 hour, 3 hours, and greater than 15 hours. Urinary excretion of metabolites, primarily phenol, is another important pathway for elimination. Most of the phenol is excreted in the form of sulfate esters and glucuronides. After a single exposure, urinary excretion of phenol and hydroquinone is highest within the first 24 hours and is essentially complete within 48 hours.

- **Benzene is absorbed well after inhalation or ingestion; in comparison, dermal absorption is slow.**
- **Benzene is metabolized in the liver and bone marrow.**
- **Benzene excretion occurs via the lungs and urine.**

ATSDR

Physiologic Effects

- **Benzene affects primarily the CNS and hematopoietic system.**

Benzene exposure affects the central nervous system (CNS) and hematopoietic system and may affect the immune system. Death due to acute benzene exposure has been attributed to asphyxiation, respiratory arrest, CNS depression, or cardiac dysrhythmia. Pathologic findings in fatal cases have included respiratory tract inflammation, lung hemorrhages, kidney congestion, and cerebral edema.

Central Nervous System Effects

- **At very high concentrations, benzene rapidly causes CNS depression, which can lead to death.**

Acute benzene exposure results in classic symptoms of CNS depression such as dizziness, ataxia, and confusion. General agreement that benzene itself is responsible for central nervous system effects, and benzene metabolite(s) are responsible for the observed blood dyscrasias, has evolved from temporal studies and the fact that agents known to alter benzene metabolism also alter benzene hematotoxicity.

Hematologic Effects

- **All three blood cell lines may be adversely affected by benzene.**
- **Pluripotential stem cells and lymphocytic cells are the probable targets of benzene toxicity.**

All three cell lines—erythrocytes, leukocytes, and platelets—may be affected by benzene to varying degrees. Benzene's most likely target is the DNA of the pluripotential stem and lymphocytic cells. Hematologic abnormalities such as anemia, leukopenia, thrombocytopenia, or pancytopenia may occur after chronic exposure. Potentially fatal infections can develop if granulocytopenia is present, and hemorrhage can occur as a result of thrombocytopenia. Paroxysmal nocturnal hemoglobinuria, a rare paraneoplastic disorder, has been associated with benzene exposure. Cytogenetic abnormalities of bone marrow cells and circulating lymphocytes have been observed in workers exposed to benzene, abnormalities not unlike those observed after exposure to ionizing radiation. Myelodysplastic effects also can be seen in the bone marrow of persons chronically exposed to benzene.

Anemia

- **Benzene-induced aplastic anemia is caused by chronic exposure at relatively high levels.**

Fatal aplastic anemia was first reported in benzene-exposed workers in the nineteenth century. Aplastic anemia is a condition caused by bone marrow failure, resulting in hypoplasia with an inadequate number of all cell lines. Generally, benzene-induced aplastic anemia is caused by chronic exposure at relatively high doses. No overt cytopenic effects have been observed in persons exposed at the previous workplace permissible exposure limit of

10 ppm. Severe aplastic anemia typically has a poor prognosis and can progress to leukemia, whereas pancytopenia may be reversible.

Leukemia

The causal relationship between benzene exposure and leukemia, which has been suspected for over 50 years, has only recently been accepted widely. Lack of adequate epidemiologic data and difficulty in producing hematologic carcinogenicity in animals impeded a consensus. Cohort studies of benzene-exposed workers in several industries (sheet rubber manufacturing, shoe manufacturing, rotogravure printing) have demonstrated significantly elevated risk of leukemia, predominantly acute myelogenous leukemia, but also erythroleukemia and acute myelomonocytic leukemia. For benzene-induced leukemia the latency period is typically 5 to 15 years after first exposure. Patients with benzene-induced aplastic anemia have been observed to progress to a preleukemic phase and develop acute myelogenous leukemia. However, a person exposed to benzene may develop leukemia without having aplastic anemia.

- **Benzene-induced leukemia has a usual latency period of 5 to 15 years and, in many cases, is preceded by aplastic anemia.**

Studies addressing the risk of leukemia associated with low-level benzene exposures have been inconclusive. Death certificates do not reveal increased leukemia mortality among workers potentially exposed to low levels of hydrocarbons and other petroleum products. However, in one recent case-control study, significantly more patients with acute nonlymphocytic leukemia were employed as truck drivers, filling station attendants, or in jobs involving exposure to low levels of petroleum products than among the controls.

Other Effects

Several reports relate benzene exposure to a variety of lymphatic tumors including non-Hodgkin's lymphoma and multiple myeloma. Although this is plausible, no scientific proof of a causal relationship exists. The association between exposure to benzene and development of nonhematologic tumors remains inconclusive.

- **The evidence is insufficient to indicate a causal relationship between benzene and nonhematologic tumors.**

Information on the reproductive toxicity of benzene in humans is meager. Benzene has not been proven teratogenic in humans or animals at doses that do not produce maternal toxicity.

- **Teratogenic effects due to benzene have been observed in animals only at high exposure levels.**

ATSDR

Clinical Evaluation

History and Physical Examination

In addition to a thorough medical history and physical examination, important factors in evaluating a patient potentially exposed to benzene include a detailed family history of blood dyscrasias including hematologic neoplasms, genetic hemoglobin abnormalities, bleeding abnormalities, and abnormal function of formed blood elements; an environmental history focusing on activities and possible sources of benzene exposure at home; and an occupational history, including past exposures to hematologic toxicants such as solvents, insecticides, and arsenic. A history of ionizing radiation exposure, medications, and smoking should also be explored.

Signs and Symptoms

Acute Exposure

- Acute benzene exposure causes CNS depression.

Acute benzene toxicity is characterized by central nervous system depression. Symptoms may progress from light-headedness, headache, and euphoria, to respiratory depression, apnea, coma, and death. Benzene concentrations of about 20,000 ppm are fatal to humans within 5 to 10 minutes.

"Benzol jag" is a term workers use to describe symptoms of confusion, euphoria, and unsteady gait associated with acute benzene exposure. Depending on the magnitude of the dose, persons who have ingested benzene may experience these effects 30 to 60 minutes after benzene ingestion. In one case report, an oral dose of 10 milliliters (mL) was reported to produce staggering gait, vomiting, tachycardia, pneumonitis, somnolence, delirium, seizures, coma, and death.

Chronic Exposure

- Symptoms of chronic benzene exposure may be nonspecific, such as fatigue and anorexia.

Early symptoms of chronic benzene exposure are often nonspecific but show marked individual variability. By the time a physician is consulted, the bone marrow may have been affected significantly. For example, conditions that first bring the patient to medical attention are typically fever due to infection or manifestations of thrombocytopenia, such as hemorrhagic diathesis with bleeding from the gums, nose, skin, gastrointestinal tract, or elsewhere.

The clinical picture of patients chronically exposed to benzene was described well in 1938 in a cohort study of about 300 workers in the rotogravure printing industry. At that time, ink solvents and thinners containing 75% to 80% benzene by volume were used in the pressroom. Initial physical examination of the workers was relatively unrevealing, but of those tested, 22 persons had severe hematologic abnormalities. Follow-up of the workers a year after exposure ceased suggested that the effects of benzene can persist or can evolve over time. Most patients recover after exposure ceases.

Laboratory Evaluation

Laboratory evaluation of benzene-exposed persons should include the following:

- **Hematologic abnormalities are the primary concern in benzene exposure.**

> CBC with differential, hematocrit, hemoglobin, erythrocyte count, erythrocyte indices (MCV, MCH, MCHC), and platelet count.

Plasma folate and vitamin B_{12} levels may be used to rule out megaloblastic anemia if the MCV is elevated.

The above laboratory tests will detect hematologic abnormalities that have been associated with relatively high levels of exposure to benzene. Persons with blood dyscrasias that persist after removal from exposure should be evaluated by a hematologist. Bone marrow aspiration and biopsy may be useful in narrowing the differential diagnosis in some cases.

Direct Biologic Indicators

Measurement of benzene in breath and blood can be useful in certain occupational settings. Because of benzene's relatively short biologic half-life, blood levels do not reflect cumulative body burden. A less invasive measurement of exposure in the workplace may be the benzene concentration in end-expired air. Studies show that 16 hours after an 8-hour exposure to benzene levels of 10 ppm and 1 ppm, steady-state exhaled benzene concentrations are 50 ppb and 10 ppb, respectively. However, these methods are not clinically useful for patients exposed to the low levels of benzene typically found in ambient air.

- **Measurement of benzene in blood and breath is generally not clinically useful in nonoccupational settings.**

- **Urinary phenol concentrations do not correlate with airborne benzene levels below 10 ppm.**

Urinary phenol concentrations generally correlate well with benzene exposure at concentrations above 10 ppm. Exposure to 10 ppm for 8 hours typically produces a postshift urinary phenol level of 45 to 50 milligrams per liter (mg/L). With exposure to air levels below 10 ppm, high background excretion of phenol from dietary and other sources can render urinary phenol levels

ATSDR

unreliable. Unexposed persons rarely have urinary phenol levels greater than 20 mg/L.

Indirect Biologic Indicators

- **MCV and lymphocyte count may aid in the diagnosis of benzene toxicity.**
- **A bone marrow aspiration and biopsy will aid in identifying aplastic anemia.**

An increase in MCV and a decrease in total lymphocytes may be early signs of benzene toxicity. A finding of benzene-induced hematotoxicity in a patient should trigger consideration that this represents a sentinel event, indicating that other persons may have been similarly exposed.

If aplastic anemia is suspected, a bone marrow aspiration and biopsy should be performed. Aspiration of the marrow space often produces no sample (dry tap) in patients with aplastic anemia; however, a dry tap is not diagnostic of aplastic anemia; therefore, a biopsy specimen should be obtained as well and examined for architecture and cellularity. In aplastic anemia, only the empty reticular meshwork of the marrow is evident with fat cells replacing all or most of the hematopoietic tissues. Islands of residual hematopoiesis may be seen, but the overall cellularity typically is less than 25%. Chromosomal changes consistent with myelodysplasia are seen on cytogenetic analysis.

Challenge

(3) What should be included in the problem list of the patient described in the case study?

_____

_____

(4) Additional Information for the Case Study: A bone marrow aspiration reveals fibrous and fatty structures with very few spicules including mononuclear phagocytes, reticulum cells, and plasma cells. Rare promyelocytes and megaloblastic nucleated erythroid cells are present. No megakaryocytes are observed.

What differential diagnosis do the patient's hematologic results suggest?

_____

_____

(5) What additional laboratory testing would you recommend?

_____

_____

Treatment and Management

Acute Exposure

Treatment for persons acutely exposed to benzene is generally supportive and symptomatic. Immediate removal of the patient from exposure and administration of oxygen and cardiopulmonary resuscitation measures are the first consideration. In cases of ingestion, respiratory distress may indicate pulmonary aspiration of gastric contents.

- **There is no antidote for acute benzene poisoning.**
- **Treatment for benzene toxicity is supportive and symptomatic.**

Contaminated clothing and shoes should be removed from an exposed person as soon as possible. If the skin or eyes have contacted liquid benzene, immediately wash the exposed skin with soap and copious water, and irrigate the eyes with running water for 3 to 5 minutes or until irritation ceases.

In cases of ingestion, emesis is recommended in alert adult patients if less than 1 hour has passed since ingestion. However, if CNS or respiratory depression are present or likely, emesis is contraindicated. Care must be taken to avoid aspiration of stomach contents during vomiting because benzene can produce a severe chemical pneumonitis. Gastric lavage may be preferable to emesis if large amounts of benzene have been ingested or if the patient is seen more than 1 hour after ingestion. Activated charcoal decreases benzene absorption in experimental animals, and the benefits are likely to be similar in humans.

When medically indicated, epinephrine should be used cautiously with careful cardiac monitoring. Benzene is one of several solvents that may increase the susceptibility of the myocardium to the dysrhythmogenic effects of catecholamines.

Chronic Exposure

In treating persons chronically exposed to benzene, the most important actions are to remove the patient from the source of benzene exposure and to prevent further exposure. Benzene-induced depression of blood elements generally reverses after exposure is terminated. Chronically exposed patients whose hematologic results do not return to normal despite removal from exposure should be managed in consultation with a hematologist or oncologist. Chemotherapy and bone marrow transplants are therapeutic options for leukemia and aplastic anemia, respectively.

- **Once chronic exposure to benzene ceases, hematologic test results typically return to normal.**

ATSDR

Challenge

(6) What are some key considerations in the treatment for the patient in the case study?

(7) What is the prognosis for this patient? What follow-up care should he receive?

Standards and Regulations

Workplace

Air

- **The current permissible exposure limit for benzene is 1 ppm.**

In 1987, the Occupational Safety and Health Administration (OSHA) instituted a permissible exposure limit for benzene of 1 ppm, measured as an 8-hour time-weighted average (TWA), and a short-term exposure limit of 5 ppm (Table 1). These legal limits were based on studies demonstrating compelling evidence of health risk to workers exposed to benzene. The risk from exposure to 1 ppm for a working lifetime has been estimated to be 5 excess leukemia deaths per 1000 employees exposed. (This estimate assumes no threshold for benzene's carcinogenic effects.) OSHA has also established an action level of 0.5 ppm to encourage even lower exposures in the workplace.

The National Institute for Occupational Safety and Health (NIOSH) recommends an exposure limit of 0.1 ppm as a 10-hour TWA. NIOSH also recommends that benzene be handled in the workplace as a human carcinogen.

Table 1. Standards and regulations for benzene

Agency *	Focus	Level	Comments
ACGIH	Air-workplace	10 ppm	Advisory; 8-hour TWA†; suspected human carcinogen
NIOSH	Air-workplace	0.1 ppm	Advisory; 10-hour TWA
		1.0 ppm	15-min ceiling limit
OSHA	Air-workplace	1 ppm	Regulation; 8-hour TWA
		5 ppm	15-min STEL§
EPA	Drinking water	5 ppb	Regulation; maximum contaminant level
FDA	Food	N/A	Regulation; may be used only as a component of packaging adhesives

* ACGIH = American Conference of Governmental Industrial Hygienists; EPA = Environmental Protection Agency; FDA = Food and Drug Administration; NIOSH = National Institute for Occupational Safety and Health; OSHA = Occupational Safety and Health Administration

† TWA (time-weighted average) = time-weighted average concentration for a normal workday and a 40-hour workweek to which nearly all workers may be repeatedly exposed.

§ STEL (short-term exposure limit) = usually determined by a 15-minute sampling period.

Environment

Air

Benzene has been designated as a hazardous air pollutant under section 112 of the Clean Air Act. EPA has not promulgated a specific ambient air standard for benzene but has imposed restrictions designed to lower industrial emissions of benzene by 90% over the next 20 years. In addition, regulations have been proposed that would control benzene emissions from industrial solvent use, waste operations, transfer operations, and gasoline marketing. At gas stations, proposed rules would require new equipment restricting benzene emissions while dealers' storage tanks are being filled.

❑ **EPA restricts benzene emissions from specific point sources.**

ATSDR

Water

- **The maximum contaminant level of benzene in drinking water is 5 ppb.**

The National Primary Drinking Water Regulations promulgated by EPA in 1987 set a maximum contaminant level for benzene of 0.005 ppm (5 ppb). This regulation is based on preventing benzene leukemogenesis. The maximum contaminant level goal (MCLG), a nonenforceable health goal that would allow an adequate margin of safety for the prevention of adverse effects, is zero benzene concentration in drinking water.

Food

- **FDA prohibits the use of benzene in foods.**

Effective April 1988, the Food and Drug Administration has mandated that benzene can only be an indirect food additive in adhesives used for food packaging.

Challenge

(8) The lawyer for the family of the patient in the case study approaches you and asks you to establish causality between the patient's condition and the benzene in the drinking water. How would you do so?

Suggested Reading List

Reviews

Austin H, Delzell E, Cole P. Benzene and leukemia. A review of the literature and a risk assessment. Am J Epidemiol 1988;127(3):419-39.

Goldstein BD. Benzene toxicity. State Art Rev Occup Med 1988;3:541-54.

Goldstein BD. Introduction: Occam's razor is dull. Environ Health Perspect 1989;82:3-6.

Marcus WL. Chemical of current interest–benzene. Toxicol Ind Health 1987;3(1):205-66.

Hematologic Effects

Aksoy M. Benzene as a leukemogenic and carcinogenic agent. Am J Ind Med 1985;8:9-20.

Infante PF, Rinsky RA, Wagoner JK, Young RJ. Leukaemia in benzene workers. Lancet 1977;2:76-8.

Infante PF, White MC. Projections of leukemia risk associated with occupational exposure to benzene. Am J Ind Med 1985;7:403-13.

Runion HE, Scott LM. Benzene exposure in the United States, 1978-1983: an overview. Am J Ind Med 1985;7:385-93.

Risk Assessment

Rinsky RA, Smith AB, Hornung R, et al. Benzene and leukemia: an epidemiologic risk assessment. N Engl J Med 1987;316:1044-9.

Related Government Publications

Agency for Toxic Substances and Disease Registry. Toxicological profile for benzene. Atlanta: US Department of Health and Human Services, Public Health Service, 1989. NTIS report no. PB/89/209464/AS.

Environmental Protection Agency. Health effects assessment for benzene. Cincinnati, OH: US Environmental Protection Agency, Office of Health and Environmental Assessment, 1984. Report no. EPA/540/1-86/037.

Sources of Information

More information on the adverse effects of benzene and the treatment and management of benzene-exposed persons can be obtained from ATSDR, your state and local health departments, and university medical centers. *Case Studies in Environmental Medicine: Benzene Toxicity* is one of a series. For other publications in this series, please use the order form on the back of page 21. For clinical inquiries, contact ATSDR, Division of Health Education, Office of the Director, at (404) 639-6204.

In addition to other resources, ATSDR has created a National Exposure Registry for benzene. This registry is one of a series mandated by the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA). ATSDR, in cooperation with the states, will establish and maintain national registries of (1) persons exposed to substances and (2) persons with serious illness or diseases possibly due to

ATSDR

exposure. The registries will collect information on the effects of low-level exposures of long duration (i.e., the exposures typically found in populations surrounding hazardous waste sites) and the health outcomes for populations receiving a one-time, high-level environmental exposure (such as those experienced at chemical spill sites). The registries will facilitate the identification and subsequent tracking of persons exposed to a defined substance at selected sites and will coordinate the clinical and research activities involving the registrants. For further information on the benzene registry, please contact ATSDR Division of Health Studies, Office of the Director, at (404) 639-6200.

Answers to Pretest and Challenge Questions

Pretest questions are found on page 1; answers are in (3) through (7) below. Challenge questions begin on page 3.

(1) Some important areas to explore include amounts and duration of exposure from the following sources:

- water supply (ingestion)
- water supply (inhalation or dermal absorption during bathing and laundering)
- ambient air (fugitive emissions from the chemical plant during its operation and since it was abandoned 9 years ago)
- occupation (activities, conditions, and time spent as a diesel mechanic)
- workplace conditions (cleaning of machinery parts, solvents used, protective equipment worn, and the adequacy of ventilation)
- home environment (use of consumer products that might contain benzene, exposure to personal or passive cigarette smoke)

(For more information, see *Case Studies in Environmental Medicine: Taking an Exposure History,* ATSDR, October 1992.)

(2) Theoretically, a person could be at increased risk of benzene's adverse effects if he or she encountered agents or conditions that increased the rate of formation of toxic benzene metabolites through induction of the MFO system. Potential agents include MFO-inducing drugs (e.g., phenobarbital, alcohol); conditions include those causing rapid synthesis of bone marrow. The patient only occasionally drinks beer and did not take medications before his illness, and so he avoids the risk factors of alcohol and medications. However, if the patient is suffering from a hematologic abnormality, as his symptoms and laboratory evaluation suggest, he will have increased risk if benzene exposure continues.

Other persons in the case who may be at increased risk of benzene exposure are those who have had contact with the water supply for a prolonged period of time, although no data exist to quantitate the risk. Included are persons who have lived in the patient's household and members of the community who share the water supply. Community and household members who are at increased risk of benzene's adverse effects theoretically include those with rapidly synthesizing bone marrows and persons with increased MFO-mediated metabolism (e.g., heavy drinkers).

(3) The patient's problem list includes a clotting disorder, fatigue, ecchymoses and petechiae, and anorexia with concomitant weight loss.

(4) The hematology study reveals significant thrombocytopenia, leukopenia, and erythropenia. Pancytopenia is caused by the accelerated destruction or decreased production of all cell lines including red blood cells, white blood cells, and platelets. Bone marrow disorders are likely to be the cause, and could result from the following: drug and chemical toxicity (such as benzene toxicity), radiation, infection, nutrient deficiencies (e.g., vitamin B_{12} and folate), hypersplenism, and marrow replacement syndromes.

(5) Additional testing for the patient might include coagulation factors, evaluation for infectious agents, and assessment of nutrient status. Evaluation of the bone marrow should include a search for malignant cells. Cytogenetic abnormalities, if observed, may be helpful in the evaluation but are not definitive.

(6) The patient must be removed from exposure to benzene and other hematologic toxicants. His home water for drinking and personal purposes should be obtained from a source with no detectable level of

benzene. Work exposure to toxic chemicals must be carefully evaluated. Adequate nutrients (vitamins and protein source) in his diet should be assured. Care to prevent injury and bleeding must be exercised until proper blood coagulation (platelets and other factors) has returned, and the patient should be carefully monitored for infection in the event of severe granulocytopenia. Prophylactic antibiotics and blood transfusions should be avoided unless a significant deterioration of his condition becomes evident.

(7) The prognosis is generally good for the resolution of the macrocytosis. Although this patient has a significant aplastic anemia, it is possible for his bone marrow to recover slowly if the damage has not reached an irreversible stage. Supportive treatment will be needed for many months. Because of the continued risk of leukemia, the patient should receive medical surveillance consisting of regularly scheduled examinations and appropriate testing of hematologic function. The peripheral smear and blood count will permit monitoring of early changes of the patient's condition. Bone marrow biopsy should be repeated in a few weeks to confirm initial findings and observe an expected bone marrow recovery.

(8) One step in your quest to establish a causal relationship between benzene-contaminated home water and the patient's condition would be to further investigate competing causes of low blood counts for this patient (e.g., drugs, radiation exposure, family history), keeping in mind that most cases of aplastic anemia are idiopathic. You would also need to explore the patient's potential exposure to chemicals other than benzene that might cause hematologic disorders. Finally, assuming the patient's condition is due to benzene exposure, you would need to weigh the significance of benzene sources other than the drinking water. For example, the patient is a diesel mechanic and most likely has inhalation and dermal exposure to gasoline (which contains benzene) at work. You would need to determine the amounts of benzene each source might have contributed to the patient's exposure. (See answer number 1 above.)

For the patient in the case study, as for most exposure cases, it will not be an easy matter to establish causality, and there is no precedent for a person developing hematologic abnormalities from benzene in drinking water.

ATSDR June1990

Case Studies in Environmental Medicine

Chromium Toxicity

Environmental ALERT . . .

- ☑ *Chromium (III) is an essential nutrient, which can be toxic in large doses.*
- ☑ *The toxicity of chromium compounds depends on the oxidation state of the metal.*
- ☑ *Occupational exposure to chromium (VI) has been associated with increased incidence of lung cancer.*
- ☑ *The efficacy of chelation therapy in chromium poisoning has not been proven.*

This monograph is one in a series of self-instructional publications designed to increase the primary care provider's knowledge of hazardous substances in the environment and to aid in the evaluation of potentially exposed patients. The Agency for Toxic Substances and Disease Registry (ATSDR) and the Centers for Disease Control (CDC) designate this continuing medical education activity for 1 credit hour in Category 1 of the Physician's Recognition Award of the American Medical Association and 0.1 continuing education units for other health professionals. See pages 21 to 23 for further information.

Guest Contributor: Vikas Kapil, DO, MPH
Guest Editor: James Keogh, MD, MPH

Peer Reviewers: Charles Becker, MD; Jonathan Borak, MD; Joseph Cannella, MD; Bernard Goldstein, MD; Alan Hall, MD; Richard J. Jackson, MD, MPH; Jonathan Rodnick, MD; Robert Wheater, MS; Brian Wummer, MD

U.S. DEPARTMENT OF HEALTH & HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry

Case Study

Chronic skin ulcers and respiratory irritation in a 35-year-old handyman

A 35-year-old man is seen at your family practice office near a large Midwestern city with complaints of "allergies" and sores on his hands and arms. Over the past 2 to 3 months, the patient has noticed the onset of "runny nose," "sinus drainage," dry cough, and occasional nosebleeds (both nares intermittently). There is no prior history of allergies. He has also had occasional nausea and is concerned because the sores and minor skin cuts on his hands do not seem to heal. The patient denies having fever, chills, dyspnea, or change in bowel or bladder habits, and he has not noticed excessive thirst or easy bruising. He recently began experiencing loss of appetite and weight loss without dieting.

With the exception of the complaints mentioned, review of systems is otherwise unremarkable. The patient has used various over-the-counter remedies for his respiratory problems without relief. He did, however, note significant improvement in symptoms when he visited his sister in Chicago for 5 weeks at the end of summer.

Medical history reveals only usual childhood diseases. Other than OTC decongestants, he is taking no medications. He denies use of illicit drugs, but admits to occasional social use of alcohol. For the last 16 years he has smoked 1 pack of low-tar cigarettes a day.

The patient has been employed as a mathematics teacher for 13 years; summers are usually spent in self-employment as a handyman. His hobbies include reading and tennis. Two years ago he moved into a ranch-style house located several hundred yards from a small manufacturing plant; a small pond intervenes. The home has central air conditioning and gas heat; it is supplied with well water and uses a septic sewage system. Four months ago the patient began digging up the sewage system to make repairs. It was shortly after he began digging that he first noticed the sores on his hands and forearms.

Physical examination reveals an alert white male with skin lesions on the exposed areas of the forearms and hands; edema of the hands is present. The dermal lesions include dermatitis and small circular areas with shallow ulcerated centers. ENT examination is unremarkable, and chest examination reveals a few scattered rhonchi that clear with coughing. His liver is slightly enlarged and tender to palpation. Cardiovascular, genito-urinary, rectal, and neurologic examinations are unremarkable.

Initial laboratory findings include evidence of 2+ proteinuria and hematuria, and slightly elevated bilirubin, SGOT (AST), and SGPT (ALT). Scrapings of the dermal lesions, done with potassium hydroxide (KOH) preparation, show no fungal elements or signs of infestation on microscopic examination. A nasal smear for eosinophils is within normal limits.

(a) Formulate an active problem list for this patient.

(b) What clues indicate this case may have an environmental etiology?

(c) What further information will you seek before making a diagnosis?

(d) What treatment will you recommend?

Answers to the Pretest can be found on page 19.

ATSDR

Exposure Pathways

- Chromium exists in three common stable valence states; in order of generally increasing toxicity, they are chromium (0), (III), and (VI).
- Chromium is released to air primarily by combustion processes and metallurgical industries.
- Nonoccupational sources of chromium include contaminated soil, air, and water.

Chromium is a hard, steel-gray metal highly resistant to oxidation even at high temperatures. It is the sixth most abundant element in the earth's crust, where it is combined with iron and oxygen in the form of chromite ore. The Soviet Union, South Africa, Albania, and Zimbabwe together account for 75% of world chromite production. Chromite ore has not been mined in the United States since 1961; in 1985 this country became completely dependent on importation for its primary chromium supply.

Chromium is used in three basic industries: metallurgical, chemical, and refractory (heat-resistant applications). In the metallurgical industry, chromium is an important component of stainless steels and various metal alloys. Metal joint prostheses made of chromium alloys are widely employed in clinical orthopedics. In the chemical industry, chromium is used primarily in paint pigments (chromium compounds can be red, yellow, orange, and green), chrome plating, leather tanning, and wood treatment. Smaller amounts are used in drilling muds, water treatment, catalysts, safety matches, copy machine toners, corrosion inhibitors, photographic chemicals, and magnetic tapes. Refractory uses of chromium include magnesite-chrome firebrick for metallurgical furnace linings and granular chromite for various other heat-resistant applications.

Chromium exists in a series of oxidation states from -2 valence to +6; the most important stable states are 0 (elemental metal), +3 (trivalent), and +6 (hexavalent). Chromium in chromite ore is in the trivalent state, whereas industrial processes also produce the elemental metal and hexavalent chromium. The health effects of chromium are at least partially related to the valence state of the metal at the time of exposure. Trivalent (Cr [III]) and hexavalent (Cr [VI]) compounds are thought to be the most biologically significant. Cr (III) is an essential dietary mineral in low doses, whereas certain compounds of Cr (VI) appear to be carcinogenic. Insufficient evidence exists to determine if Cr (III) or chromium metal can be human carcinogens.

Cr (III) and Cr (VI) are released to the environment primarily from stationary point sources resulting from human activities. Of the total atmospheric chromium emissions in the United States, approximately 64% is due to chromium (III) from fuel combustion (residential, commercial, and industrial) and from steel production; about 32% is due to chromium (VI) from chemical manufacture, chrome plating, and industrial cooling towers using chromate chemicals as rust inhibitors. A recent U.S. Environmental Protection Agency (EPA) report estimates that in the United States about 2840 metric tons of total chromium are emitted annually into the atmosphere (compared to approximately 110,000 tons of chromium metal produced each year).

Electroplating, leather tanning, and textile industries release relatively large amounts of chromium in surface waters. Solid wastes from chromate-processing facilities, when disposed of improperly in landfills, can be sources of contamination for groundwater, where the chromium residence time may be several years. The content of chromium in tap water in U.S. households is from 0.4 to 8.0 micrograms per liter (µg/L), which is slightly increased through use of stainless steel plumbing materials. (EPA's maximum contaminant level for chromium in drinking water is currently 50 µg/L.)

In the 1960s and 1970s, chromium-containing slag was used as landfill in residential, commercial, and recreational settings in over 100 locations in Hudson County, New Jersey. This fill contains chromium in carcinogenic forms and in concentrations acutely toxic in certain circumstances. Community exposure from this fill occurs in a variety of ways. Wind erosion of the soil can make slag particles airborne, increasing the opportunity for inhalation of chromium, and chromium compounds leached by rainwater have been found to migrate through cracks in soil, asphalt roadways, and masonry walls, forming high-content chromium crystals on their surfaces. In soil and roadways, these particles may be eroded by wind and foot traffic and carried as chromium-laden dust into homes and workplaces. Children playing in areas where the slag was used as fill may also be exposed through skin contact with chromium-contaminated dust, dirt, and puddles.

Less significant environmental sources of chromium include road dust contaminated by emissions of chromium-based catalytic converters or erosion products of asbestos brake linings, cement dust, tobacco smoke, and foodstuffs. Cigarettes contain 0.24 to 14.6 milligram chromium/kilogram, but neither the amount of chromium inhaled nor the chemical form is known. Processing and refining removes much of the normally small amount of chromium naturally present in foods.

Environmental and occupational sources of chromium exposure include the following:

Environmental	**Occupational**
Airborne emissions from	Welding of
• chemical plants	• alloys
• incineration facilities	• steel
Effluents from chemical plants	Leather tanning (soluble Cr [III])
Contaminated landfill	Chrome electroplating (soluble Cr [VI])
Cement dust	Chrome alloy production
Road dust from	Textile manufacturing
• catalytic converter erosion	Paints/Pigments (insoluble Cr [VI])
• asbestos brake lining erosion	Photoengraving
Tobacco smoke	Copier servicing

ATSDR

Challenge

(1) On further questioning, the patient described in the case study relates that when he had reached several feet in depth while digging to repair the sewage system, he noticed an oozing from the ground of sometimes yellowish, sometimes greenish, water; this persisted throughout the several weeks of digging. The nearby pond, which is murky, also has a generally yellow tint with small areas of greenish color at times. Suspecting an environmental link, you contact the local health department. Extremely high levels of chromium are found in the pond water, and the investigators inform you that the nearby plant is electroplating auto parts with chromium.

Discuss all sources and pathways by which this patient may be exposed to chromium.

Who's at Risk

- **Workers in industries producing and using chromium are at greatest risk of chromium's adverse effects.**
- **Risk assessment is currently underway for residents living on landfill derived from chromium-containing solid wastes.**

Workers in industries using chromium, especially stainless steel welding, chromate production, chrome plating, and chrome pigment industries, where exposure is primarily to Cr (VI), are at increased risk of chromium's effects. An estimated 175,000 workers may be exposed to Cr (VI) in the workplace on a regular basis; the number is much greater if exposure to other valence states of chromium are also considered. In many occupations, exposure is to both Cr (III) and Cr (VI) as soluble and insoluble materials.

Residents near chromate production facilities may be exposed to higher-than-background levels of chromium (VI). There is also concern that residents whose homes have been built on landfill using slag from smelters or chromate-producing facilities may be exposed to chromium through inhalation and dermal contact. Groundwater contamination may increase exposure in persons using well water as a drinking water source.

Coal and oil combustion contribute an estimated 1723 metric tons of chromium per year in atmospheric emissions; however, only 0.2% of this chromium is Cr (VI). In contrast, chrome-plating sources are estimated to contribute 700 metric tons of chromium per year to atmospheric pollution, but 100% is believed to be Cr (VI).

Despite air and water contamination from industrial pollution, no adverse health effects have been documented in persons residing near chromium point sources or in persons drinking chromium-contaminated water.

Challenge

(2) Besides the patient, who in the case study may be at risk of chromium exposure?

Biologic Fate

The entry routes of chromium into the human body are inhalation, ingestion, and dermal absorption. Occupational exposure generally occurs through inhalation and dermal contact, while the general population is exposed most often by the oral route through chromium content in soil, food, and water.

Rates of chromium uptake from the gastrointestinal tract are relatively low and depend on a number of factors, including valence state (with Cr [VI] more readily absorbed than Cr [III]), the chemical form (with organic chromium more readily absorbed than inorganic chromium), the water solubility of the compound, and gastrointestinal transit time. In humans and animals, less than 1% of inorganic Cr (III) and about 10% of inorganic Cr (VI) is absorbed from the gut; the latter amount is slightly higher in a fasting state.

The percentage of chromium absorption from the lungs cannot be estimated. Data from a few animal experiments indicate that with equal solubility, Cr (VI) compounds are absorbed more readily than Cr (III) compounds, probably because Cr (VI) readily penetrates cell membranes. Data from volunteers and indirect evidence from

- **Cr (VI) is better absorbed from the lungs, gut, and skin than Cr (III).**
- **After absorption, Cr (VI) is reduced to Cr (III).**
- **The difference in bioavailability and bioactivity between Cr (III) and Cr (VI) may account for the differences in toxicity.**
- **Only Cr (III) is excreted, primarily in the urine.**

occupational studies indicate that absorption of certain Cr (VI) compounds can occur through intact skin.

After entering the body from an exogenous source, Cr (III) does not readily cross cell membranes, but binds directly to transferrin, an iron-transporting protein in the plasma. In contrast, Cr (VI) after absorption is rapidly taken up by erythrocytes and reduced to Cr (III) inside the cell. Regardless of the source, Cr (III) is widely distributed in the body and accounts for most of the chromium in plasma or tissues. The greatest uptake of Cr (III) as a protein complex is by bone marrow, lungs, lymph nodes, spleen, kidney, and liver. Autopsies reveal chromium levels in the lungs are consistently higher than levels in other organs.

Excretion of chromium occurs primarily via the urine with no major retention in organs. In humans, the kidney excretes about 60% of an absorbed Cr (VI) dose in the form of Cr (III) within 8 hours of ingestion. Approximately 10% of an absorbed dose is eliminated by biliary excretion, and smaller amounts are excreted in hair, nails, milk, and sweat. Clearance from plasma is generally rapid (within hours), while elimination from tissues is slower (half-life of several days). In volunteers, administered doses of Cr (VI) were more rapidly eliminated than those of Cr (III).

Challenge

(3) Analysis of blood and urine specimens from the patient described in the case study reveals an elevated Cr (III) serum and urine concentration. Assuming the patient was exposed only to chromium (VI), explain the presence of chromium (III) in each of these body fluids.

Physiologic Effects

Chromium (III), an essential dietary element, plays a role in maintaining normal metabolism of glucose, fat, and cholesterol. Chromium's nutritional role has not been thoroughly delineated, but it appears to potentiate insulin action, probably in the form of glucose tolerance factor (GTF). The estimated safe and adequate daily intake of chromium for adults is in the range of 50 to 200 micrograms a day, although data are insufficient to establish a recommended daily allowance.

- **Cr (III) is an essential trace mineral in human nutrition.**

Dietary chromium deficiency is relatively uncommon; most cases occur in persons with special problems such as total parenteral nutrition, diabetes, or malnutrition. Chromium deficiency is characterized by glucose intolerance, glycosuria, hypercholesterolemia, decreased longevity, decreased sperm counts, and impaired fertility. In one patient receiving total parenteral nutrition, a peripheral neuropathy was corrected after chromium supplementation.

- **Because Cr (VI) is a powerful oxidizing agent, exposure can cause irritating and corrosive effects.**
- **The target organ of inhaled chromium is the lung; the kidneys, liver, skin, and immune system may also be affected.**

Major factors governing the toxicity of chromium compounds are oxidation state and solubility. Chromium (VI) compounds, which are powerful oxidizing agents and, as such, tend to be irritating and corrosive, appear to be much more toxic systemically than chromium (III) compounds, given similar amounts and solubilities. Although mechanisms of biologic interaction are uncertain, this differing toxicity may be related to the ease with which Cr (VI) can pass through cell membranes and its subsequent intracellular reduction to reactive intermediates.

Skin Effects

Chromic acid, dichromates, and other Cr (VI) compounds are not only powerful skin irritants but can also be corrosive. On broken skin, a penetrating round ulcer may develop. Common sites for these persistent ulcers ("chrome holes") include the nail root, knuckles and finger webs, back of the hands, and forearms. The characteristic chrome sore begins as a papule, forming an ulcer with raised hard edges. Ulcers may penetrate deep into soft tissue or become the site of secondary infection, but are not known to lead to malignancy. The progression to ulceration is generally painless, suggesting toxicity to peripheral sensory nerves. The lesions heal slowly and may persist for months.

- **Severe dermatitis and skin ulcers can result from contact with Cr (VI) salts.**
- **Chromium compounds can be sensitizers as well as irritants.**

At concentrations below those resulting in irritation, skin sensitivity is the most common effect after exposure to chromium compounds, especially Cr (VI) compounds. Up to 20% of chromium workers develop dermatitis. Allergic dermatitis with eczema has been reported in printers, cement workers, metal workers, painters, and leather tanners. Data suggest that a Cr (III)-protein complex is responsible for the allergic reaction, with Cr (III) acting as the hapten.

Respiratory Tract Effects

- **When inhaled, chromium (VI) is a respiratory tract irritant and may cause pulmonary sensitization.**
- **Chronic chromium inhalation increases the risk of lung cancer.**

Human occupational experience clearly indicates that, when inhaled, chromium (VI) is a respiratory tract irritant, resulting in airway irritation, airway obstruction, and possibly lung cancer. Dose, exposure duration, and the specific compound involved determine chromium's effects.

Pulmonary irritant effects after prolonged inhalation of chromate (VI) dust may include chronic irritation, congestion and hyperemia, chronic rhinitis, polyps of the upper respiratory tract, tracheobronchitis, and chronic pharyngitis. X-ray abnormalities reflect enlargement of the hilar region and lymph nodes, increased peribronchial and perivascular lung markings, and adhesions of the diaphragm. Consistent associations have been found between employment in the primary chromium industries and the risk for respiratory cancer (see Carcinogenic Effects section).

Pulmonary sensitization resulting in an asthmatic response is more common from Cr (VI) than from Cr (III). A delayed anaphylactoid reaction was reported in a male worker occupationally exposed to chromium vapors from chromium (VI) trioxide baths and chromium fumes from steel welding. A subsequent inhalation challenge with sodium chromate resulted in a reaction including late onset urticaria, angioedema, and bronchospasm accompanied by tripling of plasma histamine levels.

Many cases of nasal mucosa injury (inflamed mucosa, ulcerated septum, perforated septum) have been reported in workers exposed to Cr (VI) in chrome-plating plants and tanneries. A 1983 study of 43 chrome-plating plants in Sweden, where workers were exposed almost exclusively to chromic (VI) acid, revealed that all workers with nasal mucosa ulceration or perforation were periodically exposed to at least 20 $\mu g/m^3$ when working near the plating baths. (The current U.S. permissible exposure level in the workplace for chromates and chromic acid is 100 $\mu g/m^3$ over an 8-hour period.) The period of exposure for workers experiencing nasal mucosal ulceration varied from 5 months to 10 years.

Renal Effects

Studies of welders and chromium platers have found that workers with higher levels of exposure to airborne chromium (typically greater than 20 µg/m³) show damage to renal tubules. Adverse renal effects have been reported in humans after inhalation, ingestion, and dermal exposure to chromium. Renal effects in animals occurred only after parenteral administration of large doses.

- **Low-dose, chronic chromium exposures generally cause only transient renal effects.**
- **Acute Cr (VI) exposure may result in renal tubular necrosis.**

Although glomerular injury has been noted in chromium workers, the predominant renal injury is tubular, with low doses acting specifically on the proximal convoluted tubules. Low-dose, chronic chromium exposure typically results only in transient renal effects. Elevated urinary β_2-microglobulin levels (an indicator of renal tubular damage) have been found in chrome platers, and higher levels generally have been observed in younger persons exposed to higher Cr (VI) concentrations. However, in a study of tannery workers (Cr [III] exposure) whose duration of employment ranged from 1 month to 30 years, urinary β_2-microglobulin levels were within normal limits, even though urinary chromium levels clearly indicated chromium exposure. A suggested urinary threshold for nephrotoxic effects is 15 µg chromium/g creatinine.

Hepatic Effects

- **Chromium (VI) may cause mild to moderate liver abnormalities.**

Acute chromium exposure can result in hepatic necrosis. External chromic acid burns over 20% of a worker's body resulted in severe liver damage and acute renal failure. Limited data indicate that chronic inhalation of chromium compounds also can cause hepatic effects. Acute hepatitis with jaundice was reported in a woman who had been employed for 5 years at a chromium-plating factory. Tests revealed large amounts of urinary chromium, and liver biopsy showed abnormalities. Three coworkers exposed to chromic acid mists from the plating baths for 1 to 4 years also had mild to moderate liver abnormalities, as determined by liver function tests and liver biopsies.

Carcinogenic Effects

- **Occupational exposure to Cr (VI) has long been associated with increased lung cancer mortality.**
- **Latency for chromium-induced lung cancer is greater than 20 years; exposure duration may be as short as 2 years.**

Epidemiologic studies of occupational cohorts exposed to chromium aerosols provide clear evidence of carcinogenicity. In one key epidemiologic study involving workers at a chromate production plant who had worked for more than 1 year from 1931 to 1949, the percentage of deaths due to lung cancer was 18.2%; 1.2% was expected. For the 322 workers first employed from 1931 to 1937, the percentage of deaths due to lung cancer was close to 60%, with a latency period of approximately 30 years. Studies of workers in the chromium pigment, chrome-plating, and ferrochromium industries

also suggest a statistically significant association between worker exposure to chromium and lung cancer. Increased lung cancer mortality has been associated with occupational exposures as short as two or three years. On the basis of these and other studies, EPA and the International Agency for Research on Cancer (IARC) have classified inhaled chromium (VI) as a known human carcinogen. Chromium (III) has not been classified as a human carcinogen by the National Toxicology Program, EPA, or IARC.

Although epidemiologic evidence strongly points to the hexavalent form of chromium as the agent in carcinogenesis, solubility and other characteristics of chromium compounds may be important in determining cancer risk. Data from animal studies do not resolve the issues of identities and potencies of various chromium-containing compounds as respiratory carcinogens. No chromium compound has been unequivocally shown to cause a significant increase in the number of neoplasms in experimental animals after exposure by natural routes (inhalation, ingestion, or dermal absorption), unless the animals were exposed until dead. (Standard protocols for animal experiments involve termination after 24 months.) However, intratracheal instillation, intrabronchial implantation, or injection of various chromium-containing compounds have produced tumors at the site of application in some cases.

No cancers, other than lung cancer, are associated with occupational chromium exposure. All pathologic cell types have occurred in chromium-induced lung cancers; however, small cell and poorly differentiated cancers predominate. Findings of some epidemiologic studies and animal experiments suggest chromium is also associated with nonrespiratory cancers, but the evidence is insufficient to consider the nonrespiratory cancers to be of a causal nature.

Reproductive and Developmental Effects

- **Data indicate chromium is teratogenic in animals.**
- **Potential reproductive effects of chromium in humans have not been adequately investigated.**

Chromium (III) is an essential element that is transported to the developing fetus. Less than 0.5% of Cr (III) was found to cross the placenta in mice when the chromium was administered as an inorganic salt, but 20% to 25% was found in litters when chromium was administered in a biologically active form, brewer's yeast. Adverse developmental effects in animals include cleft palate, hydrocephalus, delayed ossification, edema, and incomplete neural tube closure. Data are unavailable implicating chromium in adverse human reproductive or developmental effects.

Challenge

(4) Could chromium toxicity account for the symptoms experienced by the patient described in the case study? Explain.

(5) Is the patient at increased risk of chromium-induced lung cancer?

Clinical Evaluation

History and Physical Examination

Often there are no clear diagnostic clues in chromium-poisoned patients. A thorough history is therefore critical in evaluating a potentially exposed person. The patient's recent activities are important when health effects other than cancer are the major concern. Occupation, location of residence and workplace in relation to industrial facilities or hazardous waste sites, and source of drinking water supply should be investigated. In patients with known chronic chromium exposure, the physical examination should include evaluation of the respiratory system (if inhalation is involved), kidneys, liver, and skin.

❑ **If chromium exposure is suspected, the respiratory system, kidneys, liver, and skin should be evaluated.**

ATSDR

Signs and Symptoms

Acute Exposure

- Ingestion of a lethal dose of chromate may result in cardiovascular collapse due to severe hypovolemia.
- Sublethal doses of chromate may lead to renal and hepatic necrosis 1 to 4 days after ingestion.

Severe exposures to chromium compounds are rarely occupational or environmental, but are usually accidental or suicidal. Short-term, high-level exposure to Cr (VI) produces irritation at the site of contact including ulcers of the skin, irritation of the nasal mucosa, perforation of the nasal septum, and irritation of the gastrointestinal tract. Less is known about the acute toxicity of Cr (III) compounds, although they are generally believed to be less toxic.

About 1 gram of potassium dichromate (IV) is considered a lethal dose. Persons who ingested 5 grams or more experienced gastrointestinal bleeding, massive fluid loss, and death within 12 hours after ingestion. When the ingested dose was 2 grams or less, renal tubular necrosis and diffuse hepatic necrosis resulted and contributed to death in some cases. Typically, the kidney and liver effects develop 1 to 4 days after ingestion of a sublethal dose. Other symptoms of acute Cr (VI) ingestion include vertigo, thirst, abdominal pain, and vomiting. Oliguria, anuria, shock, convulsions, coma, and death can ensue. Gastrointestinal hemorrhage and coagulopathy may also occur. Acute chromium poisonings are often fatal regardless of the therapy employed.

Dermal contact with Cr (VI) compounds can result in severe systemic toxicity. Antiscabies ointment containing Cr (VI) resulted in necrosis of skin at application sites, nausea, vomiting, shock, coma, and death. In one case, severe nephritis and death followed cauterization of an open wound with chromium (VI) oxide, and an occupational fatality was described after an accident in which a worker was burned on the arms and trunk with hot potassium dichromate. Both of these cases involved broken rather than intact skin.

Chronic Exposure

- In occupational settings, the most commonly reported effects of chronic chromium exposure are contact dermatitis, and irritation and ulceration of the nasal mucosa.
- Less common are reports of hepatic and renal damage and pulmonary effects.
- Lung cancer is a potential long-term effect of chronic Cr (VI) exposure.

Repeated skin contact with chromium dusts may lead to incapacitating eczematous dermatitis with edema. Chromate dusts may also produce irritation of the conjunctiva and mucous membranes, as well as nasal ulcers and perforations. When a solution of chromate contacts the skin, it can produce penetrating lesions known as chrome holes or chrome ulcers, particularly in areas where a break in the epidermis is already present. These ulcers are usually painless but may persist for months. Acute hepatitis with jaundice has also been observed in workers chronically exposed to Cr (VI). Lung cancer is the most serious long-term effect.

Low-level environmental exposures have not resulted in adverse effects in human populations. Long-term studies in which animals

have been exposed to low levels of chromium in food or water have produced no harmful effects.

Laboratory Tests

A general medical workup for a patient with suspected chronic chromium exposure might include the following:

Screening Tests
- Complete blood count
- Blood panel
- Liver function tests (SGOT or AST, SGPT or ALT, and bilirubin)
- BUN and creatinine
- Urinalysis

Specialized Tests
- Blood and urine chromium levels
- $ß_2$-microglobulin

If chromium inhalation has occurred, a chest X ray, pulmonary function testing, and a nasal smear for eosinophils should be included.

Direct Biologic Indicators

When obtaining biologic specimens for chromium analysis, care must be taken to avoid sample contamination and chromium loss during collection, transportation, and storage. For example, use of stainless steel utensils to collect tissue samples may raise tissue chromium levels, as will stainless steel grinding and homogenizing equipment. Some plastic containers contain significant amounts of leachable chromium; therefore, specially prepared acid-washed containers should be obtained from the laboratory. Considerable care also must be taken in the analysis to minimize chromium volatilization during sample ashing.

- **Chromium can be measured in blood and urine; hair or nail analysis has no clinical value.**
- **The correlation between exposure levels and urinary chromium excretion is useful in occupational settings.**

Another difficulty in the available techniques is the inability to distinguish between Cr (III) and Cr (VI). This is particularly important in environmental samples since Cr (VI) has been associated with serious health hazards, whereas Cr (III) is of far less concern.

Blood or serum chromium levels. Blood distribution of chromium appears to be divided evenly between plasma and erythrocytes. In the absence of known exposure, whole blood chromium concentrations are in the range of 2.0 to 3.0 µg/100 mL; lower levels are seen in rural areas, and higher levels occur in large urban centers. Values above background levels are considered potentially toxic, but levels have not been correlated with specific physiologic effects. Chro-

ATSDR

mium rapidly clears from the blood, and measurements relate only to recent exposure.

Urinary chromium levels. Wide individual variation in metabolism and rapid depletion of body burden limit the value of urinary chromium monitoring. Urinary chromium excretion reflects absorption over the previous 1 or 2 days only. If sufficient time has elapsed for urinary clearance, a negative biomonitoring result can occur even with injurious past exposure. Assuming no source of excessive exposure, urinary chromium values are typically less than 10 µg for a twenty-four-hour period.

In occupational settings, a urinary chromium concentration of 40 to 50 µg/L immediately after a workshift reflects exposure to air levels of 50 µg/m³ of soluble Cr (VI) compounds, a concentration associated with nasal perforations in some studies. The American Conference of Governmental Industrial Hygienists (ACGIH) intends to recommend a workplace biologic exposure index (BEI) for total urinary chromium as follows: no more than 10 µg chromium/g creatinine increase during a work shift, and a urinary value of less than 30 µg chromium/g creatinine at the end of the work week.

Chromium levels in hair and nails. Hair or nail analysis is of little use in evaluating an individual patient since it is impossible to distinguish chromium bound within the hair during protein synthesis from chromium deposited on the hair from dust, water, or other external sources. Populations with no known chromium exposure reportedly have hair levels ranging from 50 to 1000 ppm chromium.

Challenge

(6) Analysis of the tap water in the patient's home reveals a greenish tinge and a chromium concentration of 746 µg/L. Your diagnosis is chromium toxicity. Are there any other tests the patient should undergo?

(7) The patient described in the case study insists on obtaining a hair analysis. The chromium content of the hair sample is 1038 ppm. How will you interpret this result?

Treatment and Management

Acute Exposure

- **No proven antidote is available for chromium poisoning.**
- **Acute poisonings are often fatal regardless of therapy.**

Treatment in cases of acute, high-level chromium exposure is usually supportive and symptomatic. Supportive measures may include ventilatory support, cardiovascular support, and monitoring for renal and hepatic function. When renal function is compromised, urine alkalinization and maintenance of adequate urine flow are important. Progression to anuria is associated with poor prognosis.

If the eyes and skin are directly exposed, flush with copious amounts of water. Topical ascorbic acid has been successfully used to prevent chromium dermatitis and dermal burns caused by dichromate.

Gastric lavage with magnesium hydroxide or another antacid may be useful in cases of chromium ingestion. Fluid and electrolyte balance is critical. The efficacy of activated charcoal has not been proven. Hemodialysis, exchange transfusions, or chelating agents such as BAL (dimercaprol) or EDTA (ethylenediaminetetraacetic acid) have not been shown to be effective in the treatment of human poisoning. Orally administered ascorbic acid was found to be protective in experimental animals and was reported beneficial in at least one patient after chromium ingestion.

Chronic Exposure

- **Treatment consists of removal of the patient from further chromium exposure, reliance on the body's naturally rapid clearance of the metal, and symptomatic management.**

In most patients with chronic, low-dose exposure, no specific treatment is needed. The mainstay of management is removing the patient from further exposure and relying on the urinary and fecal clearance of the body burden. Although normal urinary excretion is quite rapid, forced diuresis has been used. Except in the lungs, only small amounts of chromium are retained several weeks after exposure has ceased. Dermatitis and liver and renal injury will not progress after removal from exposure and, in most cases, the patient will recover. Weeping dermatitis can be treated with 1% aluminum acetate wet dressings, and chrome ulcers can be treated with topical ascorbic acid.

If the exposure has been lengthy (i.e., 2 to 3 years), the increased risk of lung cancer should be discussed with the patient. Although no reliable tests are currently available to screen patients for lung cancer, the physician can intervene with advice and education in smoking cessation, exposure to other known pulmonary carcinogens, and in general, preventive health education. Annual chest X rays may be advisable in carefully selected cases.

ATSDR

Challenge

(8) What is the recommended treatment for the patient described in the case study?

Standards and Regulations

Table 1 summarizes the U.S. standards and regulations for chromium salts, which are discussed in more detail below.

The Workplace

Air

OSHA mandates an 8-hour time-weighted average of 100 µg/m³ for chromic acid and chromates.

In 1985, the Occupational Safety and Health Administration (OSHA) mandated an 8-hour workday, 40-hour workweek permissible exposure limit (PEL) of 100 µg CrO_3/m^3 for chromic acid and chromates (ceiling). For soluble Cr (VI) salts the PEL is an 8-hour time-weighted average (TWA) of 500 µg Cr/m³. For chromium metal and for insoluble salts the TWA is 1000 µg Cr/m³.

NIOSH's recommended exposure limit is a 10-hour TWA for carcinogenic Cr (VI) compounds of 1 µg Cr (VI)/m³. For noncarcinogenic Cr (VI) compounds (a category which includes chromic acid), the recommended exposure limit is 25 µg Cr (VI)/m³ as a 10-hour TWA and a 15-minute ceiling of 50 µg Cr (VI)/m³. Based on current evidence, NIOSH considers the noncarcinogenic Cr (VI) compounds to be the mono- and dichromates of hydrogen, lithium, sodium, potassium, rubidium, cesium, and ammonia, and chromic acid anhydride. Carcinogenic Cr (VI) compounds comprise any and all Cr (VI) materials not mentioned in the noncarcinogenic group above.

Environment

Air

EPA does not have an emission standard for chromium and, therefore, does not regulate chromium levels in ambient air.

No federal emission standard currently exists for chromium.

Drinking Water

EPA has a current enforceable standard of 50 µg/L (50 ppb) total chromium in drinking water. In May 1989, EPA recommended a maximum contaminant level (MCL) of total chromium in drinking water of 100 µg/L (100 ppb). Action on the proposed standard has received public comment, and action will likely be taken by EPA in December 1990.

The current maximum contaminant level for chromium in drinking water is 50 µg/L.

Table 1. Standards and regulations for chromium

Agency *	Focus	Level	Comments
ACGIH	Air -Workplace	50 µg/m^3	Advisory; TWA† to avoid carcinogenic risk from certain insoluble chromium compounds
NIOSH	Air -Workplace	1 µg/m^3	Advisory; TWA† (10-hour) for carcinogenic Cr (VI) salts
		25 µg/m^3	TWA† (10-hour) for noncarcinogenic Cr (VI) salts, including chromic acid
		50 µg/m^3	15-minute ceiling limit for noncarcinogenic Cr (VI) salts
OSHA	Air -Workplace	100 µg/m^3	Regulation; PEL§ for chromic acid and chromates (ceiling)
		500 µg/m^3	PEL§ for soluble chromic salts (8-hour TWA†)
		1000 µg/m^3	PEL for chromium metal and insoluble salts (8-hour TWA†)
EPA	Air-Environment	N/A	Under review
	Drinking-Water	50 µg/L	Regulation; current MCL¶ for total chromium; proposed MCL is 100 µg/L

* ACGIH = American Conference of Governmental Industrial Hygienists; EPA = Environmental Protection Agency; NIOSH = National Institute for Occupational Safety and Health; OSHA = Occupational Safety and Health Administration

† TWA (Time-Weighted Average) = time-weighted average concentration for a normal workday and 40-hour workweek to which nearly all workers may be repeatedly exposed

§ PEL (Permissible Exposure Limit) = an allowable exposure level in workplace air

¶ MCL (Maximum Contaminant Level) = enforceable standard for drinking water

ATSDR

Suggested Reading List

General Reviews

Burrows D, ed. Chromium: metabolism and toxicity. Boca Raton, Fl: CRC Press, Inc., 1983.

Mertz W. Clinical and public health significance of chromium. In: Clinical, biochemical and nutritional aspects of trace elements. New York: Alan R. Liss Inc., 1982:315-23.

Sawyer HJ. Chromium and its compounds. In: Zenz C, ed. Occupational medicine, principles and practical applications. Chicago: Year Book Medical Publishers, Inc.,1988: 531-9.

Carcinogenicity

Davies JM. Lung cancer mortality among workers making lead chromate and zinc chromate pigments at three English factories. Br J Ind Med 1984;41:158-69.

Hayes RB. Review of occupational epidemiology of chromium chemicals and respiratory cancer. Sci Total Environ 1988;71:331-9.

Levy LS, Martin PA, Venitt S. Correspondence: carcinogenicity of chromium and its salts. Br J Ind Med 1987;44:355-7.

Norseth T. The carcinogenicity of chromium. Environ Health Perspect 1981;40:121-30.

Norseth T. The carcinogenicity of chromium and its salts. Br J Ind Med 1986;43:649-51.

Renal Effects and Urinary Excretion

Kirschbaum BB, Sprinkel FM, Oken DE. Proximal tubule brush border alterations during the course of chromate nephropathy. Toxicol Appl Pharmacol 1981;52:19-30.

Lindberg E, Vesterberg O. Monitoring exposure to chromic acid in chromeplating by measuring chromium in urine. Scand J Work Environ Health 1983;9:333-40.

Lindberg E, Vesterberg O. Urinary excretion of proteins in chromeplaters, exchromeplaters and referents. Scand J Work Environ Health 1983;9:505-10.

Powers WJ, Gad SC, Siino KM, Pechman JC. Effects of therapeutic agents on chromium-induced acute nephrotoxicity. In: Serrone DM, ed. Chromium symposium 1986: an update. Pittsburgh, PA: Industrial Health Foundation, Inc., 1986:79-86.

Related Government Documents

Agency for Toxic Substances and Disease Registry. Toxicological profile for chromium. Atlanta: Department of Health and Human Services, Public Health Service, 1989. NTIS report no. PB/89/236665/AS.

Environmental Protection Agency. Health assessment document for chromium. Research Triangle Park, NC: Environmental Protection Agency, Environmental and Criteria Assessment Office, 1984. EPA report no. 600/8-83-014F.

Environmental Protection Agency. Health effects assessment for hexavalent chromium. Cincinnati, OH: Environmental Protection Agency, Environmental and Criteria Assessment Office, 1984. EPA report no. 540/1-86-019.

Environmental Protection Agency. Health effects assessment for trivalent chromium. Cincinnati, OH: Environmental Protection Agency, Environmental and Criteria Assessment Office, 1984. EPA report no. 540/1-86-035.

Answers to Pretest and Challenge Questions

Pretest

The Pretest can be found on page 1.

(a) A problem list for this patient would include the following:
upper and lower respiratory irritation
multiple skin lesions and edema of the hands
loss of appetite and weight loss
liver and renal dysfunction
cigarette smoking

(b) Information suggesting an environmental etiology includes the following: onset of the patient's symptoms coincide with activity outside the usual routine; the patient mentions he first noticed the sores on his hands and forearms while digging up the sewage system to make repairs. Another clue to a possible environmental cause is temporary relief of symptoms when the patient leaves his usual habitus, as occurred when he visited Chicago. Proximity of the patient's home to an industrial facility (i.e., the electroplating plant) is also an important clue.

(c) You may identify possible causes for the dermal lesions by consulting a dermatologist. The cause of the persistent (2 to 3 months) respiratory symptoms that do not respond to OTC decongestants in a person with no history of allergies should be pursued; the patient should be queried about whether the onset of symptoms coincided with the move to his home, whether odors have emanated from the plant, etc. More information regarding the patient's observations and activities while digging up the sewage system also may be helpful.

(d) See answer to Challenge question 8.

Challenge

Challenge questions begin on page 4.

(1) The most important pathways for possible chromium exposure in this case are dermal contact during the unearthing of the sewage system; inhalation of emissions from the plant or soil particles if the pond dries up; and ingestion, if the drinking water has been contaminated by effluents from the plant.

ATSDR

Minor sources (inhalation) of chromium may be road and cement dust, erosion products of brake linings and emissions from automotive catalytic converters, and tobacco smoke. Cigarettes contain 0.24 to 14.6 mg/kg chromium, although it is not known how much of this is inhaled. Foodstuffs (ingestion) generally contain extremely low chromium levels.

(2) If effluent from the plant has reached the groundwater, community residents who drink well water may be at risk. Airborne plant emissions may have also reached nearby residents. Workers at the plant who prepare the plating baths and work near them may be receiving significant exposure.

(3) Chromium (VI) is a powerful oxidizing agent. In the plasma and cells, it is readily reduced to chromium (III), which is excreted in the urine.

(4) Yes, persistent dermal ulcers, respiratory tract irritation, and pulmonary sensitization are all possible effects of chromium exposure.

(5) While it cannot be ruled out, it is unlikely that the dermal and inhalation chromium exposure of this patient will cause lung cancer. Persons who have developed lung cancer after chromium exposure were workers who had significant inhalation exposure for 2 years or longer. Because this patient's inhalation exposure is at ambient air levels and probably of 2 years duration at most, any increase in his relative risk would not be great. The patient should be advised to stop smoking cigarettes because smoking may act synergistically to increase risk and is itself a significant risk factor for lung cancer. The data is insufficient to estimate the risk from ingestion of the contaminated drinking water.

(6) If exposure was recent, chromium levels in blood or urine may be used to confirm exposure. Renal function should be tested (urinalysis, BUN, creatinine, and β_2-microglobulin) to determine if renal tubular damage has occurred.

(7) No useful interpretations can be drawn from the hair analysis. A result of 1038 ppm is beyond the range for unexposed persons (50 to 1000 ppm); however, the sample could have been environmentally contaminated with chromium from the water during bathing, or by chromium in ambient air polluted by the plant emissions. There are no standard methods for obtaining a hair sample nor for washing and preparing it for analysis, and these techniques can greatly influence results. Finally, there is no research that proves a correlation between chromium content of hair and exposure levels or physiologic effects; therefore, the result has no clinical significance.

(8) If the sources of chromium exposure can be eliminated for this patient, except for the skin lesions, no further treatment would be required. Topical ascorbic acid has been useful in the treatment of chrome ulcers and 1% aluminum acetate wet dressings can be used to treat the dermatitis.

This patient's case may be a sentinel for community exposure. You should contact the local health department, OSHA, and EPA to report your patient's adverse effects and discuss your suspicions of the chromium source. Chromium levels in and around the plant should be measured. If a hazard exists, workers should be provided proper protective gear, trained, and medically monitored. Since EPA does not currently have an emission standard, it may be difficult to abate the atmospheric source of chromium. Decontamination of the pond site may require regulatory action and litigation. Residents who use well water should be encouraged to use an alternate water source for drinking and cooking.

BRAIN INJURY, 1994, VOL. 8, NO. 3, 285–294

Case report

Neuropsychological toxicology of methylene diphenyl diisocyanate: a report of five cases

THOMAS J. REIDY† and JOHN F. BOLTER‡

†Independent Practice, Salinas, California, USA
‡The NeuroMedical Center, Baton Rouge, Louisiana, USA

(*Received 13 September 1992; accepted 17 October 1992*)

The neuropsychological functioning of five men suffering alleged physical, cognitive and behavioural changes following exposure to methylene diphenyl diisocyanate (MDI), an industrial chemical, was investigated in the present study. At the time of assessment, four of the five patients remained symptomatic despite having no contact with MDI for periods ranging from 5 to 9 months. All patients reported experiencing subjective symptoms consisting of respiratory distress, headaches, depression, irritability, forgetfulness, decreased calculating ability, word-finding problems and reduced concentration. While the pattern of neuropsychological deficits varied among the patients, common findings for the group included intact psychomotor, psychosensory, visuographic and language functions accompanied by deceased concentration, mental efficiency, rate of information processing, learning ability and abstract reasoning. All five patients also revealed significant emotional distress on an objective personality measure. In general, the neuropsychological test data support the presence of behavioural and cognitive correlates of CNS injury following exposure to MDI.

Introduction

The emergence of neuropsychological toxicology as a subspecialty of neuropsychology [1] has led to a proliferation of studies documenting cognitive and behavioural correlates of acute and subacute exposure to toxic chemicals in the workplace. A wide array of chemicals acting on the nervous system (for example, solvents, metals, pesticides and carbon monoxide) have been studied using epidemiological, experimental and case study formats [2–4].

The central nervous system (CNS) functions most adversely impacted by neurotoxins typically include attention, concentration, rate of information processing, memory, rate of new learning, psychomotor speed, fine motor dexterity, visuoconstructive ability and reaction time [2, 4, 5]. Vague subjective physical complaints and affective disturbance may also accompany neurotoxic exposure arising from either primary neurotoxin-induced effects [5, 7, 8], functional reaction to illness [9, 10] or mixed organic-functional psychological disturbance [11]. Various neurological symptoms have additionally been reported as sequelae following exposure to neurotoxic chemicals, including vestibular changes [12, 13], impaired colour discrimination [14, 15], olfactory hypersensitivity [16], optic neuropathy, peripheral neuropathy [5] and Parkinsonism [17].

Industrial workers are at great risk of potential toxic exposure, but few data are available on the CNS effects of many industrially employed chemicals. Methylene diphenyl diisocyanate (MDI), an organic isocyanide, is one such chemical used in a

Address correspondence to: Thomas J. Reidy, 154 Central Avenue, Solinas, CA 93901, USA.

0269-9052/94 $10.00

variety of industrial applications and is the key ingredient in spray foam packaging. Although infrequent, hazardous airborne exposures can occur with MDI if it is heated [18]. The current permissible exposure limit for MDI is 0·005 parts per million (ppm). While it is known that workers exposed to MDI are at risk for adverse respiratory effects, including occupational asthma, allergic disease and immunologic injury [19], no studies pertaining to the neuropsychological effects of MDI exposure have been reported in the literature. However, a related isocyanide, toluene diisocyanate (TDI), has been shown to cause headache, fatigue, concentration problems, irritability, depression, sleep disturbance, memory and sexual dysfunction [20].

Neuropsychological reactions to airborne neurotoxins do not constitute a single syndrome, but rather a number of syndromes, each of which is associated with a particular type of chemical. Case studies have the advantage of yielding detailed documentation of neuropsychological dysfunction which may constitute a particular pattern of neuropsychological disturbance for a specific substance or set of substances [21]. The purpose of the present case report is to evaluate the pattern of neuropsychological functions in five men suffering from physical, cognitive and emotional changes following acute and chronic MDI exposure.

Method

Subjects

Five right-handed male patients referred by their workers' compensation attorney for a comprehensive neuropsychological evaluation served as subjects for the study. Each subject had been exposed to MDI and hydrocarbon solvent vapours within the workplace. While hydrocarbon solvent vapours are known to cause acute and chronic CNS intoxication, none of the subjects became symptomatic until MDI was introduced into their work environment. The duration and severity of exposure to MDI also varied considerably among the subjects owing to differences in work-related activities and responsibilities. Instances of exposure for each subject occurred over a 2-year span, and adequate ventilation or other safety precautions were not used during that time period (for example, heating MID-based glue beyond the temperature recommended by the manufacturer). The time since MDI exposure assessment varied as well. In all but one case, several months had elapsed since MDI exposure when the subjects were first seen for the neuropsychological evaluation. Subject 5 reported that he continued to be intermittently exposed to MDI in his workplace. All other subjects were not gainfully employed at the time of assessment. Formal analysis of MDI exposure levels was unfortunately not completed during any periods in which the subjects were being exposed.

The demographic characteristics for each subject can be found in Table 1. Except in the case of Subject 3, the premorbid medical, neurological, ethanol/drug use and psychological history for each subject was unremarkable and non-contributory. Subject 3 reported a positive history of having had a minor closed head injury from an automobile accident approximately 6 years earlier. He did not, however, report any residual sequelae from the head injury. Subject 5 was also suspected of having a learning disability associated with early life academic difficulties. While he did not recall participating in special education services, none of his academic records was made available. None of the subjects had completed neuroradiographic (that is, CT, MRI), electrophysiological (that is, EEG, BEAM, BEAR, VER, SEP) or functional

Table 1.

Test
Demographics
Age
Education
Months, post-exposure
Intellectual (WAIS–R)
VIQ
PIQ
FSIQ
Information
Digit Span
Vocabulary
Arithmetic
Comprehension
Similarities
Picture Completion
Picture Arrangement
Block Design
Object Assembly
Digit Symbol
Motor speed and co-ordination
Grip strength (kg)
Finger oscillation
Grooved Pegboard (sec
Tactile sensory-perceptual (RH
Suppressions
Finger gnosis errors
Graphesthesia errors
Form recognition error
Auditory sensory-perceptual
Suppressions (RE/LE)
Speech sounds per. err
Seashore tonal mem. e
Visual sensory-perceptual
Suppressions (RF/LF)
HVOT errors
Embedded figure erro
Attention-concentration
WMS–R attent/conc. in
PASAT time/correct r
Trial one
Trial two
Trial three
Trial four
Cognitive efficiency-flexibi
Reaction time (msec
Simple
Complex
Trail Making Test
Part A
Part B

ıy foam packaging.
ur with MDI if it is
).005 parts per million
:e at risk for adverse
:ase and immunologic
ects of MDI exposure
isocyanide, toluene
ncentration problems,
l dysfunction [20].
ıot constitute a single
ı is associated with a
: of yielding detailed
constitute a particular
substance or set of
valuate the pattern of
ysical, cognitive and
re.

ımpensation attorney
ubjects for the study.
ıt vapours within the
: acute and chronic
ıDI was introduced
cposure to MDI also
vork-related activities
curred over a 2-year
not used during that
nd the temperature
ıre assessment varied
MDI exposure when
n. Subject 5 reported
workplace. All other
t. Formal analysis of
ıny periods in which

ıd in Table 1. Except
al, ethanol/drug use
d non-contributory.
ied head injury from
ıot, however, report
suspected of having
es. While he did not
:ademic records was
graphic (that is, CT,
, SEP) or functional

Table 1. Neuropsychological test results by subject

Test	Subjects 1	2	3	4	5
Demographics					
Age	52	43	29	40	29
Education	13	16	11	12	12
Months, post-exposure	8	5	9	6	Cont.
Intellectual (WAIS–R)					
VIQ	122	105	100	96	86
PIQ	117	105	104	94	93
FSIQ	122	106	101	95	87
Information	15	10	9	9	7
Digit Span	9	12	9	7	8
Vocabulary	15	10	12	9	8
Arithmetic	10	9	9	8	4
Comprehension	15	10	10	11	7
Similarities	14	15	13	12	13
Picture Completion	11	11	12	12	8
Picture Arrangement	12	10	11	8	8
Block Design	11	13	11	10	13
Object Assembly	12	10	13	10	10
Digit Symbol	8	11	6	6	6
Motor speed and co-ordination (DH/NDH)					
Grip strength (kg)	34/34	38/36	29/27	45/44	42/43
Finger oscillation	62/57	55/53	51/49	50/49	54/37
Grooved Pegboard (sec)	77/94	66/71	67/68	47/53	59/56
Tactile sensory-perceptual (RH/LH)					
Suppressions	0/0	0/0	0/0	0/0	0/0
Finger gnosis errors	0/0	0/0	0/0	0/0	0/0
Graphesthesia errors	1/1	0/0	0/0	9/10	0/7
Form recognition errors	0/0	0/0	0/0	0/0	0/0
Auditory sensory-perceptual					
Suppressions (RE/LE)	0/0	0/0	0/0	0/0	0/0
Speech sounds per. err.	2	5	17	5	8
Seashore tonal mem. err.	4	3	15	14	7
Visual sensory-perceptual					
Suppressions (RF/LF)	0/0	0/0	0/0	0/0	0/0
HVOT errors	1	6	3	4	3
Embedded figure errors	0	3	2	1	2
Attention-concentration					
WMS–R attent/conc. index	97	110	106	81	78
PASAT time/correct resp.					
Trial one	5·14	4·36	2·94	8·47	20·50
Trial two	4·62	3·53	3·16	5·45	d/c
Trial three	4·17	5·33	3·43	5·64	d/c
Trial four	24·00	6·00	3·60	4·50	d/c
Cognitive efficiency-flexibility					
Reaction time (msec)					
Simple	259	293	257	290	287
Complex	538	510	563	496	500
Trail Making Test (sec)					
Part A	52	21	26	35	28
Part B	71	68	71	104	97

Stroop Color Word (*t* score)					
Words	37	41	44	31	20
Colors	31	49	49	38	<20
Color Words	26	36	47	39	20
Language					
COWA (raw)	33	31	41	33	34
Boston Naming (raw)	60	NA	NA	57	53
Benton Sentence Rep (raw)	10	10	13	13	13
Visuographic					
Rey CFD	36	36	36	35	36
Memory-learning					
WMS–R					
General index	120	76	96	90	92
Verbal index	110	77	103	95	84
Visual index	132	80	86	84	116
Delay index	125	92	97	79	97
CVLT (raw)					
Total list A	50	35	43	39	41
Trial 1	6	6	6	6	6
Trial 5	14	9	11	9	10
Learning slope	2·1	0·9	1·1	0·8	0·7
% Recall consistency	92	85	86	87	77
Semantic clustering	3·0	1·2	0·7	1·3	1·4
Serial clustering	1	9	5·9	4·1	3·9
List B	3	4	6	4	5
Short delay free rec.	11	6	7	4	8
Short delay cued rec.	11	8	12	6	12
Long delay free rec.	13	8	9	7	12
Long delay cued rec.	12	9	10	7	11
Perseverations	3	4	1	3	0
Intrusions	2	4	0	17	1
Correct recognition	15	16	15	11	15
Discriminability (%)	91	91	93	80	95
False positives	3	4	2	4	1
Response bias	0·5	0·6	0·3	−0·11	0
Abstraction					
Category test (errors)	23	103	73	60	71
Shipley abstr. (*t*-score)	66	56	56	36	48
Emotionality					
MMPI (*t*-score)					
L	53	66	53	53	40
F	60	50	64	66	56
K	44	57	61	44	44
Sum O/S difference	40	−59	36	16	138
TR (raw)	2	1	0	0	1
Carelessness (raw)	3	2	1	1	2
Hs	67	62	103	62	65
D	75	75	84	87	77
Hy	65	75	91	62	60
Pd	48	62	62	48	74
Mf	63	61	49	53	73
Pa	44	47	62	65	67
Pt	50	71	75	48	93
Sc	57	71	90	63	94
Ma	53	63	60	53	68
Si	64	46	46	73	75

metabolic/perfusion studies t
could have otherwise assistec
associated with MDI exposu

Procedure

A comprehensive neuropsyc
the primary author (T.J.R.).
instructions provided by eacl
session. The order of test adn
for inclusion in the battery
array of neuropsychologica
ordination, sensory-percep
concentration, cognitive eff
learning, abstract reasonin
Wechsler Adult Intelligence
Reitan Neuropsychologica
Memory Test [25], Hoop
Figures Test [27], Paced A
Complex Reaction Time [2
Association Test (COW
Test [31], Rey Complex F
Revised (WMS–R) [22],
Institute of Living Scale
(MMPI) [37].

Results

At initial contact, all su
consisting of flu-like sym
forgetfulness, disoriente
reduced concentration, n
fatigue, decreased libido
the only patient diagno
Otherwise neurological
of cerebellar, motor or
diagnosed with isocyan
physically supports the
small sample size and he
A total of 12 functiona
on each subject. The in

Intellectual. WAIS–
range of ability, with t
Subject 5 was, howev
difficulties, which cou
level of general intelli
educational and occ
Performance IQ discr
cortical dysfunction.
variability between s
(Digit Span and Ar

31	20
38	<20
39	20
33	34
57	53
13	13
35	36
90	92
95	84
84	116
79	97
39	41
6	6
9	10
0·8	0·7
87	77
1·3	1·4
4·1	3·9
4	5
4	8
6	12
7	12
7	11
3	0
17	1
11	15
80	95
4	1
−0·11	0
60	71
36	48
53	40
66	56
44	44
16	138
0	1
1	2
62	65
87	77
62	60
48	74
53	73
65	67
48	93
63	94
53	68
3	75

metabolic/perfusion studies (that is, PET, SPECT) at the time of the assessment that could have otherwise assisted in confirming structural or metabolic CNS anomalies associated with MDI exposure.

Procedure

A comprehensive neuropsychological evaluation was completed for each subject by the primary author (T.J.R.). All tests were administered according to standardized instructions provided by each publisher. Subjects completed the test battery in a single session. The order of test administration was the same for all subjects. The tests selected for inclusion in the battery of tests were chosen so as to adequately assess a broad array of neuropsychological functions involving intellectual, motor speed and co-ordination, sensory-perceptual (tactile, auditory and visual modalities), attention-concentration, cognitive efficiency-flexibility, language, visuographic, memory and learning, abstract reasoning and emotionality. The administered tests included the Wechsler Adult Intelligence Scale—Revised (WAIS—R) [22], portions of the Halstead Reitan Neuropsychological Test Battery [23], Grooved Pegboard Test [24], Tonal Memory Test [25], Hooper Visual Organization Test (HVOT) [26], Embedded Figures Test [27], Paced Auditory Serial Addition Test (PASAT) [28], Simple and Complex Reaction Time [29], Stroop Colour Word Test [30], Controlled Oral Word Association Test (COWA) [31], Boston Naming Test [32], Sentence Repetition Test [31], Rey Complex Figure Drawing (RCFD) [33], Wechsler Memory Scale—Revised (WMS—R) [22], California Verbal Learning Test (CVLT) [35], Shipley Institute of Living Scale [36] and Minnesota Multiphasic Personality Inventory (MMPI) [37].

Results

At initial contact, all subjects reported a similar pattern of subjective symptoms consisting of flu-like symptoms, headaches, respiratory distress, depression, irritability, forgetfulness, disorientation, decreased calculating ability, word-finding problems, reduced concentration, numbness of the hands and feet, altered sense of smell, chronic fatigue, decreased libido, decreased exercise tolerance and skin rash. Subject 4 was the only patient diagnosed as having a mild sensory peripheral polyneuropathy. Otherwise neurological examinations on each of the subjects did not reveal a pattern of cerebellar, motor or sensory disturbances. All of the subjects, however, were diagnosed with isocyanate-induced occupational asthma and allergic rhinitis, which physically supports the fact that they did suffer from MDI exposure. Owing to the small sample size and heterogeneous nature of the sample, no group data are presented. A total of 12 functional categories were evaluated for a total of 84 reportable scores on each subject. The individual test results for each subject are presented in Table 1.

Intellectual. WAIS—R Full Scale IQ (FSIQ) scores fell within or above the average range of ability, with the exception of Subject 5, who earned a Low Average FSIQ. Subject 5 was, however, the only patient suspected of having premorbid learning difficulties, which could account for his lower-than-average measured intellect. The level of general intelligence for the group appeared to be consistent with premorbid educational and occupational histories. Also, no consistent pattern of Verbal-Performance IQ discrepancy emerged across the subjects in support of a lateralized cortical dysfunction. While the age-corrected subtest profiles revealed considerable variability between subjects, in most instances weaknesses were found on subtests (Digit Span and Arithmetic) comprising the Freedom from Distractability (FD)

factor [38]. Four of five subjects also revealed an additional relative weakness on the Digit Symbol subtest, a measure which inconsistently loads the FD factor among adults. Taken together, however, these findings imply subtle difficulties with concentration and encoding of information used in cognitive processing among the subjects.

Motor speed and co-ordination. On tests of motor speed and co-ordination, mild variability was noted with respect to left–right discrepancies among the subjects, the most notable being Subject 5's poor rate of finger oscillation in the left hand. There was, however, no consistent pattern of lateralized deficits in motor functions observed and the results typically fell within the normal range.

Tactile sensory-perceptual. Except in a few instances, the subjects typically manifested intact tactile sensory-perceptual functions. Consistent with his diagnosed peripheral polyneuropathy, Subject 4 revealed bilateral graphaesthesia errors but otherwise intact cortical tactile functions. The left-sided graphaesthesia errors seen in Subject 5 also correlated with his poor finger oscillation rate in that extremity. Tactile suppressions, finger agnosia or astereognosis errors were otherwise not observed in the subjects.

Auditory sensory perceptual. The subjects did not reveal auditory suppression errors with bilateral stimulation. With the exception of Subjects 3 and 4, the group appeared to have intact speech sound and tonal discrimination abilities. Subject 3 performed poorly on both the speech sound and tontal discrimination, while Subject 4 only revealed difficulty with the latter task. In view of the fact that auditory perceptual tasks such as these are easily disrupted by attention problems [33], it is possible that their difficulty can be explained on the basis of poor concentration as opposed to centrally mediated auditory processing deficits. Furthermore, each of these subjects reported a premorbid history of auditory acuity weakness of peripheral origin. This problem was particularly evident in Subject 3.

Visual sensory-perceptual. Visual sensory-perceptual functions appeared to be intact for each subject. No cortical suppressions were observed in either visual hemifield for subjects and each appeared to have adequate perceptual organization skills and figure–ground perception.

Attention-concentration. While the major of subjects revealed an intact simple attention span, significant weaknesses were observed in concentration for the group as a whole. Except in the case of Subject 3, subjects' performances across the four trials of the PASAT were above the established cut-off for impairment of 3·45 seconds per correct response [28]. The PASAT results almost uniformly revealed marked slowing in rate of information processing for the subjects despite the presence of at least average measured intelligence.

Cognitive efficiency—flexibility. Simple and four-choice reaction times appeared to be adequate in the group as a whole. Some variability was evident between the subjects with the simple mental and double mental tracking tasks but the results largely fell within normal limits for the group. Although no specific pattern of impaired mental flexibility was evident on the Stroop, reduced rate of mental processing contributed to a generalized slowing on this task in the majority of the subjects.

Language. Despite the subjects' commonly reported complaint of word retrieval problems, no language deficits were observed in the group. The subjects revealed intact verbal fluency, naming ability and sentence repetition.

Visuographic. As a group, the subjects performed well on the measure of visuographic reproduction. Their reproductions were free of significant perceptual distortions, misalignments, omissions or other visuoconstructive deficits.

Memory-learning. The majo
store and immediately recall r
Index below average. No co
vs. visual) deficit at immedi
the subjects revealed signific
to immediately recall verbal v
large (11) discrepancy. The
term retention of learning n
of forgetting with a Delayed

Despite their average cap
following a single exposure
in learning ability on a list-l
16 words demonstrated gene
slope and frequent reliance
consistency across learning
poor performance for imm
proactive interference, whi
Short-Delay Free Recall a
retroactive interference. Th
Delay Cued Recall, suggest
poor free recall. An underly
by the groups normal rec

Abstraction. Simple verb
performed adequately by
abstract reasoning requi
monitoring and use of fe
poorly by the majority of
errors on the Category t

Emotionality. Except i
consistently and accurate
a tendency to endorse ob
more subtle indicators [3
subject revealed at least o
had two or more such sca
for the group, clinically
Depression, three on Ps
of these scales implies t
subjects in the form of
has become increasingly

Discussion

Although the present
psychological deficits as
of compromised cogni
patients reported a high
seen in neurotoxic exp
decreased concentratio
logical findings of int

al relative weakness on the
oads the FD factor among
y subtle difficulties with
tive processing among the

l and co-ordination, mild
.es among the subjects, the
ion in the left hand. There
: motor functions observed

ubjects typically manifested
h his diagnosed peripheral
errors but otherwise intact
ors seen in Subject 5 also
nity. Tactile suppressions,
observed in the subjects.
ditory suppression errors
.nd 4, the group appeared
:es. Subject 3 performed
on, while Subject 4 only
t that auditory perceptual
ms [33], it is possible that
ncentration as opposed to
ore, each of these subjects
of peripheral origin. This

ions appeared to be intact
in either visual hemifield
al organization skills and

l an intact simple attention
for the group as a whole.
ross the four trials of the
f 3·45 seconds per correct
d marked slowing in rate
sence of at least average

action times appeared to
ident between the subjects
ut the results largely fell
ttern of impaired mental
al processing contributed
ne subjects.
nplaint of word retrieval
p. The subjects revealed
on.
vell on the measure of
of significant perceptual
uctive deficits.

Memory-learning. The majority of subjects demonstrated at least average abil store and immediately recall new material. Only Subject 2 earned a General Me Index below average. No consistent pattern of a modality-specific memory (v vs. visual) deficit at immediate recall emerged for the subjects. However, thr the subjects revealed significant discrepancies (greater than 15) between their al to immediately recall verbal vs. non-verbal material, and one demonstrated a sim large (11) discrepancy. The majority of subjects also revealed at least average l term retention of learning material. Only Subject 4 revealed a relatively rapid of forgetting with a Delayed Recall Index falling within the Borderline ability ra

Despite their average capacity to store and recall material over time (WMS following a single exposure, the majority of subjects revealed marked deficie in learning ability on a list-learning task (CVLT). Repetitive exposure with a li 16 words demonstrated generally poor learning ability with a low incremental lear slope and frequent reliance on an ineffective learning strategy, serial clustering. R consistency across learning trials otherwise appeared to be adequate. The gener poor performance for immediate recall with List B implicated a tendency tow proactive interference, while the large discrepancy (three or more words) betw Short-Delay Free Recall and Trial 5 of List A implied forgetting in the form retroactive interference. The subjects also revealed improved performances at Sh Delay Cued Recall, suggesting that problems with retrieval were contributing to poor free recall. An underlying weakness in retrieval processes was further suppor by the groups normal recall at Recognition testing and adequate Discriminabil

Abstraction. Simple verbal abstract reasoning, as measured by the Shipley test, v performed adequately by the majority of the subjects. More complex non-ver abstract reasoning requiring hypothesis formulation, hypothesis testing, s monitoring and use of feedback, and rule generalization was, however, perform poorly by the majority of the subjects. Four of the five patients scored 51 or m errors on the Category test, a value established as the cut-off for impairment [2

Emotionality. Except in the case of Subject 5, all subjects appeared to respo consistently and accurately to the objective personality inventory. Subject 5 reveal a tendency to endorse obvious indicators of psychopathology when contrasted agai more subtle indicators [39], which no doubt contributed to his profile elevation. Eve subject revealed at least one clinical scale from the MMPI above 70T, and the majori had two or more such scales elevated. While there was no common codetype observ for the group, clinically significant elevations were evident for all five subjects Depression, three on Psychaesthenia and three on Schizophrenia. The combinati of these scales implies the presence of significant emotional distress among the subjects in the form of depression, anxiety and altered mentation, a finding whic has become increasingly evident among neurotoxic-exposed patients [11].

Discussion

Although the present results do not clearly identify a single pattern of neuro psychological deficits associated with MDI exposure, the data do suggest the presenc of compromised cognitive functions characteristic of CNS involvement. All of th patients reported a high incidence of vague subjective complaints of the type typicall seen in neurotoxic exposure, such as headaches, mood alterations, forgetfulness an decreased concentration [5, 7, 8, 11]. Similarly, the common group neuropsycho- logical findings of intact psychomotor, psychosensory, visuographic and language

skills accompanied by decreased concentration, mental efficiency, rate of information processing, learning ability and abstract reasoning are largely consistent with neuropsychological deficits typically reported in neurotoxic studies [2, 5, 6, 40].

Additionally, the high incidence of emotional distress revealed by the subjects on the objective personality measure appears consistent with previous studies that have found affective and mood disturbances following toxic exposure [11]. Whether the emotional distress identified in these subjects arises from reactive issues, an organic affective disturbance, or some combination of the two is unclear. It is interesting to note, however, that decrements in regional cerebral blood flow have been identified in structures known to mediate emotions (for example, prefrontal and frontotemporal structures) among patients exposed to organic solvents [41]. It is unfortunate that specific neurodiagnostic information pertaining to the structural and functional integrity of these regions was not available for the present subjects. There is also a possibility that the associated cognitive deficits noted above are in part the direct result of emotional distress in these patients. Within a clinical setting, however, there appears to be no certain established way to rule this out, except to treat the affective distress and follow the patient over time to see if there is a correlated improvement in cognition.

The mechanism by which MDI acts on the nervous system to produce neuropsychological deficits is not known at the present time. Moreover, since it is possible that toxins affect the central nervous system through a number of mechanisms, it would be entirely speculative to comment on MDI's mode of action [42]. The possibilities, however, include interfering with energy production required to maintain normal neural structure and function through inactivation of enzymes or coenzymes essential for oxidative energy mechanisms, interfering with nutrition of the neural cells through involvement of the nutrient vessels, giving rise to allergic or immunologic responses that ultimately lead to structural or functional neural impairment, and altering neural function through effects on the neurochemistry, including neurotransmitters, acid–base balance and ionic concentrations. Equally speculative would be to posit a primary focus of central nervous system involvement. While it might be suggested the neuropsychological test results reflect a frontal diencephalic pattern of weakness, such a pattern is similarly evident in other non-specific disorders, such as closed head injury and chronic alcohol abuse [6] and may be of little value in localization.

Limitations imposed by the small sample make generalizing the present results to other clinical cases difficult. There is quite obviously a need for replication of the present findings. Since all of the subjects were involved in personal injury litigation, it can also be argued that the results reflect more the needs of the patients to be functionally impaired as opposed to their true neuropsychological status. However, there appears to be little support for simple malingering or exaggeration by the subjects given the data. In addition to weaknesses on testing, each of the subjects revealed areas of relative strengths. Moreover, each patient was re-evaluated approximately 1 year following their initial assessment and while some areas of continued weaknesses were identified, there were also clear indicators of functional improvement found in the group as a whole. In fact, one subject was found to be symptom-free at the 1 year follow-up. This pattern of improvement among the subjects arose in the presence of unsettled personal litigation. The results of this follow-up study will be published in the near future.

One additional confound with the study represents selection bias. Unfortunately, there were additional workers who suffered MDI exposure but did not come forward

with complaints. Several of
speculated they were reluctan
plausible that the test subje
therefore more likely to repo
from foreign cultures who ha
with subjective reporting as

References

1. HARTMAN, D. E.: *Neuro*
Neurotoxic Syndromes. (Per
2. BOWLER, R., MERGLER, D
former microelectronics w
3. DICK, R. B.: Short durat
neurobehavioral test result
1988.
4. JOHNSON, B. L.: *Prevention*
New York), 1987.
5. BAKER, E. and FINE, L.: S
Medicine, **28:** 126–129, 1
6. LEZAK, M.: Neuropsyc
techniques and interpreta
10: 25–29, 1984.
7. ESKENAZI, B. and MAL
neurobehavioral function
Neuropsychology: The Impa
1988.
8. WEISS, B.: Behavioral t
challenge for psycholog
9. BOLLA-WILSON, K., WIL
after neurotoxic exposur
10. SCHOTTENFELD, R. and C
American Journal of Psych
11. BOWLER, R., RAUCH, S.
neurotoxic exposure. *A*
12. WENNGREN, B. and
occupational hazards. *A*
13. HODGSON, M., FURMAN
short-term hydrocarbo
14. BRAUN, C., DAIGNEAU
printshop solvent neur
Clinical Neuropsychology.
15. MERGLER, D. and BLAI
Journal of Industrial Med
16. RYAN, C., MORROW, L
associated with occupa
of Psychiatry, **145:** 1442
17. DAVIS, K., YESAVAGE,
induced parkinsonism.
18. BANKS, D., BUTCHER,
of Allergy, **57:** 389–39
19. KORBEE, L. and BERN
of chemicals. *Occupati*
20. SINGER, R. and SCOTT
diisocyanate exposure
21. TROSTER, A. and RU
hydrocarbon solvent t

with complaints. Several of the test subjects commented on these workers and speculated they were reluctant to come forward for fear of losing their jobs. It is also plausible that the test subjects were actually more affected by the exposure and therefore more likely to report symptoms. There were also a number of co-workers from foreign cultures who had obvious language and cultural differences that interfered with subjective reporting as well as objective neuropsychological testing.

References

1. HARTMAN, D. E.: *Neuropsychological Toxicology: Identification and Assessment of Human Neurotoxic Syndromes*. (Pergamon Press, New York), 1988.
2. BOWLER, R., MERGLER, D., HARRISON, R. *et al.*: Neuropsychological impairment among former microelectronics workers. *NeuroToxicology*, **12**: 87–103, 1991.
3. DICK, R. B.: Short duration exposures to organic solvents: The relationship between neurobehavioral test results and other indicators. *Neurotoxicology and Teratology*, **10**: 39–50, 1988.
4. JOHNSON, B. L.: *Prevention of Neurotoxic Illness in Working Populations*. (John Wiley & Sons, New York), 1987.
5. BAKER, E. and FINE, L.: Solvent neurotoxicity: The current evidence. *Journal of Occupational Medicine*, **28**: 126–129, 1986.
6. LEZAK, M.: Neuropsychological assessment in behavioral toxicology-developing techniques and interpretative issues. *Scandinavian Journal of Work Environment and Health*, **10**: 25–29, 1984.
7. ESKENAZI, B. and MAIZLISH, N.: Effects of occupational exposure to chemicals on neurobehavioral functioning. In: R. Tarter, D. Van Thiel and K. Edwards (editors). *Medical Neuropsychology: The Impact of Disease on Behavior*. (Plenum Press, New York), pp. 223–264, 1988.
8. WEISS, B.: Behavioral toxicology and environmental health science: opportunity and challenge for psychology. *American Psychologist*, **38**: 1174–1187, 1983.
9. BOLLA-WILSON, K., WILSON, R. and BLEECKER, M.: Conditioning of physical symptoms after neurotoxic exposure. *Journal of Occupational Medicine*, **30**: 684–686, 1988.
10. SCHOTTENFELD, R. and CULLEN, M.: Occupation-induced post-traumatic stress disorders. *American Journal of Psychiatry*, **142**: 198–202, 1985.
11. BOWLER, R., RAUCH, S., BECKER, C. *et al.*: Three patterns of MMPI profiles following neurotoxic exposure. *American Journal of Forensic Psychology*, **7**: 15–31, 1989.
12. WENNGREN, B. and ODKVIST, L.: Vestibulo-oculomotor disturbances caused by occupational hazards. *Acta Otolaryngologica Supplement*, **455**: 7–10, 1988.
13. HODGSON, M., FURMAN, J. and RYAN, C.: Encephalopathy and vestibulopathy following short-term hydrocarbon exposure. *Journal of Occupational Medicine*, **31**: 51–54, 1989.
14. BRAUN, C., DAIGNEAULT, S. and GILBERT, B.: Color discrimination testing reveals early printshop solvent neurotoxicity better than a neuropsychological test battery. *Archives of Clinical Neuropsychology*, **4**: 1–13, 1989.
15. MERGLER, D. and BLAIN, L.: Assessing color vision loss among exposed workers. *American Journal of Industrial Medicine*, **12**: 195–203, 1987.
16. RYAN, C., MORROW, L. and HODGSON, M.: Cacosmia and neurobehavioral dysfunction associated with occupational exposure to mixtures of organic solvents. *American Journal of Psychiatry*, **145**: 1442–1445, 1988.
17. DAVIS, K., YESAVAGE, J. and BERGER, P.: Single case study: Possible organophosphate-induced parkinsonism. *The Journal of Nervous and Mental Disease*, **166**: 222–225, 1978.
18. BANKS, D., BUTCHER, B. and SALVAGIO, J.: Isocyanate-induced respiratory disease. *Annals of Allergy*, **57**: 389–396, 1986.
19. KORBEE, L. and BERNSTEIN, L.: Workers risk occupational asthma by exposure to myriad of chemicals. *Occupational Health and Safety*, **57**: 28–31, 1988.
20. SINGER, R. and SCOTT, N.: Progression of neuropsychological deficits following toluene diisocyanate exposure. *Archives of Clinical Neuropsychology*, **2**: 135–144, 1987.
21. TROSTER, A. and RUFF, R.: Neuropsychological sequelae of exposure to the chlorinated hydrocarbon solvent trichloroethylene. *Archives of Clinical Neuropsychology*, **5**: 31–47, 1990.

22. WECHSLER, D.A.: Wechsler Adult Intelligence Scale—Revised: Manual. (Psychological Corporation, New York), 1981.
23. REITAN, R. and WOLFSON, D.: *The Halstead-Reitan Neuropsychological Test Battery*. (Neuropsychology Press, Tucson, AZ), 1985.
24. KLOVE, H.: Clinical Neuropsychology. In: J. F. M. Forster (editor). *The Medical Clinics of North America*. (Saunders, New York), **47**, pp. 1647–1658, 1963.
25. SEASHORE, C. E., LEWIS, D. and SALTVEIT, D. L.: *Seashore Measures of Musical Talents* (revised edition). (Psychological Corporation, New York), 1960.
26. HOOPER, H. E.: *The Hooper Visual Organization Test*. (Western Psychological Services, Los Angeles), 1958.
27. TALLAND, G. A.: *Deranged Memory*. (Academic Press, New York), 1965.
28. GRONWALL, D. M.: Paced auditory serial addition task: a measure of recovery from concussion. *Perceptual and Motor Skills*, **44:** 367–373, 1977.
29. PIROZZOLO, F., CHRISTENSEN, K., OGLE, K. *et al.*: Simple and choice reaction time in dementia: Clinical implications. *Neurobiology of Aging*, **2:** 113–117, 1981.
30. GOLDEN, C. J.: A group version of the Stroop Color and Word Test. *Journal of Personality Assessment*, **39:** 386–391, 1975.
31. BENTON, A. L. and HAMSHER, K. de S.: *Multilingual Aphasia Examination* (manual, revised).
32. KAPLAN, E. F., GOODGLASS, H. and WEINTRAUB, S.: *The Boston Naming Test*. (E. Kaplan & H. Goodglass, Boston), 1978.
33. LEZAK, M.: *Neuropsychological Assessment*. (Oxford University Press, New York), 1983.
34. WECHSLER, D. A.: *Wechsler Memory Scale-Revised: Manual*. (Psychological Corporation, New York), 1987.
35. DELIS, D. C., KRAMER, J. H., KAPLAN, E. *et al.*: *California Verbal Learning Test*, Research Edition Manual. (Psychological Corporation, New York , 1987.
36. SHIPLEY, W. C.: *Manual: Shipley-Institute of Living Scale*. (Western Psychological Services, Los Angeles), 1967.
37. HATHAWAY, S. R. and MCKINLEY, J. C.: *Minnesota Multiphasic Personality Inventory*. (Psychological Corporation, New York), 1967.
38. KAUFMAN, A. S.: *Assessing Adolescent and Adult Intelligence*. (Allyn & Bacon, Boston), 1990.
39. GREEN, R. L.: *The MMPI: An Introspective Manual*. (Grune & Stratton, New York), 1980.
40. MORROW, L., RYAN, C., STEIN, D. *et al.*: *Neuropsychological deficits in workers exposed to mixtures of organic solvents*. Paper presented at the American Psychological Association Annual Meeting, Los Angeles, CA, August 1986.
41. HAGSTADIUS, S., ORBAEK, P., RISBERG, J. *et al.*: Regional cerebral blood flow at the time of diagnosis of chronic toxic encephalopathy induced by organic-solvent exposure and after the cessation of exposure. *Scandinavian Journal of Work Environment and Health*, **15:** 130–135, 1989.
42. GOETZ, C. G.: *Neurotoxins in Clinical Practice*. (Spectrum, New York), 1985.