CABLE: "UAW DETROIT"

Solidarity House
8000 EAST JEFFERSON AVE.
DETROIT, MICHIGAN 48214
PHONE (313) 926-5000

INTERNATIONAL UNION, UNITED AUTOMOBILE, AEROSPACE & AGRICULTURAL IMPLEMENT WORKERS OF AMERICA—UAW

STEPHEN P. YOKICH, *PRESIDENT*
ROY O. WYSE, *SECRETARY-TREASURER*

VICE-PRESIDENTS: CAROLYN FORREST • JACK LASKOWSKI • ERNEST LOFTON • RICHARD SHOEMAKER

April 4, 1996

Ms. Diane Manning
Education and Information Division
National Institute of Occupational
Safety and Health (NIOSH)
4676 Columbia Parkway - Mail Stop C-34
Cincinnati, Ohio 45226-1998

Dear Ms. Manning:

These are preliminary comments on the draft Criteria Document for metalworking fluids (dated February, 1996). Additional comments from the UAW will follow. It is obvious that a great deal of effort and thought has gone into this work at a time of severe constraints on NIOSH resources and other distractions. My general impression of the draft is that it is comprehensive but overly cautious. The regulatory proposal needs more detail and innovation. Enclosed is a marked-up copy of the draft, identifying issues and details that, in my view, need further consideration.

The major issues that we perceive are as follows.

1. **Real-World Focus** The document needs an additional real-world focus. For example, the use of reclaimed or reprocessed oils is not mentioned (p.4,7,130). Similarly tramp oils (although ubiquitous, a major cause of MWF rejection, and potentially a significant ingredient of health concern) are not discussed (hydraulic oils, slide oils etc.). Base oils are discussed in the sanitized language of API ("highly refined," p.4,7). In reality, some oils used in soluble oil systems are procured separately from the coolant concentrate package, and can come from any source, including tramp oils scavenged from in-plant sources such as parts washers or degreasers. The draft document implies that "heavily hydrotreated" oils are free of significant risk (p.131,151). The questions of which PAHs are important, what levels are acceptable, and analytical methods need to be discussed (p.130). There is no acknowledgment of chemical deterioration of oils with use. Chlorinated oils and waxes were discussed as though C-12 and C-23 constitute the universe whereas in reality, crude, poorly characterized and unstudied mixtures are produced by chlorination from both paraffinic and naphthenic hydrocarbon stocks (p.134,135). The sulfur chemistry discussed needs to be more detailed. Many ingredients in some modern coolants have never been evaluated for health risks in a metalworking context: for example, organophosphates, glycol ethers. Maintenance issues for ventilation (p.203) and mist control systems (p.166) need to be included as is a critical examination of mist collectors on principle: do they generate mists? what about vapors? House-keeping is not examined in terms, for example, of coolant on the floor dripping - or being mopped - into flumes (p.209).

RECEIVED

APR 16 1996

NIOSH DOCKET OFFICE

opeiu494
PRINTED IN USA

2. **Historical Decline of MWF Mist Levels** The historical rate of decline of MWF mist levels is taken uncritically from the Harvard study and the rather limited OSHA database (p.17,46,167). The plant in the Kriebel study (p.17) is cited even though it has atypically low oil mist levels, probably because of the type of production and absence of central coolant systems. The discussion concerning historical trends acknowledges that past exposure measurements have been complaint-driven (p.46) in contrast to the systematic campaign sampling for the recent research studies, but needs to qualify or adjust the conclusion on steepness of downward trends accordingly. Technological issues not addressed include the much higher operating temperatures of modern cutting tools (carbide, ceramic) compared with the pre-1950s, with unknown consequences for oil deterioration and other chemical changes. Higher cutting speeds presumably also impact aerosol size distributions.

3. **Microbial Ecology Problems** The microbial ecology problems are regarded as almost trivial, due to sloppy biocide management (p.149), rather than identified as a potentially major problem-technology that is evolving out of control. Coolant system and machine design is needed that minimizes coolant dispersal and recapture from the general plant environment. Trial-and-error biocide approaches to system control place workers at risk for unpredictable exposure effects, both chemical and microbiological. Hypersensitivity pneumonitis is no longer a rare event (p.173-174).

4. **Cancer Risk Assessment** For cancer risk assessment, no quantitative estimates were attempted for the expected deaths (cancers of larynx, esophagus, rectum) associated with 0.5 mg/m^3 exposures, making various assumptions about ingredients (p.170). Given the mortality study findings (very likely underestimates due to exposure misclassification), even a 20-fold reduction in etiologic exposures would result in significant work-related mortality. Liver disease was totally ignored.

5. **Feasibility** Feasibility was examined in a narrow context, relying largely on what has already been achieved. No regulatory distinctions were made for new installations (e.g., REL=0.1 mg/m^3) vs. old systems (REL=0.3 mg/m^3). The proposed RELs should be "starting", not "final," positions. No-effect levels are clearly far below 0.5 mg/m^3 for both malignant and nonmalignant health effects.

Sincerely,

Robert M Park

Robert M. Park
Project Epidemiologist
Health and Safety Department
International Union, UAW

RMP/mkh
opeiu494afl-cio
cc: Frank Mirer
Jim Howe
Barrie Brooks
Andrea Taylor
Lori Abrams

F:\DEPT\H&S\HOME\BPARK\DOCS\MWFCRTD2.DOC

Following a review by NIOSH of comments on this draft document, we will prepare a final draft version of the criteria document. We request that written comments be submitted by May 31, 1996, to Diane Manning (513-533-8450), Education and Information Division, NIOSH; 4676 Columbia Parkway, Mail Stop C-34; Cincinnati, OH 45226-1998

Linda Rosenstock, M.D., M.P.H.
Director
February 23, 1996

CONTENTS

Page

Foreword v
Abbreviations ix
1. **Introduction** 1
2. **Background** 2
    - 2.1 Production and Use
    - 2.2 MWF Application
    - 2.3 Worker Exposure
    - 2.4 Formulations
        - 2.4.1 Straight or Insoluble MWFs
        - 2.4.2 Soluble MWFs Soluble Oil MWFs
        - 2.4.3 Synthetic MWFs
        - 2.4.4 Semisynthetic MWFs
        - 2.4.5 MWF Ingredients and Additives
    - 2.5 Metalworking Fluid Environment
3. **Occupational Exposures to MWFs** 12
    - 3.1 National Occupational Exposure Survey (NOES, 1981-1983)
    - 3.2 Occupational Exposures to Mineral Oil Mists (IMIS, 1979-1995)
    - 3.3 NIOSH Health Hazard Evaluations
    - 3.4 Reported Exposures in the Automotive Industry
4. **Occupational Health Risks for Workers Exposed to MWFs** 18
    - 4.1 Cancer Risks for Workers Exposed to MWFs
        - 4.1.1 Criteria for Inclusion
        - 4.1.2 Studies of Cancer in Broad Occupational Groups
        - 4.1.3 Investigations of Selected Cancers
            - 4.1.3.1 Skin cancer
            - 4.1.3.2 Stomach cancer
            - 4.1.3.3 Pancreatic cancer
            - 4.1.3.4 Laryngeal cancer
            - 4.1.3.5 Rectal cancer
            - 4.1.3.6 Bladder cancer
            - 4.1.3.7 Esophageal cancer
            - 4.1.3.8 Brain cancer and other nervous system cancers
            - 4.1.3.9 Prostatic cancer
            - 4.1.3.10 Lung cancer
            - 4.1.3.11 Colon cancer
            - 4.1.3.12 Hematopoietic cancer
        - 4.1.4 Genetic Effects

CONTENTS (continued)

Page

- 4.1.5 Information on Exposure Concentrations
- 4.1.6 Route of Exposure
- 4.1.7 Conclusion

4.2. Nonmalignant Respiratory Effects 67
- 4.2.1 Diseases of the Lung Parenchyma
  - 4.2.1.1 Lipid pneumonia
  - 4.2.1.2 Hard metal disease
  - 4.2.1.3 Legionellosis
  - 4.2.1.4 Hypersensitivity pneumonitis
- 4.2.2 Discussion
- 4.2.3 Asthma and Other Disorders of the Pulmonary Airways
  - 4.2.3.1 General background
  - 4.2.3.2 Asthma-Case reports and observations from surveillance programs
  - 4.2.3.3 Asthma-Research findings
  - 4.2.3.4 Asthma-Summary
  - 4.2.3.5 Symptoms of airways disorders-Summary
- 4.2.4 Cross-Sectional Studies of Lung Function
  - 4.2.4.1 Pulmonary function
  - 4.2.4.2 Cross-sectional studies of lung function-Summary
  - 4.2.4.3 Cross-shift studies of acute effects on lung function
  - 4.2.4.4 Cross-shift studies of acute effects on lung function-Summary
- 4.2.5 Concluding Discussion

4.3 Dermatologic Conditions 120
- 4.3.1 Cutaneous Disorders
- 4.3.2 Irritants
- 4.3.3 Prognosis and Preventive measures ← 4.4 ← LIVER EFFECTS

5. Selected Potentially Hazardous Chemical Ingredients or Additives 126
- 5.1 Triethanolamine ALKANOLAMINES
- 5.2 Mineral Oil
- 5.3 Biocides
- 5.4 Chlorinated Paraffins + NAPHTHALENES
- 5.5 Potentially Sensory or Pulmonary Irritants

6. Potentially Hazardous Contaminants 138
- 6.1 Nitrosamines
  - 6.1.1 NIOSH Reports of N-Nitrosamine Contamination
  - 6.1.2 Carcinogenicity of N-Nitrosamines

CONTENTS (continued)

insert

Page

6.2 Microbial Contamination
   6.2.1 Ecology
   6.2.2. Potential Hazards
7. Current Occupational Recommendations and Standards 150
8. Sampling and Analytical Methods 156
   8.1 Background of Current Methods
   8.2 Gravimetric Analysis Error and Limit of Quantitation
   8.3 Potential Sampling and Analytical Method Bias and Sources of Error in Measurement
      8.3.1 Estimation of Total Method Bias
      8.3.2 Estimation of Total Method Precision
   8.4 Recommendations
9. Basis for a Recommended Exposure Limit (REL) 164
   9.1 Introduction
      9.1.1 Industry Trends
      9.1.2 Control Technology Issues
   9.2 Effects from Exposure
      9.2.1 Cancer
      9.2.2 Nonmalignant Respiratory Effects
      9.2.3 Dermatological Conditions
      9.2.4 Microbial Contamination
   9.3 Rationale for the Recommended Exposure Limit
   9.4 Technological Feasibility of Controlling Exposures to MWFs
10. Recommendations for an Occupational Safety and Health Program 193
   10.1 Safety and Health Training
   10.2 Workplace Analysis
   10.3 Environmental Sampling Strategy
   10.4 Hazard Prevention and Control
      10.4.1 Fluid Use and Application
      10.4.2 Fluid Maintenance
      10.4.3 Isolation
      10.4.4 Ventilation Systems
      10.4.5 Containment Structures
   10.5 Protective Clothing and Equipment
      10.5.1 Protective Clothing
      10.5.2 Respiratory Protection
   10.6 Sanitation and Hygiene
   10.7 Labeling and Posting

ABBREVIATIONS (continued)

MSDS	material safety data sheet
NIOSH	National Institute for Occupational Safety and Health
NO	nitrous oxide
NO_2^-	nitrite
NO_3^-	nitrate
NDBA	N-nitrosodibutylamine
NDELA	N-nitrosodiethanolamine
NDEA	N-nitrosodiethylamine
NDMA	N-nitrosodimethylamine
NMOR	N-nitrosomorpholine
NPIP	N-nitrosopiperidine
NDPA	N-nitrosodipropylamine
NPYR	N-nitrosoppyrolidine
NTP	National Toxicology Program
OHAB	GM-UAW Occupational Health Advisory Board
OR	odds ratio
OSHA	Occupational Safety and Health Administration
P	probability
PEL	OSHA permissible exposure limit
PAH	polyaromatic hydrocarbons
PMR	proportional mortality ratio
PNOC	particulates not otherwise classified
ppm	parts per million
RD_{50}	50% reduction in f
REL	NIOSH recommended exposure limit
RR	relative risk, rate ratio
RTECS	Registry of Toxic Effects of Chemical Substances
SD	Standard Deviation
SENSOR	Surveillance program for occupational asthma in Michigan, instituted in 1988 by the Michigan Department of Public Health
SNC	sinonasal cancer
spp.	species
STEL	short-term exposure limit
TEA	triethanolamine
TCFE	trichlorotrifluoroethane
TLV	Threshold Limit Value
TWA	time-weighted average
UAW	International Union, United Automobile, Aerospace & Agricultural Implement Workers of America

SMR

ABBREVIATIONS (continued)

VT	mean tidal volume
wk	week
yr	year

I. Introduction

The intent of this draft *Criteria for a Recommended Standard: Occupational Exposures to Metalworking Fluids* is first, to identify populations potentially exposed to metalworking fluids (MWFs) and assess worker exposures; second, examine adverse health effects associated with MWF exposures; and finally, in order to reduce occupational exposures to all classes of MWFs, recommend a total particulate exposure limit and an occupational safety and health program.

A variety of factors within metalworking (MW) environments must be examined in order to thoroughly evaluate worker exposures to MWFs. Many of these factors influence the stability, functionality, and the extent and safety of MWF exposures, and ultimately the safety of the MWF-associated, working environment. Airborne and contact exposures within the MW environment include those resulting from aerosolization and splashing of MWFs from machining processes and operations. Types of exposures in addition to MWFs include exposures from: metals being machined; chemical residues from preceding operations; non-MWF process and auxiliary chemicals; additives to the MWFs; substance contamination of MWFs from housekeeping and cleaning processes, biological (bacterial toxins and metabolic products) and physical contamination (e.g, chips and fines) of the MWFs. Excessive exposure conditions may exist due to inadequate enclosures and ventilation systems, and suboptimal engineering ~~contro~~ls.

design

MWFs have been grouped into four major classes: straight oil, soluble oil, synthetic, and semisynthetic. General information on MWF formulation, production, and use follows.

2. Background

2.1 Production and Use

MWFs were first used in the early 1900s to prolong the tool life of metalworking equipment [Newhouse 1982]. The Independent Lubricant Manufacturers Association (ILMA) reported that 71.5 million gallons of MWFs were produced in the United States in 1992 [ILMA 1993]. These fluids (i.e., cutting oils, machining fluids, lubricants, and coolants) reduce friction between the cutting tool and the work surface, reduce wear and galling and protect surface characteristics, reduce surface adhesion or welding, carry away generated heat, and flush away swarf, chips, fines, and residues [Nachtman and Kalpakjian 1985]. MWFs are specifically designed for use in a variety of metalworking operations such as turning, grinding, boring, drawing, tapping, gear shaping, reaming, rolling, hobbing, and band and hack sawing [Weindel 1982].

milling
broaching
drilling

2.2 MWF Application

MWF~~s~~ delivery ranges from ~~may be~~ manual~~ly~~ appli~~ed~~cation to the cutting zone of the tool and the work ~~or~~ to ~~they may be~~ deliver~~ed~~ as a mist in a high velocity air stream. ~~Alternatively~~ Typically, a continuous stream of MWF delivered by a low or high pressure pump may be directed at the cutting edge of the machine tool and over the work to carry away the metal chips or swarf. The MWFs are routinely collected and recirculated back to the cutting zone of the machine tool. The recirculating system can be self contained in a single machine or, complex, serving hundreds of machines ~~and~~ and containing large amounts of MWFs.

2.3 Worker Exposure

Workers can be exposed by skin contact to MWFs by splashes and mists during immersion or flooding of the machine tool or work and by handling equipment, tools and parts covered with MWFs. Workers may also be exposed to MWFs by inhalation or ingestion of particulates, mists, and aerosols [Bennett and Bennett 1987].

2.4 Formulations

MWFs are grouped into four major classes (Table 2-1, General Composition of the Four MWF classes):

- Straight oils (insoluble)MWFs contain no water (neat oil).
- Soluble oils MWFs are emulsions of petroleum based (mineral oil) and water.
- Synthetic MWFs contain no petroleum based oils
- Semisynthetic MWFs are also oil and water emulsions that are sometimes distinguished from soluble MWFs by the degree of emulsification.

2.4.1 Straight or Insoluble MWF

Straight oils (cutting oils) function as lubricants, improve the finish on the metal cut, and prevent rusting [Frazier 1982; CRC 1985]. Depending on the application, petroleum oils used in insoluble MWFs are usually mineral oils from highly refined naphthenic (generally saturated, ring-type structures) or paraffinic oils (straight, or branched-chain saturated hydrocarbons) [Bigda 1980, SME 1992]. They may also be reprocessed oils from a variety of possibly poorly characterized sources.

Table 2-1, General composition of the four MWF classes*

Component	Function	~~Insoluble~~ straight oils (amount)	Soluble oils (amount)	Synthetics (amount)	Semi-synthetics (amount)
Mineral oil	Lubrication Carrier	60-100%	60-85%	---	5-30%
Emulsifier	Emulsifies	---	5-20%	5-10%	5-10%
Chelating agents	Ties up ions in solution	---	0-1%	0-1%	0-1%
Coupling agents	Stabilizes	---	1-3%	1-3%	1-3%
Viscosity index improvers	Maintains viscosity	✓	---	---	---
Detergent	Prevents deposit formation	✓	✓	✓	✓
Plasticizer	Reduce tackiness	---	✓	✓	✓
Anti-mist agent	Reduces misting	✓	✓	---	---
Anti-weld agent	Prevents welding	0-20%	0-20%	0-10%	0-10%
Oiliness agent	Increase film strength	✓	---	---	---
Surfactant wetting agent	Reduce surface tension	0-10%	5-20%	10-20%	10-20%

* Adapted from Key et al.[1983], Niemeier [1990], and ILMA [1994].

--- Not present in this MWF.

✓ Usually present in this MWF.

Table 2-1, General composition of the four MWF classes* (continued)

Component	Function	Insoluble oils (amount)	Soluble oils (amount)	Synthetics (amount)	Semi-synthetics (amount)
Dispersants	Prevent fine agglomeration and deposit formation	✓	---	---	---
Passivator	Prevents staining	✓	---	---	---
Anti-foaming agents	Prevents foaming	0-500 ppm	0-500 ppm	0-500 ppm	0-500 ppm
Alkaline reserve	Buffer control	---	2-5%	2-5%	2-5%
Dyes	Identifies, leak detection	---	0-500 ppm	0-500 ppm	0-500 ppm
Odorant	Mask odor	✓	✓	✓	✓
Corrosion inhibitors, anti-rust	Prevent rust film barrier	0-10%	3-10%	10-20%	10-20%
Biocides bioresistant components	Control of bacterial and fungal contaminants	---	0-2%	0-2%	0-2%
Extreme Pressure additives	Reaction lubricant films	0-95%(?)	0-20%	0-10%	0-10%

* Adapted from Key et al. [1983], Niemeier [1990], and ILMA [1994].
--- Not present in this MWF.
✓ Usually present in this MWF.

Mineral oils may serve as a blending medium or as a additive carrier in straights or water-soluble emulsions [SME 1992]. Animal, marine, or vegetable oils may be used singly or in combination with mineral oils and in straights to increase the wetting action and lubricity [Cookson 1971, SME 1992]. Straight oil MWFs containing both fatty oil and sulfur additives provide greater lubricity, whereas those containing sulfo-chlorinated mineral oils have improved antiweld (prevention of welding of tool with workpiece and/or chips) properties over a wide temperature range. Sulfo-chlorinated mineral oil with fatty oils added are good for heavy-duty, slow-speed operations [CRC 1985].

chemically more

2.4.2 Soluble MWFs

Soluble MWFs (emulsions and water soluble oils) cool and lubricate to prevent welding of the cutting tool and the work surface, reduce abrasive wear of the tool at high temperatures, and prevent distortion caused by residual heat [Frazier 1982]. The ~~highly refined~~ mineral oils (paraffinic or naphthenic base oils) of soluble MWFs are blended from higher viscosity oil bases than are ~~insoluble~~ straight oils. Soluble oil MWF concentrates are diluted 60 to 85% with water before use [ILMA, 1995]. They contain a surface-active emulsifying agent to maintain the oil-water mix in an emulsified oil and water phase [Cookson 1971; Menter et al. 1975]. Superfatted emulsions of soluble MWFs are produced by the addition of fatty oils, fatty acids, or esters; extreme-pressure emulsions for very heavy-duty operations are produced with the addition of sulfur~~,~~ chlorine~~,~~ or phosphorous derivatives [CRC 1985; SME 1992]. They can range from highly refined petroleum stocks to reprocessed oils of unknown origin.

2.4.3 Synthetic MWFs

Synthetic MWFs also function as coolants and lubricants. They eliminate smoking, reduce misting, and provide detergent action and oxidative stability [Vahle 1982]. Synthetic MWFs contain no oil. The simplest synthetics are made with organic and inorganic salts dissolved in water. They offer rust protection and good heat removal, but usually have very low lubricating ability [SME 1992]. Others may be formulated with synthesized hydrocarbons, organic esters, polyglycols, phosphate esters, and other synthetic lubricating fluids [CRC 1985]. Synthetics are stable, can be made bioresistant [Passman 1992], and provide effective cooling capacity at high speeds and feeds.

2.4.4 Semisynthetic MWFs

Semisynthetic MWFs contain small amounts of oil (5%-30% in the concentrate) and may be formulated with fatty acids, sulfur, chlorine, and phosphorous derivatives to provide lubrication for higher speed and feed operations to medium and heavy operations [CRC 1985]. The investigators cited in the health studies presented in this draft document do not always differentiate between soluble and semisynthetic MWFs. As a result, in some parts of this draft document, semisynthetics have not been separated as a major class of MWFs.

2.4.5 MWF Ingredients and Additives

Refined petroleum oils may be used as base oils in all MWFs except the synthetics. The types and amounts of chemical constituents in these refined oils depend on the original crude and the refining processes. Refined petroleum oils are complex mixtures of hydrocarbons (aromatics, naphthenes, paraffins, and cycloparaffins), metal compounds, and organic compounds containing sulfur, oxygen, and nitrogen. There is less variability among the finished oils as the refining processes increase in severity. Solvent extraction or severe hydrotreating can reduce the total aromatic hydrocarbon content, and severe treatment with fuming sulfuric acid can almost completely remove aromatics, including polyaromatic hydrocarbons (PAHs) [IARC 1984].

2.5 Metalworking Fluid Environment

Machining or parts manufacture include a variety of process operations from parts forming to assembly of the finished product. During many of these operations, process chemicals and ancillary lubricants may contaminate the MWFs. In-process cleaners, as well as metal and general environmental contaminants such as floor detergents may also contaminate MWFs. **Table 2-2, General formulation and application of MWFs** and **Table 2-3, MWF operations including process chemicals and ancillary lubricants** provides a listing of general applications of MWFs and general types of process and ancillary chemicals.

Table 2-2, General formulation and application of MWFs*

Formulation Technology	General Application	General Categories
Straight or Neat Oil, Soluble or Emulsifiable Oil, Synthetic, Semisymthetic	Machining and Grinding	Removal Fluids
Straight or Neat Oil, Soluble or Emulsifiable Oil, Synthetic, Semisymthetic	Stamping, Drawing, Honing, Coining, Cold Heading, Wire/Bar/Rod Drawing, Piercing, Forging, Rolling, Other	Cold Forming Fluids
Straight or Neat Oil Soluble or Emulsifiable Oil	Fingerprint Displacing, Indoor or Outdoor Storage, Other	Protecting Fluids†
Straight Oil, Soluble or Emulsifiable, Synthetic	Quenching Others	Treating Fluids

* Reprinted from "Lubes and Greases," July 1995, Vol 1, Issue 4, based on information provided by the ILMA.

† Protecting fluids often are brought in on sheet steel products during stamping operations and offer shorter term protection compared to the protection provided by coatings or phosphatized surfaces.

hot forming category

Table 2-3, MWF operations including process chemicals and ancillary Lubricants*

Operation	Ancillary Lubricants	Process	Process Chemicals
Casting, forging, rolling, stamping, piercing, coining, drawing, press forming	Hydraulic fluids, greases, and bearing lubes	Forming	hot Die cast lubes, Forging compounds, cold Rolling oils, Drawing lubricants
Deburring, boring, milling, honing, drilling, grooving, turning, tapping, chamfering, broaching, and grinding	Spindle oils, gear lubes, way lubes, hydraulic fluids, greases, chain lubes, and bearing lubes	Machining	All classes of MWFs
Quenching, martempering, and carburization	Hydraulic fluids, greases, and bearing lubes	Heat Treating	All types of quenching fluids, martempering oil, and carburizer
Reaming, honing, lapping, grinding, and straightening	Spindle oils, gear lubes, way lubes, hydraulic fluids, greases, chain lubes, and bearing lubes	Finishing	Honing oil, tapping compounds, and MWFs
Cleaning, drying, degreasing, phosphatizing, and painting	Greases and bearing lubes	Cleaning and Surface Preparation	Cleaning compounds, degreasers, paint, and phosphatizing agents
Assembling	Hydraulic fluids and greases	Assembly	Degreasers and cleaning compounds

* Reprinted from "Lubes and Greases," July 1995, Vol 1, Issue 4, based on information provided by the ILMA.

3. Occupational Exposures to MWFs

3.1 The National Occupational Exposure Survey (NOES 1981-1983)

The National Occupational Exposure Survey (NOES, 1981-83) conduced by NIOSH provides estimates of the number of workers potentially exposed to chemical, physical, and biological agents. The NOES database consists of a stratified probability sample of 4,490 businesses in 98 different United States geographic locations representative of the non-agricultural, non-mining, and non-governmental businesses covered under the Occupational Safety and Health Act of 1970.

The NOES lists an estimated 1,115,900 workers as potentially exposed to agents collectively called "metalworking fluids" in 39 industry type codes (2-digit Standard Industrial Classification (SIC) codes). Approximately 58.7% of all workers potentially exposed to MWFs represented workers in three industry types (**Table 3-1, Industries showing the largest number of workers in specific occupations potentially exposed to MWFs (NOES, 1981-1983)** with almost 34% of them being employed in the "Machinery, Except Electrical" industry (SIC 35).

period was 0.92 mg/m^3 (total particulate). The percentage of total airborne exposure concentrations of less than 0.5 mg/m^3 increased from 36.7% of samples prior to 1980, to 42.5% of samples from 1980 to 1984, to 64% of samples from 1985 to 1990, and to 73% of samples after 1990. The arithmatic mean concentration for the period 1989-1994 is 0.49 mg/m^3

Table 3-3, OSHA-Integrated Management Information System (IMIS). Number of oil mist (mineral) samples and % by year ranges (February 1979-December 1994)

Mg/M^3	~~≤1980~~ 1979		1980-1984		1985-1990		>1990		TOTAL	
	# SAMPLES /	%	# SAMPLES /	%	# SAMPLES /	%	# SAMPLES /	%	# SAMPLES /	%
0.00*	22	(20.18)	62	(12.25)	221	(25.40)	182	(34.60)	487	(24.21)
> .0 - ≤ .1	1	(0.90)	15	(2.96)	58	(6.66)	37	(7.03)	111	(5.51)
> .1 - ≤ .3	5	(4.58)	72	(14.22)	166	(19.08)	114	(21.67)	357	(17.75)
> .3 - ≤ .5	12	(11.00)	66	(13.04)	108	(12.41)	51	(9.69)	237	(11.78)
> .5 - ≤ 1	20	(18.34)	32	(6.32)	23	(2.64)	26	(4.94)	101	(5.02)
> 1	49	(44.95)	259	(51.18)	294	(33.79)	116	(22.05)	718	(35.70)
TOTAL	109	(100%)	506	(100%)	870	(100%)	526	(100%)	2011	(100%)

* Non-detectable

Note: Table includes personal and area samples

mg/m^3 - Range 0 - 160 Mean 0.922 S.D. +/- 5.15

3.3 NIOSH Health Hazard Evaluations

Since 1967, NIOSH has conducted more than 70 onsite Health Hazard Evaluations (HHEs) of industries with occupational exposures to MWFs or mineral oil aerosols (**Table A.1, Health hazard evaluation of MWF and mineral oil mist exposures**). Dermatitis (skin irritation, eczema, rashes, oil acne) was the most frequently reported health problem, followed by worker complaints of eye, nose, and throat irritation (mucous membrane irritation), and respiratory symptoms or disorders (breathing problems, cough, chest tightness, asthma).

Exposure data compiled from 38 of the HHEs indicate that airborne MWF exposures have decreased over time. The arithmetic mean personal exposure concentrations (total particulates) were 1.23 mg/m^3 (n=21 plants) in the 1970s, 0.57 mg/m^3 in the 1980s (n=15 plants), and 1.0 mg/m^3 in the 1990s (n=2 plants). The overall mean concentration for the 38 plant-based HHEs was 0.96 mg/m^3. The exposure data collected at these 38 plants show airborne concentrations similar to those found in the OSHA IMIS dataset. Together, the two data sets suggest that the MWF mean total particulate exposure concentrations were between 1.0 and 0.5 mg/m^3 in many MW industries during the 1990s, indicating an overal reduction in airborne MWF exposures since 1980.

3.4 Reported Exposures in the Automotive Industry

Kriebel et al, 1994; Greaves et al, 1995 a,b; Robins et al, 1994 examined the respiratory effects and associated MWF airborne concentrations for exposed automobile manufacturing workers.

The arithmetic mean MWF airborne exposure concentration was reported by all three investigators to be <1.0 mg/m^3. Kriebel et al.[1994] reported mean exposure concentrations of 0.24 mg/m^3 (thoracic fraction) for aerosols of straight fluids and 0.22 mg/m^3 for soluble fluids; similar concentrations were reported by Greaves et al. [1995 a,b] with mean concentrations (thoracic fraction) for several plant surveys ranging from 0.2-0.68 mg/m^3 (straight fluids), 0.35-0.65 mg/m^3 (soluble fluids) and 0.41 mg/m^3 (synthetic fluids). Likewise, Robins et al. [1994] reported soluble MWFs exposures for automotive parts manufacturing workers of 0.1 to 0.6 mg/m^3 (thoracic fraction). The thoracic fraction comprises approximately 2/3 of the total particulate mass based on sample collection with a personal seven hole sampler with a 37 mm, 0.4 μ filter cassette [Kriebel et al, 1994]. Airborne MWF concentrations have significantly declined over the last 30-year period (1958-1987) with an arithmetic mean concentration of 5.42 mg/m^3 (total particulate) observed prior to 1970, and 1.82 mg/m^3 after 1980 [Hallock et al.1994]. The three data sources (OSHA IMIS, NIOSH HHEs discussed previously and the epidemiologic studies mentioned above and presented in **4.2 Nonmalignant Respiratory Effects**) suggest that the airborne exposure concentrations in the 1990s average lower (< 1.0 mg/m^3) than the 1.8 mg/m^3 average airborne exposure concentrations suggested for the 1980s by Hallock et al. [1994]. ← 3 plants

*

*quantify + critique

KRIEBEL - ABERRANTLY LOW PLANT

4. Occupational Health Risks for Workers Exposed to MWFs

4.1 Cancer Risks for Workers Exposed to MWFs

There has been concern since the 1940s that occupational exposures to some MWFs may be associated with skin and scrotal cancer, and since the 1970s the concern has included cancer at other organ sites. This chapter provides a review of the epidemiological studies that examined the association between MWF exposure and cancer.

4.1.1 Criteria for Inclusion

In order to be included in this review an article had to be published in a peer-reviewed journal. Articles were identified from computerized database searches, recommendations of reviewers of earlier drafts of the review, and from references cited in relevant articles. Studies providing data on the association between MWF exposure and cancer were grouped into three categories based on their study design : 1) retrospective cohort mortality and cancer incidence studies of MWF-exposed cohorts; 2) proportionate mortality ratio (PMR) studies of occupational groups exposed to MWF; and 3) population based studies (primarily case-control interview studies of specific cancer sites which examined cancer risks associated with MWF exposure, or with occupations likely to have MWF exposure (metal machinists, grinders, toolmakers). The category "population based studies" include hospital based case-control studies because their usual intent is to estimate risks in the general population. Some cohort and PMR studies include nested case-control analyses to examine risks associated with particular departments, jobs or exposures. For fuller discussion on these study designs and their interpretations, the reader is referred to a textbooks on occupational epidemiology (Monson, 1990) as well as standard epidemiologic textbooks (Mausner and Kramer, 1985; Kleinbaum, Kupper, and Morgenstern, 1982). In order to present the results of the review systematically, the data have been summarized by cancer site in both tables and text.

Not included in the reviews by cancer site are hypothesis-generating studies which examined broad occupational categories based on census or death certificate data. The results of such studies have been summarized separately for all sites combined. A single epidemiologic stud~~ies~~y of genetic endpoints will also be briefly reviewed.

4.1.2 Studies of Cancer in Broad Occupational Groups

Studies have been conducted which evaluated the risks for many specific cancers among many different occupations, some of which had the potential for MWF exposure (Hrubec et al., 1992; Guralnick, 1963; Howe et al., 1983; Tola et al., 1988; Milham, 1983; Petersen and Milham, 1980, Gallagher and Threlfall, 1983; Williams et al., 1977; Decoufle et al., 1977;

Dubrow and Wegman, 1984; Bulbulyan et al., 1992). For the most part, the intent of these studies was hypothesis generation. These studies have included populations from many different geographic areas (e.g. United States, United Kingdom, Finland, Canada, Australia, the Netherlands, Sweden).

Inherent weaknesses of this type of study include the use of broad occupational categories to define MWF exposure, use of potentially inaccurate sources (death certificates, census data) to define the occupation, the large number of associations tested, which means that some associations are expected to be statistically significant by chance alone, and the inability to control for important confounders such as smoking and alcohol. With these limitations in mind, the findings from these studies suggest an association between MWF exposures and certain cancers. For each of the following categories, two or more of the studies found that occupations with potential MWF exposure had significantly increased risk: stomach cancer (Howe, 1983; Hrubec, 1992), colorectal cancer (Guralnick, 1963; Dubrow et al., 1984), lung cancer (Milham, 1983; Petersen and Milham, 1980; Hrubec et al., 1992; Gallagher and Threlfall, 1983), bladder cancer (Milham, 1983; Petersen and Milham, 1980; Hrubec et al., 1992), leukemia (Petersen and Milham, 1980; Howe et al., 1983; Decoufle et al., 1977), and all cancers combined (Hrubec et al., 1992; Gallagher and Threlfall, 1983).

Studies of MWF-exposed Populations)

4.1.3.2a Cohort studies

Two of 7 cohort studies found a significantly elevated risk for stomach cancer (Rotimi et al, 1993; Park et al, 1994). In one, stomach cancer mortality was increased among white hourly workers employed at two Ohio engine manufacturing plants (standardized mortality ratio [SMR]=2.54, 95%CI=1.42, 4.20) (Rotimi et al., 1993). Using local mortality rates to account for a high proportion of foreign-born workers, the magnitude of association decreased but the SMR for stomach cancer remained significantly elevated and displayed a dose-response relationship with duration of exposure (SMR not provided). Most of the excess in this cohort occurs among those hired before 1955, with 20 or more years since first hire, who were employed for 20 or more years. Park et al. (1994) found a significantly increased mortality odds ratio (MOR) (9.65, 95% CI:2.3, 40) among "tool and die" workers exposed to MWFs at a stamping plant~~s~~ within the automobile industry.

4.1.3.2b PMR Studies

Two of 4 PMR studies found a significantly elevated risk for stomach cancer (Silverstein et al., 1988; Park et al., 1988). Silverstein et al. (1988) found that white men employed 5 or more years in a ball bearing manufacturing plant had an elevated risk for stomach cancer mortality (proportionate mortality ratio [PMR]=1.97, p<0.001). The risk was greatest among white men with 10 or more years employment in grinding operations which used primarily soluble oils (PMR=3.39, p<0.001), and was not found among machinists who used straight oils. The association with grinding persisted even after adjusting for Central European origin using logistic regression. Silverstein et al. (1988) concluded that their studies provided strong evidence that grinding operations using soluble cutting fluids increase the risk for stomach cancer. Grinding operations generally have high concentratons of total particulate exposure [Hallock et al. 1994]. White men employed in grinding operations at another ball bearing manufacturing plant were also found to have excess stomach cancer mortality (PMR=3.8, p=0.006) (Park et al., 1988). Workers classified as having soluble MF exposure in a nested case-control analysis were found to have an odds ratio for stomach cancer of 6.2 (p=0.05) (Park et al., 1988). In interpreting these findings Park et al. did not think that there was confounding by the Central European origin of the workers because they found no association between such origin and soluble MF exposure.

4.1.3.2c Population-based studies

One of 3 population-based studies found a significant association between working in an occupation with potential MWF exposure and risk of stomach cancer (Kneller et al., 1990). A standardized incidence ratio (SIR) study of incident stomach cancer cases reported to the Shanghai, China Cancer Registry found that men with a current occupation of metal grinder, polisher, tool sharpener or machine-tool operator had an elevated risk for stomach cancer (SIR=1.41, $p<0.01$)(Kneller et al., 1990).

Among those studies which did not report significant elevations of stomach cancer, the Eisen et al. (1992) and Tolbert et al. (1992) studies were the largest and best designed. Nonsignificant elevations in stomach cancer mortality for white workers were observed in 2 of the 3 study plants (Plant 1: SMR=1.08, 95%CI=0.84-1.36); Plant 2: SMR=1.26, 95%CI=0.87-1.77; Plant 3: SMR=0.59, 95%CI=0.16-1.50) (Eisen et al., 1992). Black males did not have an elevated SMR for stomach cancer associated with either exposure to straight oils or soluble oils (**Table 4.1-3**). Analysis of risks associated with specific fluid types (**Table 4.1-3**) indicated slight excesses of stomach cancer in each of the exposure groups. Poisson modeling did not suggest strong exposure-response trends for any of the 3 exposure types, although there was a slight elevation in the highest exposure group for soluble oils relative to other strata (Tolbert et al., 1992). Although not statistically significant, the results of the Tolbert et al. (1992) and Eisen et al. (1992) studies tend more towards suggesting a relationship between cutting oil exposure and stomach cancer, than arguing against it.

In conclusion, the evidence from several studies suggests an association between stomach cancer and MWF exposure, especially among workers with prolonged MWF exposure. The findings from Park et al. (1988) and Silverstein et al. (1988) suggest that grinding operations using soluble ~~oil~~ MF (sol, semi and synth) may be associated with an elevated risk for stomach cancer.

4.1.3.3 Pancreatic cancer (Table 4.1-4, Results for Pancreatic Cancer from Epidemiologic Studies of MWF-exposed Populations)

4.1.3.3a Cohort studies

Among five cohort studies reporting site-specific data for pancreatic cancer, one found a significant excess for white workers in a subgroup analysis (Acquavella et al., 1993), and one found a significant excess for black but not white workers (Rotimi et al., 1993) (**Table 4.1-4**). Acquavella et al. (1993) reported that workers employed at an Iowa metalworking facility had an increased risk for pancreatic cancer mortality (SMR=2.0, 95%CI=0.9,3.8). The risk appeared to be greatest among workers employed 10 or more years who were hired between 1950 and 1959 (SMR=3.6, 95%CI=1.2-8.3). Assembly workers, who Acquavella et al. state are unlikely to have MWF exposure, were the occupational group with the highest risk (SMR=3.0, 95%CI=1.0,7.5). Rotimi et al. (1993) found that black men employed at two Ohio engine manufacturing plants had an excess pancreatic cancer mortality (SMR=3.03, 95%CI=1.21,6.24). The authors report that there was no consistent pattern with respect to time since hire or duration of employment. No excess was observed in white workers. Tolbert et al. (1992) found excess pancreatic cancer mortality among black workers exposed to soluble oils at Plants I and II in Michigan (SMR=1.6, 95%CI=1.0,2.5). In a Poisson regression analysis that controlled for race, age, and gender, an increased risk for pancreatic cancer mortality was observed in those workers with the highest exposures to synthetic MWFs (rate ratio=2.04, 95%CI=0.88-4.72) (Tolbert et al., 1992). The authors concluded that their study provided "limited evidence" of an association between synthetic oil exposure and pancreatic cancer.

4.1.3.3b PMR Studies

Among 4 studies reporting site-specific data for pancreatic cancer, one found significantly elevated PMR among white workers (Vena et al., 1985) and one found a significantly elevated PMR among black but not white workers ((Mallin et al., 1986). White men employed at an engine plant for at least 10 years had an excess of pancreatic cancer mortality (PMR=1.89, $p<0.05$), which was higher for those employed > 20 years (PMR=2.32, $p<0.05$) (Vena et al., 1985). Use of county referent rates results in higher PMR's (employed > 20 years, PMR=2.97, $p<0.05$; employed $\leq$ 1950, PMR=3.19, $p<0.05$). PMR's for non-whites employed in the engine plants were not reported due to small numbers of deaths. All three types of cutting fluids were used in the engine plants. Mallin et al. (1986) found a significant excess of pancreatic cancer among black (PMR=3.57, $p<0.05$) but not white (1.19, n.s.) men employed in the manufacture of diesel engines and construction equipment. The PMR for pancreatic cancer was still higher for black men who died after 20 years of service (PMR=4.79, $p<0.01$). When county referent rates were used, only the PMR for blacks with ≥ 20 years of service was statistically significant. Another PMR study found a nearly significant elevation in pancreatic cancer mortality among whites at a ball bearing manufacturing plant (PMR=1.43, 95%CI=0.96,2.12) (Silverstein et al., 1988). Case-control analyses revealed substantially elevated risks associated with 10 or more years employment in grinding (OR=3.10, $p=0.05$) and machining (OR=3.71, $p=0.05$). There were too few deaths among non-white men for analysis. Logistic regression models of mortality odds ratios revealed highly significant elevated risks with a) machining in straight oil (OR=9.6, p=.002) and b) grinding, but with a downward trend (OR=4.9 p=.005) for year of hire = 1955 (Silverstein et al. 1988).

4.1.3.3c Population-based studies

One study of white males in Los Angeles county during the period 1972 to 1977 reported a nonsignificant "proportional index ratio" for employment as a machinist (Mack and Paganini-Hill, 1981).

In conclusion, several studies have found significantly increased risks of pancreatic cancer among workers exposed to MWF. These risks were not associated with any specific type of MWF, with the exception of "limited evidence" for an association with synthetic MWF's in the

Tolbert et al. (1992) study. Although a number of the studies did not have internally consistent findings (i.e. excesses in black but not white workers, lack of association with duration or intensity of MWF exposure) the number of studies with statistically significant findings suggests that exposure to some MWF's (or a less likely confounding exposure present in some work environments where MWF's are used) may increase the risk of pancreatic cancer.

4.1.3.4 Laryngeal cancer (Table 4.1-5, Results for Laryngeal Cancer from Epidemiologic Studies of MWF-exposed Populations)

4.1.3.4a Cohort studies

Aside from the studies of three automobile manufacturing plants conducted by Eisen et al. (1992) and Tolbert et al. (1992), only one other cohort study has reported site-specific data for laryngeal cancer (Rotimi et al., 1993) (**Table 4.1-5**). Tolbert et al. (1992) reported a statistically significant SMR of 1.98 for laryngeal cancer among whites ever exposed to straight oil, and a nearly significant SMR of 1.41 for soluble oil exposure in Plants I and II combined. The SMR for laryngeal cancer in Plant 3 was not elevated (Eisen et al., 1992). In a case-control analysis including all three plants, and incident as well as deceased cases, a categorized exposure analysis found an OR of 2.23 (95% CI 1.25,3.98) among individuals with > 0.5 mg/m^3 years straight oil particulate exposure. Eisen et al. (1994) also examined the association between laryngeal cancer and specific components or contaminants of MWFs (biocides, steel, iron, aluminum, sulfur, and chlorine). There was some evidence for confounding by sulfur, but models including both exposure variables still had a significantly elevated OR in the highest straight MWF exposure category (OR=1.91, 95%CI=1.01,3.62). Although unable to adjust for smoking and alcohol, two important risk factors for laryngeal cancer (Austin, 1982), the authors did not think that these risk factors confounded their results. They found that the risk of lung cancer and cirrhosis did not increase with increasing exposure to straight oils, suggesting that an association did not exist between straight oil exposure and cigarette smoking or alcohol use. Eisen et al. (1992) reported a decreased risk for laryngeal cancer among white workers employed at plant III (SMR=0.77, 95%CI=0.09, 2.79). The risk for laryngeal cancer reported in the Rotimi et al. (1993) study was < 1.00.

4.1.3.4b PMR studies

Among the two PMR studies reporting site-specific results for larynx cancer, both reported PMR's of 1.8 which were not statistically significant (Vena et al., 1985, Mallin et al., 1986). In the Vena et al. (1985) study, a significantly elevated PMR was found for workers employed less than 20 years and those who were employed after 1950 (PMR=3.95, P<0.005 for both subgroups).

4.1.3.4c Population-based studies

Among 5 studies which defined occupational categories in sufficient detail to examine risk associated with exposure to MWF, one found a significant risk for "ever employment as a machinist" (SMR=2.5, 95%CI=1.2,5.2) or metal grinder ((SMR=2.1, 95%CI=1.0-4.7) after adjustment for smoking and alcohol (Zagraniski et al., 1986). A case-control study (hospital-based) of 100 laryngeal cancer cases and 100 controls found a non-significantly elevated risk among those who self-reported ever having mineral oil exposure (OR=2.2, 95%CI=0.9,5.3, adjusted for smoking and alcohol) (Ahrens et al., 1991).

In conclusion, several studies suggest that MWF exposure may be associated with laryngeal cancer. In particular, the studies by Eisen et al. (1992, 1994) and Tolbert et al. (1994) suggest that laryngeal cancer is associated with exposure to straight oils. Negative data from mortality studies must be interpreted with caution for this site, because of its high (> 60%) 5 year survival rate (SEER, 1994). and low expected incidence.

4.1.3.5 Rectal cancer (Table 4.1-6, Results for Rectal Cancer from Epidemiologic Studies of MWF-exposed Populations)

4.1.3.5a Cohort studies

Aside from the studies of three automobile manufacturing plants conducted by Eisen et al. (1992) and Tolbert et al. (1992), only one cohort study has reported site-specific results for rectal cancer (Decoufle, 1978). Tolbert et al. (1992) reported an association between straight oil exposure and rectal cancer among whites but not black workers. The SMR for ever-exposure to straight oils among whites was 1.47 (95%CI=1.04-2.03). Poisson regression analyses revealed a trend of increasing rectal cancer risk in relation to years of exposure to straight oils ($p < .0001$). The rate ratio for the most highly exposed group was 3.2 (95%CI=1.6-6.2). Plant III had a non-significant excess (SMR = 1.70) (Eisen et al., 1992). The Decoufle (1978) study found a slight excess of rectal cancer mortality which was not statistically significant.

NOT IN
TABLE
4.1-6

4.1.3.5b PMR Studies

Among the 5 PMR studies reporting data for rectal cancer, one found a significant excess in the cohort overall (PMR=3.07, 95%CI=1.54,5.50) (Park et al., 1988) which was not associated with any particular process. A second PMR study found a significant excess in a subgroup with employment in an engine plant for > 20 years (PMR=2.76, $p<0.05$) (Vena et al., 1985).

system, colon cancer and hematopoietic and lymphopoietic cancer is equivocal (Tables 8-13), and will only be briefly summarized here.

4.1.3.7 Esophageal cancer (Table 4.1-8, Results for Esophageal Cancer from Epidemiologic Studies of MWF-exposed Populations)

← Brain?

Among 4 cohort studies which reported site-specific results for this cancer, none found a significant excess. One of 5 PMR studies reported a significant excess (Silverstein et al., 1988). However, no further analyses by exposure or job category were performed.

4.1.3.8 Brain/Nervous System Cancer (Table 4.1-9, Results for Brain/Nervous System Cancer from Epidemiologic Studies of MWF-exposed Populations)

Among 3 cohort studies which reported site-specific results for brain/nervous system cancer, none found a significant excess. Tolbert et al. (1992) reported a nearly significant association with soluble oil exposure among whites (SMR=1.24, 95%CI=0.91,1.66). None of 5 PMR studies which reported results for brain/nervous system cancer found a significant elevation. A case-control study using death certificate data to classify occupation reported a significant association between all brain cancers and "usual occupation of precision metal worker" (OR=2.1, 95% CI=1.2,3.6) (Thomas et al., 1986). An case-control interview study of astrocytic brain tumors (including cases identified in the Thomas et al., 1986 study) reported an odds ratio of 1.6 (95%CI=1.0,2.6) for "ever employed at a job with potential cutting oil exposure (Thomas et al., 1987).

4.1.3.9 Prostate cancer (Table 4.1-10, Results for Prostate Cancer from Epidemiologic Studies of MWF-exposed Populations)

Among 4 cohort and 4 PMR studies which reported site-specific results for prostate cancer, none showed a significant excess. Tolbert et al. (1992) observed slight excesses for each exposure group among the white males, and a mild exposure-response trend for exposure to straight machining fluid (p=.03). The rate ratio for ≥ 7.5 years of exposure was 1.5 (95%CI=1.01-2.29). One study found a significant deficit of prostate cancer among "grinders and turners" (SMR=0.34, 95%CI=0.1,0.7) and "grinders and turners" with at least 20 years since onset of exposure (SMR=0.38, CI=0.1,0.8) (Jarvholm and Lavenius, 1987).

4.1.3.10 Lung cancer (Table 4.1-11, Results for Lung/Respiratory system Cancer from Epidemiologic Studies of MWF-exposed Populations)

Of 5 cohort studies reporting site specific results for lung cancer, only one reported a statistically significant increased risk, and this was for a subgroup of workers employed > 10 years and hired between 1950 and 1959 (Acquavella et al., 1993). Some studies, in fact, provide evidence for a negative association between MWF exposure and lung cancer. Although the SMR analyses in the Tolbert et al.(1992) study showed SMR's $>$ 1.00 for "ever exposure" to each of three types of machining fluids, the poisson regression analysis found a negative association between lung cancer risk and both synthetic oil exposure (p=0.006), and soluble oil exposure (p=0.09). Tolbert et al. (1992) suggested that the negative findings may be due to contamination of the water-based fluids by endotoxin-producing gram-negative bacteria. The mechanism proposed by these authors was that endotoxins may stimulate immunologic factors that inhibit the growth of malignant cells in the lung. Jarvholm and Lavenius (1987) also found that workers exposed to oil mist for 5 or more years had a significantly decreased risk for lung cancer (SMR=0.4, 95%CI=0.1,0.9), even among those with at least 20 years since onset of exposure (SIR=0.30, 95%CI=0.1,0.9) **(Table 4.1-11)**. Three of 4 PMR studies report PMR's $>$ 1.0, but none are statistically significant. A case-control study within a PMR study reported a significantly increased risk for females employed as grinders, but the absence of any cases in the comparison group made this an unstable estimate (Park et al., 1988). Two population based case-control studies report statistically significant odds ratios associated with various definitions of MWF exposure (Jockel et al., 1992, Coggan et al, 1984).

4.1.3.11 Colon cancer (Table 4.1-12, Results for Colon Cancer from Epidemiologic Studies of MWF-exposed Populations)

Among 5 cohort studies reporting site-specific data, none show significant excesses. One of 4 PMR studies was significantly positive (Silverstein et al., 1988). Silverstein et al. found a significant excess in colon cancer (PMR=1.39, 95%CI=1.03, 1.88), which appeared to be concentrated in workers employed in grinding operations (13 observed, 6.9 expected, PMR=1.89, p=0.02). Further analyses using logistic regression found no exposure associations for colon cancer.

4.1.3.12 Hematopoietic and lymphopoietic cancer (Table 4.1-13, Results for Hematopoietic and Lymphopoietic Cancer from Epidemiologic Studies of MWF-exposed Populations)

One of 4 cohort studies reporting data on these cancers found a significantly increased risk. Tolbert et al. (1992) found that white males ever exposed to soluble oils had an increased risk for leukemia (SMR=1.33, 95%CI=1.05, 1.67), but Poisson regression models found no evidence for an association between leukemia and any class of MWF. Another SMR study found an elevated ~~leukemia~~ hematopoietic/lymphopoietic cancer risk in a small subgroup (workers employed one or more months in the tool and die area of an automotive stamping plant) (MOR=5.38, 95%CI=1.6, 18.0) (n=3) (Park et al., 1994). Among 4 PMR studies, none found significantly elevated PMRs in the overall analyses or in major subgroups. Silverstein et al. (1988) found that workers employed as tool grinders for 10 or more years had an increased risk for lymphopoietic cancer (PMR=4.75, p=0.02). Mallin et al. (1986) found an elevated risk for non-Hodgkin's lymphoma among black workers (PMR=687 6.87, p<0.05). In each of these three studies, the authors speculated that solvent exposure, rather than MWF fluid exposure, was likely to be responsible. A population-based case-control study that examined the association between several cancer sites and occupational exposure to several petroleum-derived liquids found evidence suggesting a dose-response relationship between cutting oil exposure and an increased risk for non-Hodgkin's lymphoma (OR among those defined as substantially exposed=1.9, 90%CI=1.0,3.1) (Siemiatycki et al.,1987).

NOT IN TABLE 4.1-13

4.1.4 Genetic effects

Only one epidemiologic study was identified which examined genotoxicity among workers exposed to MWF. In a German study of 65 male metal workers exposed to synthetic oils in seven small to medium sized plants, those who worked in areas having a N-nitrosodiethanolamine (NDELA) concentration greater than 500 ng/m3 had a significantly elevated mean level of DNA strand breaks in mononuclear blood cells compared to workers employed in areas with less than 50 ng/m3 NDELA (1.69± 0.34 among those working in areas with greater than 500 ng/m3 NDELA versus 0.76± 0.05 among those working in areas with less than 50 ng/m3 NDELA, $p<0.01$)(Fuchs, 1995). The average level of NDELA present in the cutting fluids at these plants was 20.6 ppm (range 2-135 ppm). In addition, nonsmokers who worked more than 4.5 hours per day were found to have a significantly elevated mean level of DNA strand breaks compared to nonsmokers who worked less than 4.5 hours per day (1.34± 0.12 among those working more than 4.5 hours per day versus 0.91± 0.12 among those working less than 4.5 hours per day, $p<0.02$). Airborne concentrations of MWFs were not reported. NDELA can be formed in MWFs when diethanolamine or triethanolamine reacts with a nitrosating agent (e.g. nitrite). This study provides evidence that nitrosamine exposure may be genetoxic. However, in the United States in 1984, EPA prohibited the addition of nitrosating agents to MWF as previously discussed in **6. Potentially Hazardous Contaminants**.

4.1.5 Information on Exposure Concentrations

Only a few studies described in this chapter provided information on the exposure levels of MWFs during the decades when the MWF-exposed cohorts were employed (Jarvholm and Lavenius, 1987; Hallock et al., 1994; Silverstein et al., 1988 ; Park et al., 1988). A summary of the exposure information from these studies is provided below. Additional detail about MWF exposures can be found elsewhere is this document.

In the Jarvholm study, the investigators estimated that cutting oil mist concentrations prior to 1965 were 5 mg/m^3 or greater in the grinding and turning departments. All workers in this study were exposed to these levels since the criteria for inclusion in the study required employment in the turning or grinding department at any time between 1950 and 1966 and a duration of employment of at least 5 years. In the late 1970s, oil mist concentration was reduced to 2 mg/m^3 in the turning departments and to 3 mg/m^3 in the grinding departments.

Estimates of cutting oil exposures were also made for the three plants studied by Eisen et al. (1992) (Hallock et al., 1994). The estimates were made by fitting industrial hygiene aerosol concentration measurements made between 1958 and 1987 into a linear statistical model. As demonstrated in the **Table 4.1-14, Estimated Mean Aerosol Concentrations for Grinding, Machining, and Assembly Operations,** MWF exposure from grinding operations were higher than the exposure from machining or assembly operations, for all three time periods.

Table 4.1-14, Estimated Mean Aerosol Concentrations for Grinding, Machining, and Assembly Operations

Operation	Mean Aerosol Concentration (mg/m^3) by Time Period		
	n 1958-1969 $\bar{x}$	n 1970-1979 $\bar{x}$	n 1980-1987 $\bar{x}$
Grinding	17.96	3.44	2.28
Machining	3.35	2.13	1.66
Assembly	0.94	0.52	0.64

↑

need nos. of samples, based on

[illegible] - very no!

Exposure measurements from which these summaries are compiled may not be representative of the plant environment as a whole. Most (57%) of the industrial hygiene reports did not indicate the reason for sampling. The remainder were done at employee or management request, or were performed by the state health department ((Hallock et al., 1994). It is almost certain ~~possible~~, therefore, that these averages may be higher than the true plant averages because the measurements represent complaint sampling of higher than average exposures (Hallock et al., 1994).

Similar data were reported by Silverstein et al. (1988) and Park et al. (1988). Silverstein et al. (1988) reported that between 1949 and 1961, industrial hygiene breathing zone samples for total particulates in machining areas had a mean level of 15.9 mg/m^3. Breathing zone samples from grinders taken from 1977-1979, and in 1980, had mean total particulates of 1.7 mg/m^3, and 4.3 mg/m^3, respectively. Park et al. (1988) found that breathing zone total particulate concentrations near jobs performed with straight oils (machining operations) ranged from 0.07-2.8 mg/m^3 between 1972 and 1980. During these same years, concentrations near grinding operations, which were presumed to be using soluble oils, ranged from 0.6-7.2 mg/m^3.

This evidence suggests that grinding operations are associated with higher MWF exposures

PAHs from MWFs began in the 1950s and EPA regulations in the 1980s were directed at reducing nitrosamine exposures. Given the small number of epidemiologic studies that have adequate exposure characterization to associate~~d~~ the individual constituents of MWF exposure with site-specific cancers, the specific MWF constituent(s) responsible for the elevated risk for the various site-specific cancers remains to be determined.

Within the Tolbert et al. study, straight oil exposure was found to be associated with an increased risk for laryngeal cancer and rectal cancer, while synthetic oil exposure was associated with an increased risk for pancreatic cancer. With the exception of an association with stomach cancer w/ SOL MFs (including semi synth + synthetics) observed in two PMR studies (Park et al., 1988; Silverstein et al., 1988), there is no evidence that soluble oil exposure is consistently associated with cancer at any specific site. Soluble oils contain many of the ingredients found in straight oils, but in different concentrations. Tolbert et al. (1992) speculate that the absence of an association with soluble oil exposure may be due either to an alteration of the carcinogenic agent as a result of the emulsification of the oils, or to the reduction of the amount of carcinogen in the soluble oils, thereby requiring a much more statistically powerful study to detect any cancer associations.

Smoking and alcohol are associated with some of the cancers observed to be associated with MWF exposure. When appropriate, based on cancer site, most of the case-control studies controlled for these exposures or determined that these exposures were unlikely confounders.

Information on these lifestyle factors are not often collected in occupational cohort mortality or PMR studies. However, it has been demonstrated that smoking is unlikely to account for relative risks >1.3 for lung cancer and other smoking related diseases (Siemiatycki, 1988).

The studies that provide the bulk of the evidence suggesting an association between MWF exposure and cancer involved workers employed as early as the 1930s and as late as the mid-1980s. Because there is a latency period of 10-20 years, on average, between initial exposure to a carcinogen and the initial appearance of a solid organ cancer caused by that carcinogen, the excess cancer mortality observed in these cohort studies most likely reflects the cancer risk associated with exposure conditions in the mid-1970s and earlier. Over the last several decades, substantial changes have been made in the metalworking industry, including changes in MWF composition and reduction of some impurities. However, since there is little epidemiological data to support the association of any of these factors with the cancer risks observed in earlier cohorts, there is insufficient data to conclude that these changes will have prevented any further carcinogenic risks. Reductions in the exposure concentrations likely have reduced the risk. Most workers entering MWF exposed jobs in the mid-1970's or later have not yet reached the minimum latency period of 15-20 years since first exposure necessary to evaluate their cancer risk, nor has anyone assembled a large cohort of such workers for a definitive study. The substantial evidence that MWFs in commercial use prior to the mid-1970s ~~is~~ are associated with cancer at several sites, and the possible potential for current MWF to pose a similar carcinogenic hazard, support the recommendation based on respiratory disease risks for a reduction in allowable airborne MWF exposures.

Table 4.1-1, Description of the MWF-exposed cohort and proportionate mortality studies

Author	Type of Study/ Analysis	Study Population	Employment Criteria for Inclusion	Total number of subjects	Number of malignant neoplasms	Years of Follow-up
Jarvholm & Lavenius, 1987	SIR	Grinding & turning department workers employed at a bearing ring manuf. plant -Sweden	Exposed at least 5 years and employed at any time between 1950-1966	White males = 792	67	1958-1983
Jarvholm et al., 1985	SIR	Turning department workers employed at bearing ring manuf. plant -Sweden.	Employed at any time between 1950-1980	White males = 682	24	1960-1980
Tolbert et al., 1992	SMR	Hourly workers. Plant I: Gear and Axle, Hamtramak. Produces axles and gears. Plant II. Hydra-Matic, Ypsilanti. Produces transmissions.	Employed at least 3 years prior to 1/1/85.	White males = 17,743 Black males = 5,641	White = 164 8 Black = -224 (Plant I only)	Through 1/1/85
Eisen et al., 1992	SMR	Plant III. Saginaw Steering Gear. Manuf. steering gears.	Employed at least 3 yrs between 1939-1/1/85.	White males = 8,983	White = 183	Through 1/1/85
Rotimi et al., 1993	SMR	Hourly workers at Ohio engine manuf. plants	Employed anytime between 1973-1986. Also retirees alive as of 1970. No minimum employment	White males = 5,331 Black males = 1,180	White = 178 Black = 60	1970-1987
Decoufle, 1978	SMR	Blue-collar workers at a metal machining plant in North Central part of US. Machined grey iron castings.	At least 5 years employment in oil mist exposed jobs between 1938-1967.	White males = 2,485	139	To 1/1/68
Acquavella et al., 1993	SMR	White workers at a metal working facility, approx. 59% held factory jobs. Mortality data in following tables only includes the factory workers.	Hired between 1950-1967, and employed at least 6 months.	White males = 2664 White females = 966	White = 103	1950-1987
Park et al. 1994	MOR	workers employed in a "tool & die" area, automotive stamping & assembly plant	two year employment before 1/1/89	NA	NA	1978-1988
Silverstein et al., 1988	PMR	Union workers at a ball bearing manufacturing plant, Connecticut.	Died between 1950-1982, and employed 5 or more years.	White males = 1,532	White = 342	1950-6/30/82
Park et al., 1988	PMR	Hourly workers at a ball bearing manufacturing plant, Connecticut. Soluble oils predominantly used in grinding operations.	Died between 1/1/69 and 7/31/82 and employed 10 or more years.	White = 610	White = 157	1969-1982

Soluble MFs
(incl. semi, syn.)

Vena et al., 1985	PMR	Union workers at an engine plant (machine & assembly), New York. Before 1950, soluble and insoluble oils used. Widespread use of synthetic oils in mid-1950s.	Died between 1/1/70 and 12/31/79 and employed 10 or more years. Plant began operation in 1938.	White men = 472 Black men = 37	White = 128	1970-12/31/79
Mallin et al., 1986	PMR	Diesel engine & construction equip manufacturer, Illinois. 10+yrs employed. 461 eligible deaths among union workers.	Died between 1/1/70 and 3/31/82 and employed 10 or more years. Plant began operation in 1948.	White males = 351 black males = 110	White = 92 Black = 36	1970-2/31/82

Table 4.1-2, Results for Skin Cancer from Epidemiologic Studies of MWF-exposed Populations

Author	Location	Type of Study/ Analysis	# with CA or # Exposed Cases	Risk Estimate	95% CI (or p-value)	Study Population/Cancer Site
Cohort studies						
Jarvholm et al., 1985	Sweden	SIR	5	16.6	P<0.001	turners employed between 1960 and 1980/squamous cell cancer of the skin
Jarvholm & Lavenius, 1987	Sweden	SIR	7	– –[1]	– –	turners only/scrotal cancer
Eisen et al., 1992	Michigan	SMR	10	0.61	0.29,1.13	white autoworkers, Plant I
			11	1.06	0.53,1.89	white autoworkers, Plant II
			7	1.27	0.51,2.62	white autoworkers, Plant III
Proportionate Mortality Studies						
Silverstein et al., 1988	Connecticut	PMR	4	1.00	0.70,1.43	white
Park et al., 1988	Connecticut	PMR	4	1.88	0.51,4.80	white
Vena et al., 1985	New York	PMR	1	0.60	NS	based on US mortality, white
Population-Based Studies						
Rousch et al., 1982	Connecticut	case/control	26	10.5	4.0,36.9	Ever employed as toolmaker, setter, set-up man, hardener, polisher, automatic screw operator, machinist, or machine operator/squamous cell cancer of the scrotum

---

[1] There were too few expected cases of scrotal cancer to make a reliable estimate of risk.

Table 4.1-3, Results for Stomach Cancer from Epidemiologic Studies of MWF-exposed Populations

Author	Location	Type of Study/ Analysis	# with CA or # Exposed Cases	Risk Estimate	95% CI (or p-value)	Study Population/Cancer Site
Cohort studies						
Tolbert et al., 1992	Michigan	SMR	49	1.12	0.84,1.48	ever straight oil exposure, white
			99	1.19	0.97,1.45	ever soluble oil exposure,white
			21	1.28	0.79,1.96	ever synthetic oil exposure, white
			5	0.76	0.24,1.77	ever straight oil exposure, black
			17	1.01	0.59,1.62	ever soluble oil exposure, black
Eisen et al., 1992	Michigan	SMR	4	0.59	0.16,1.50	white autoworkers, Plant III
Rotimi et al., 1993	Ohio	SMR	15	2.54	1.42,4.20	engine plant,white
			2	0.85	0.1,3.06	engine plant, black
Acquavella et al.,1993	Iowa	SMR	5	1.4	0.4,3.2	total workforce
			2	2.3	0.3,8.1	factory workers employed > 10 years, hired between 1950-1959
Decoufle, 1978	Michigan	SMR	17	1.26	NS	white
			11	1.67	NS	white, 5 or more years of heavy exposure to oil mist
Park et al., 1994	Ohio	SMR	6	4.4	1.6,9.6	stamping plant
		MOR	2	9.55	2.3,40	ever tool and die worker
Jarvholm & Lavenius, 1987	Sweden	SIR	8	1.11	0.5,2.1	grinders and turners
Proportionate mortality studies						
Silverstein et al., 1988	Connecticut	PMR	35	1.97	1.43,2.72	white
			13	3.39	p<0.001	employed in grinding 10 or more years
Park et al., 1988	Connecticut	PMR	11	1.99	1.12,3.54	white
		MOR	8	6.2	p=0.06	nested case-control study-those workers ever exposed to soluble oil
Vena et al., 1985	New York	PMR	4	0.91	NS	based on US mortality, white
			3	1.37	NS	employed in engine plant > 20 years
Mallin et al., 1986	Illinois	PMR	6	1.86	NS	white

Population-based studies

Kneller et al., 1990	China	SIR	191	1.41	P<0.01	metal grinder, polisher, tool sharpener, machine-tool operator
			193	1.11	NS	toolmaker, metal patternmaker, metal worker
Chow et al., 1994	Sweden	SIR	376	1.11	NS	toolmakers and machinists
Siemiatycki et al., 1987	Montreal	case/control	24	1.1	90%CI-0.8,1.4	ever exposed to cutting oils

Table 4.1-4, Results for Pancreatic Cancer from Epidemiologic Studies of MWF-exposed Populations

Author	Location	Type of Study/ Analysis	# with CA or # Exposed Cases	Risk Estimate	95% CI (or p-value)	Study Population/Cancer Site
Cohort Studies						
Tolbert et al., 1992	Michigan	SMR	34	0.80	0.55,1.11	ever straight oil exposure, white
			61	0.77	0.59,1.00	ever soluble oil exposure,white
			19	1.03	0.62,1.61	ever synthetic oil exposure, white
			8	1.40	0.60,2.77	ever straight oil exposure, black
			19	1.62	0.98,2.64	ever soluble oil exposure,black
Eisen et al., 1992	Michigan	SMR	8	0.87	0.37,1.71	white autoworkers, Plant III
Rotimi et al., 1993	Ohio	SMR	8	0.91	0.39,1.79	engine plant,white
			7	2.03	1.21,6.24	engine plant, black
Acquavella et al.,1993	Iowa	SMR	11	2.0	1.0,3.6	total workforce
			5	3.6	1.2,8.2	factory workers employed >10 years, hired between 1950-1959
Decoufle, 1978	Michigan	SMR	8	1.05	NS	white
			1	0.27	NS	white, 5+years of heavy oil mist exposure
Proportionate Mortality Studies						
Silverstein et al., 1988 *	Connecticut	PMR	24	1.43	0.96,2.12	white
		MOR	9	3.10	p=0.05	employed in grinding 10 + years
		MOR	5	3.71	P~~<~~0.05 =.05	employed in machinery 10+ years
Park et al., 1988	Connecticut	PMR	8	1.09	0.55,2.18	white
Vena et al., 1985	New York	PMR	11	1.89	p<0.05	based on US mortality, white
			7	2.32	p<0.05	employed in engine plant > 20 years
Mallin et al., 1986	Illinois	PMR	5	1.19	NS	white
			5	3.57	p<0.05	black
Population-Based Studies						
Mack and Paganini, 1981	Los Angeles	Incidence	21	1.30	NA	machinists, white males

*doesn't report regression MOR. models

Table 4.1-5, Results for Laryngeal Cancer from Epidemiologic Studies of MWF-exposed Populations

Author	Location	Type of Study/ Analysis	# with CA or # Exposed Cases	Risk Estimate	95% CI (or p-value)	Study Population/Cancer Site
Cohort Studies						
Tolbert et al., 1992	Michigan	SMR	23	1.98	1.28,2.98	ever straight oil exposure, white
			30	1.41	0.95,2.01	ever soluble oil exposure, white
			8	1.57	0.68,3.09	ever synthetic oil exposure, white
			1	0.50	0.01,2.78	ever straight oil exposure, black
			64	0.91	0.70,1.17	ever soluble oil exposure, black
Eisen et al., 1992	Michigan	SMR	2	0.77	0.09,2.79	white autoworkers, Plant III
Eisen et al., 1994	Michigan	nested case/control	28	2.23	1.25,3.98	highest exposure to MWFs
Proportionate Mortality Studies						
Vena et al., 1985	New York	PMR	3	1.81	NS	based on US mortality, white
Mallin et al., 1986	Illinois	PMR	7	1.79	NS	white
Population-Based Studies						
Zagraniski et al., 1986	Connecticut	case/control	22	2.5	1.2,5.2	ever worked as a machinist
			17	2.1	1.0,4.7	ever worked as a metal grinder
Wortley et al.,1992	Washington State	case/control	NA	1.8	0.5,6.2	ever employed as grinding, abrading, or buffing operator
			19	1.0	0.5,1.9	ever employed in precision metal working
Haguenoer et al., 1990	France	case/control	7	1.8	NS	employed in metal work or as mechanic for at least 15 years
Brown et al.,1988	Texas	case/control	5	0.53	0.18,1.58	ever machinists
Ahrens et al., 1991	Germany	case/control	NA	2.2	0.9,5.3	ever mineral oil exposure

Table 4.1-6, Results for Rectal Cancer from Epidemiologic Studies of MWF-exposed Populations

Author	Location	Type of Study/ Analysis	# with CA or # Exposed Cases	Risk Estimate	95% CI (or p-value)	Study Population/Cancer Site
Cohort Studies						
Tolbert et al., 1992	Michigan	SMR	37	1.47	1.04,2.03	ever straight oil exposure, white
			51	1.09	0.81,1.43	ever soluble oil exposure, white
			9	0.92	0.42,1.74	ever synthetic oil exposure, white
			1	0.45	0.01,2.52	ever straight oil exposure, black
			3	0.68	0.14,1.99	ever soluble oil exposure, black
Eisen et al., 1992	Michigan	SMR	7	1.70	0.68,3.50	white autoworkers, Plant III
Decoufle, 1978	Michigan	SMR	8	1.25	NS	white
			4	1.29	NS	white, 5+ years of heavy oil mist exposure
Proportionate Mortality Studies						
Silverstein et al., 1988	Connecticut	PMR	14	1.36	0.81,2.29	white
Park et al., 1988	Connecticut	PMR	11	3.07	1.54,5.50	white
Vena et al., 1985	New York	PMR	4	1.38	NS	based on US mortality, white
			4	2.76	p<0.05	employed in engine plant > 20 years
Mallin et al., 1986	Illinois	PMR	2	0.80	NS	white
Population-Based Studies						
Gerhardsson de Verdier et al., 1992	Sweden	case/control	25	2.0	1.0,4.2	ever exposed to cutting oils
Siemiatycki et al., 1987	Montreal	case/control	13	0.7	90%CI=0.4,1.0	ever exposed to cutting oils

Table 4.1-7, Results for Bladder and Urinary Organ Cancer from Epidemiologic Studies of MWF-exposed Populations

Author	Location	Type of Study/ Analysis	# with CA or # Exposed Cases	Risk Estimate	95% CI (or p-value)	Study Population/Cancer Site
Cohort Studies						
Decoufle, 1978	Michigan	SMR	6	1.2	NS	white/bladder & other urinary organs
			2	0.8	NS	white, 5 + years heavy oil mist exposure / bladder & other urinary organs
Jarvholm & Lavenius,1987	Sweden	SIR	7	1.04	0.4,2.2	grinders & turners
Proportionate Mortality Studies						
Silverstein et al., 1988	Connecticut	PMR	14	1.26	0.75,2.19	white
Park et al., 1988	Connecticut	PMR	1	0.24	0.01,1.31	white
Vena et al., 1985	New York	PMR	7	2.28	p<0.05	based on US mortality, white
			4	2.76	NS	employed in engine plant > 20 years
Mallin et al., 1986	Illinois	PMR	2	0.78	NS	white
Population-Based Studies						
Silverman et al., 1989	US	case/control	102	1.3	1.0,1.7	ever machinist 6 months or more
			51	1.4	0.9,2.1	ever drill press operator ≥6 months
Siemiatycki et al., 1987	Montreal	case/control	47	1.2	90%CI=1.0,1.6	ever exposed to cutting oils
Claude et al.,1988	Germany	case/control	18	2.25	1.0,5.6	ever turner
			43	0.84	0.54,1.3	ever metal worker
Gonzalez et al., 1989	Spain	case/control	21	0.77	0.5,1.1	ever toolmaker ≥6 months
			NA	1.86	1.2,2.8	ever machinery adjuster, assembler or mechanic ≥6 months
Steenland, 1987	Ohio	case/control	11	2.00	NS	ever grinding machine operator
			45	0.69	p<0.05	ever machinist
Vinois & Magnani,1985	Italy	case/control	16	1.5	0.7,3.3	ever employed in machine tools ≥6 months
Schifflers et al.,1987	Belgium	case/control	34	2.45	1.28,4.69	all metal workers
			8	2.57	0.92,7.16	turners
Howe & Lindsay, 1980	Canada	case/control	NA	2.7	1.1,7.7	ever metal machinist
Coggon et al., 1984	Britain	case/control	52	1.3	0.9,1.9	ever had an occupation with potential cutting oil exposure
			21	1.5	0.8,2.8	ever had an occupation with potential high cutting oil exposure

Table 4.1-11, Results for Lung/Respiratory System Cancer from Epidemiologic Studies of MWF-exposed Populations

Author	Location	Type of Study/ Analysis	# with CA or # Exposed Cases	Risk Estimate	95% CI (or p-value)	Study Population/Cancer Site or Cell Type
Cohort Studies						
Tolbert et al., 1992	Michigan	SMR	251	1.02	0.90,1.15	ever straight oil exposure, white
			478	1.07	0.97,1.17	ever soluble oil exposure, white
			116	1.01	0.82,1.21	ever synthetic oil exposure, white
			35	1.06	0.74,1.48	ever straight oil exposure, black
			64	0.91	0.70,1.17	ever soluble oil exposure, black
Eisen et al., 1992	Michigan	SMR	60	0.91	0.70,1.17	white autoworkers, Plant III
Rotimi et al., 1993	Ohio	SMR	81	1.20	0.95,1.40	engine plant, white
			23	1.35	0.86,2.02	engine plant, black
Acquavella et al., 1993	Iowa	SMR	42	1.3	0.9,1.8	total workforce
			18	2.2	1.3,3.4	factory workers employed >10 years, hired between 1950-1959
Jarvholm & Lavenius, 1987	Sweden	SIR	5	0.40	0.1,0.9	grinders and turners
			3	0.30	0.1,0.9	grinders and turners, at least 20 years since onset of exposure
Park et al., 1994	Ohio	SMR	15	1.25	0.73,2.2	stamping plant
		MOR	4	1.64	0.56,4.8	ever tool and die worker
Proportionate Mortality Studies						
Silverstein et al., 1988	Connecticut	PMR	83	0.92	0.76,1.13	white/both primary and secondary lung cancer
			13	0.62	NS	employed in grinding 10 or more years
Park et al., 1988	Connecticut	PMR	59	1.23	0.96, 1.57	white men/both primary and secondary lung cancer
		case/control	5	19.3	p=0.008	female, ever employed in grinding
Vena et al., 1985	New York	PMR	48	1.26	NS	based on US mortality, white
			29	1.40	NS	employed in engine plant > 20 years
Mallin et al., 1986	Illinois	PMR	34	1.27	NS	white
			12	1.29	NS	black
Population-Based Studies						
Siemiatycki et al., 1987	Montreal	case/control	23	1.5	90%CI=1.0,2.1	ever exposed to cutting oils/oat cell cancer of the lung
Jockel et al., 1992	Germany	case/control	NA	2.2	1.05,4.75	employed 6 months or more as a turner, grinder, driller or cutter.

Coggon et al., 1984	Britian	case/control	113	1.4	1.1,1.8	ever had an occupation with potential MWF exposure
			25	1.0	0.6,1.6	ever had an occupation with potential high MWF exposure

Table 4.1-12, Results for Colon Cancer from Epidemiologic Studies of MWF-exposed Populations

Author	Location	Type of Study/ Analysis	# with CA or # Exposed Cases	Risk Estimate	95% CI (or p-value)	Study Population/Cancer Site
Cohort Studies						
Tolbert et al., 1992	Michigan	SMR	59	0.79	0.61,1.03	ever straight oil exposure, white
			116	0.86	0.70,1.02	ever soluble oil exposure, white
			26	0.83	0.54,1.22	ever synthetic oil exposure, white
			3	0.42	0.08,1.22	ever straight oil exposure, black
			8	0.56	0.24,1.09	ever soluble oil exposure, black
Eisen et al., 1992	Michigan	SMR	22	1.47	0.92,2.22	white autoworkers, Plant III
Acquavella et al.,1993	Iowa	SMR	1	0.1	0,0.5	total workforce
Decoufle, 1978	Michigan	SMR	17	1.3	NS	white
			7	1.1	NS	white, 5+years of heavy oil mist exposure
Proportionate Mortality Studies						
Silverstein et al., 1988	Connecticut	PMR	41	1.39	1.03,1.88	white
			13	1.89	p=0.02	employed in grinding 10 or more years
Park et al., 1988	Connecticut	PMR	15	1.18	0.71,1.94	white
Vena et al., 1985	New York	PMR	14	1.49	NS	based on US mortality, white
			8	1.70	NS	employed in engine plant > 20 years
Mallin et al., 1986	Illinois	PMR	12	1.17	NS	white
			2	1.04	NS	black
Population-Based Studies						
Siemiatycki et al., 1987	Montreal	case/control	32	1.0	90%CI=0.8,1.4	ever exposed to cutting oils
Gerhardsson de Verdier et al., 1992	Sweden	case/control	25	1.5	0.8,2.8	ever exposed to cutting fluids

Table 4.1-13, Results for Hematopoietic and Lymphopoietic Cancer from Epidemiologic Studies of MWF-exposed Populations

Author	Location	Type of Study/ Analysis	# with CA or # Exposed Cases	Risk Estimate	95% CI (or p-value)	Study Population/Cancer Site
Cohort Studies						
Tolbert et al., 1992	Michigan	SMR	38	1.26	0.98,1.71	ever straight oil exposure, white/leukemia
			75	1.33	1.05,1.67	ever soluble oil exposure, white/leukemia
			16	1.22	0.70,1.98	ever synthetic oil exposure, white/leukemia
			2	0.55	0.[illegible],1.13	ever straight oil exposure, black/leukemia
			4	0.74	0.20,1.90	ever soluble oil exposure, black/leukemia
Eisen et al., 1992	Michigan	SMR	9	1.07	0.49,2.02	white autoworkers, Plant III/leukemia
Decoufle, 1978	Michigan	SMR	3	0.55	NS	white/leukemia
			2	0.76	NS	white, 5+years of heavy oil mist exposure /leukemia
Park et al. 1988	Ohio	MOR	3	5.36	1.6,19	ever tool and die worker
Proportionate Mortality Studies						
Silverstein et al., 1988	Connecticut	PMR	27	1.03	0.71,1.60	white/all lymphopoietic cancer
			12	1.10	0.63, 1.94	White/leukemia
			5	0.84	NS	employed in grinding 10 or more years/all lymphopoietic cancer
Park et al., 1988	Connecticut	PMR	7	0.60	0.29, 1.23	white/all lymphopoietic cancer
			1	0.23	0.01, 1.26	white/leukemia
Vena et al., 1985	New York	PMR	8	0.86	NS	based on US mortality, white/lymphatic & hematopoietic
			4	0.85	NS	employed in engine plant > 20 years/lymphatic & hematopoietic
Mallin et al., 1986	Illinois	PMR	8	1.19	NS	white/lymphatic & hematopoietic
			3	1.36	NS	white/Non-Hodgkins lymphoma
			1	0.38	NS	white/leukemia
			6	3.54	$P<0.01$	black//lymphatic & hematopoietic
			3	6.87	$p<0.05$	black/Non-Hodgkin's lymphoma
			1	- nos. too small to calculate		black/leukemia
Population-Based Studies						
Siemiatycki et al., 1987	Montreal	case/control	22	1.3	90%CI=0.9,1.8	ever exposed to cutting oils/Non-Hodgkin's lymphoma

4.2.1.3 Legionellosis

A large outbreak of Pontiac Fever, a self-limited, nonpneumonic form of legionellosis with influenza-like symptoms, was shown to be caused by exposure to contaminated MWF aerosol in an engine ~~assembly~~ manufacturing plant [Herwaldt et al. 1984]. The outbreak occurred on startup following an 8-day shutdown that had allowed bacterial growth in the MWF reservoir. A newly identified species of *Legionella* was isolated from this soluble MWF. Compared with controls, workers with symptoms meeting the case definition criteria had significantly elevated antibody titer to this organism ($P<0.0001$). To date, no cases of legionnaire's disease (the sometimes fatal pneumonic form of legionellosis) have been associated with exposure to contaminated MWF, and no other outbreaks of MWF-associated nonpneumonic legionellosis have been reported in the scientific literature.

4.2.1.4 Hypersensitivity pneumonitis

?

Hypersensitivity pneumonitis (HP), also known as allergic alveolitis, involves an ~~auto~~immunologic reaction to inhaled antigen and requires prior sensitization to the antigen. This disease is characterized in its acute phase by alveolar inflammation and influenza-like symptoms and in its chronic phase (following repeated exposures) by pulmonary fibrosis associated with respiratory impairment. Common antigens associated with HP in non-MW occupational settings include airborne microbes (especially fungal spores such as *Alternaria* and *Aspergillus*) and various large molecular weight compounds, including proteins. Two cases of HP associated with MWFs were reported during a 3-yr period to an occupational respiratory disease surveillance program operating in the United Kingdom [Merideth and McDonald 1994]. The Michigan SENSOR program for occupational asthma surveillance documented several HP outbreaks involving large numbers of workers exposed to MWF aerosols [Rosenman et al., 1994]. Classes of MWFs, implicated MWF components, and contaminants used in these facilities were not identified. During 1995, NIOSH initiated two HHE investigations triggered by case reports of apparent HP occurring among MWF-exposed workers. These NIOSH investigations, HETA 94-0325 and HETA 95-0172, are currently in progress. Bernstein et al. [1995] published the first reported cases of HP associated with occupational exposure to MWF. The small MW shop introduced a water-based (synthetic) MWF in 1991; 6 to 11 months later, 6 workers developed HP symptoms. MWF sump samples were contaminated with bacteria and all six affected workers had precipitating antibodies to one of the bacterial contaminants, *Pseudomonas fluorescens*. Symptoms and other clinical abnormalities resolved in all six workers after they were removed from the workplace (and after additional corticosteroid treatment in two of the workers). On the basis of this investigation and analogy with other occupational settings in which HP is known to occur, microbes that contaminate MWFs would be likely etiologic suspects. However, it is possible that other agents such as MWF components or additives may also induce symptoms of HP. In addition to the *Pseudomonas* reported by Bernstein et al.[1995], several other genera of

HP - emphasize new epidemic,
unknown distinction

bacteria, including acid-fast bacteria and actinomycetes, and fungi are found in MWFs and have been associated with HP in other work environments.

4.2.2 Discussion

These four diseases of the lung parenchyma (lipid pneumonia, hard metal disease, legionellosis, and hypersensitivity pneumonitis) appear to be relatively unusual in workers exposed to MWF aerosols, though the large numbers of workers exposed justify considerable concern [Blanc 1995]. Prevention depends on reducing and eliminating worker exposures to the causative agents. In the case of lipid pneumonia, no reliable quantitative, exposure-response data are available, and the apparent rarity of the disorder among MWF-exposed workers suggests that current exposure concentrations are generally not associated with the disease. Prevention of hard metal disease depends largely on controlling exposures to cobalt-containing aerosol, to below the current NIOSH recommended exposure limit (REL) of 50 $\mu g/m^3$ [NIOSH 1988]. The prevention of contamination of MWFs by *Legionella* species would eliminate Legionellosis associated with occupational exposure to MWF. The etiologic agent(s) for HP is unknown, but in general, reduced exposures to MWF aerosols, contol of microbial growth, or reformulation of MWF to eliminate specific components (if identified as causitive agent(s)) may be necessary.

4.2.3 Asthma and Other Disorders of the Pulmonary Airways

4.2.3.1 General background

Recent concerns about the respiratory hazards of occupational exposure to MWF aerosols have focused on airways disorders. A variety of components, additives, or contaminants of MWFs are sensitizers or irritants known to induce new-onset asthma, aggravate pre-existing asthma, and irritate the airways of non-asthmatic individuals. These sensitizers, irritants, or toxicants include ethanolamine and other amines, colophony, pine oil, metals and metallic salts (e.g., chromium, nickel, cobalt, and tungsten carbide), castor oil, formaldehyde, chlorine, various acids, fungal and other microbial contaminants (including Gram-negative bacterial endotoxin).

tall oil & derivatives

Symptoms of airways irritation (e.g., cough) occur with sufficient exposure to airborne irritants. In addition to symptoms, the acute airways response to an irritant often involves short-term, apparently reversible decrements in measured pulmonary function. Repeated exposure to an irritant can evolve into chronic bronchitis, a condition characterized by chronic production of phlegm from the bronchus. Chronic airways irritation may also result in accelerated decline in lung function, which can ultimately result in symptomatic functional impairment and pulmonary disability.

reacted to volatiles from stirred (not nebulized) soluble MWFs, to volatiles from the pine oil reodorant contained in the soluble MWF, and to colophony (an agent known to induce occupational asthma), which is a component of the emulsifier used in the soluble MWF. Further challenges with other constituents of the soluble MWF failed to identify any other specific agent(s) responsible for the asthmatic reaction in this individual.

On the basis of data from a physician reporting system for occupational asthma in the West Midlands Region of England, Gannon and Burge [1991] reported that MWF aerosols and machine tool operators were among the four most frequently implicated agents and occupations, respectively. They also estimated an annual incidence for occupational asthma of 36 per million among metal and electrical manufacturing and repair workers, compared with a rate of less than 12 per million in professional and clerical workers (suggesting a 3-fold or greater excess risk).

An occupational respiratory disease surveillance program operating in the United Kingdom has provided additional evidence regarding the incidence of work-related asthma associated with MWF aerosols. In 1989, 7 cases of occupational asthma were attributed to MWF exposure, and the incidence of reported occupational asthma was approximately 250 per million in the "metal making and treating" occupational group compared with less than 10 per million among the "professional, managerial, clerical, and selling occupational" group [Meredith et al. 1991]. By the end of 1991, a total of 22 cases of MWF-associated occupational asthma were reported in that program [Merideth and McDonald 1994].

An occupational asthma surveillance program in Michigan (SENSOR) also provides evidence regarding asthma resulting from exposure to MWF aerosol [Rosenman et al. 1995]. MWFs were the second most common cause of work-related asthma in Michigan, accounting for 13% of the 725 cases reported in the 1988-1994 period. The cases worked at 54 different facilities, and the majority were employed in metal parts manufacture. Seventy-five of the cases were employed in the automobile parts manufacturing industry. The Michigan Department of Public Health interviewed 661 co-workers of reported cases at 35 of the facilities where MWFs were used, and found that 22% of the interviewed coworkers had developed since hire, new asthma or new symptoms consistent with occupational asthma (work-associated, daily or weekly shortness of breath, wheezing, or chest tightness). The rate was 19% of 345 coworkers at 19 facilities where measured MWF aerosol concentrations were below the current OSHA permissible exposure limit (PEL) of 5 mg/m^3 (measured exposure range: non-detectable to 3.57 mg/m^3) to and 25% of 308 coworkers at 15 other facilities where no air sampling was conducted. No new-onset asthma or symptoms consistent with occupational asthma were reported by the eight workers Coworkers? interviewed at the one facility where air sampling data indicated that exposures exceeded the current OSHA PEL of 5.0 mg/m^3.

SENSOR: should qualify with a) response rates among coworker surveys; b) crosssectional assessments

Rosenman et al. [1995]also found that new-onset asthma or symptoms suggestive of work-related asthma were reported by 12% (13/105) of those exposed to straight MWFs in 7 facilities, 20% (20/102) of those exposed to soluble MWFs in 7 facilities, 25% (100/393) of those exposed to synthetic MWFs in 10 facilities, and 29% (4/14) of those exposed to semi-synthetic MWFs in 2 facilities. Measures of association calculated from this data include odds ratios of 1.7 (95% CI: 0.8-4.0) for soluble MWFs and 2.4 (95% CI: 1.3-4.9) for synthetic/semi-synthetic MWFs, relative to straight MWFs.

An additional 8 cases of occupational asthma associated with exposure to MWFs have been reported in New Jersey and Massachusetts, the only other states which have had similar occupational asthma surveillance programs under development over the same period [SENSOR: unpublished observations; Reilly et al. 1994]. Also, 6 newly diagnosed cases of occupational asthma attributed to "cutting oils" were voluntarily reported by several occupational medicine clinics to the "Occupational and Environmental Disease Surveillance Database" between 1991-1993 [Hunting et al. 1995].

4.2.2.3 Asthma-Research findings

Ameille et al. [1995] evaluated self-reported responses to the question "Have you ever had asthma?" of workers at a gear-box machining shop with at least one year of MWF exposure. Three currently exposed and one unexposed groups were identified: 40 workers with exposure only to straight MWFs; 51 with exposure only to soluble MWFs; 139 with mixed exposure to soluble and straight MWFs; and the unexposed group of 78 assembly workers. The four groups were similar with respect to smoking habits. Currently exposed workers tended to be less likely to report asthma than assembly workers. The arithmetic mean exposure (measured as airborne concentration of oil mist using a solvent extraction procedure) was 2.6 mg/m^3 (SD 1.8; GM 2.2, GSD 1.9), in areas using straight MWFs. No sampling was done in areas using soluble MWFs. Based on data provided by Ameille et al. [1995], calculated asthma odds ratios (ORs) were 0.9 (0.26-3.34) for current exposure to straight MWFs and 0.8 (0.24-3.13) for current exposure to soluble MWFs, in comparison to the nonexposed assembly workers. The authors suggested that affected workers may have self-selected away from exposure, particularly those currently exposed to soluble MWFs.

Not
Published

In a preliminary analysis of a major cross-sectional respiratory morbidity study of 1811 workers at three automobile parts manufacturing facilities, Monson et al. [1993] found a decreased odds ratio for prevalence of self-reported physician-diagnosed asthma among grinders exposed to synthetic MWF. The mean exposure for this group was 0.48 mg/m^3 (thoracic fraction). The authors suggested that this finding could most likely be explained by the exposure actually causing asthma with subsequent removal of affected workers, or by self-removal of asthmatic workers intolerant of synthetic MWF aerosols. In reevaluating these same data, Eisen [1994] described an inverse exposure-response relationship between the synthetic MWF aerosol exposure concentration and prevalence of self-reported physician-diagnosed asthma. Excluding asthma cases who had developed the disease before employment as a machinist, and using an analysis designed to control for transfer bias, Eisen demonstrated that the incident asthma cases were over twice as likely as the nonasthmatic machinists to have been exposed to synthetic MWF aerosol in the year of asthma onset. She also observed indications of selective transfer of incident asthma cases away from jobs with exposure to synthetic MWFs ($p<0.10$) [Eisen 1994; Eisen and Greaves 1995].

Greaves et al. [1995b] reported a comprehensive analysis of the data previously reported on by Monson et al. [1993] and Eisen [1994]. Although there was no clear relationship between self-reported physician-diagnosed asthma and current aerosol exposure concentrations of straight, soluble, or synthetic MWFs, this analysis suggests that cumulative exposure to soluble fluids was related to asthma among these workers. Controlling for age, race, smoking, plant, and grinding, past (cumulative) exposure to soluble MWF aerosol (thoracic fraction) was significantly associated with asthma 1.02 (OR=1.02 per mg/m^3-year; $p<0.05$), even though the OR for asthma was decreased among workers with current exposure to soluble MWFs (OR=0.6; p=0.06). These and related findings again suggest possible selective transfer of affected workers to jobs with less exposure. (In evaluating the significance of these results, it should be noted that an OR of 1.02 per mg/m^3-year exposure suggests a greater than 2-fold risk of developing occupational asthma over a 45-year working lifetime of exposure to 1 mg/m^3 MWF aerosol (thoracic fraction). This is likely to be an underestimate of effect, as only current workers were included in the study, and workers who left these three plants as a result of their asthma would not have been included in the study.

heavy truck and other

In a recent study of ~~automobile~~ transmission manufacturing, workers exposed to soluble and straight MWFs in a major machine shop complex, Kriebel et al. [1994] found evidence for an association between self-reported physician-diagnosed asthma and work as a machinist. After controlling for age, race, gender, and smoking, machinists exposed to soluble MWF reported asthma twice as often as non-machinists (OR=2.1; 95% CI: 0.9-4.6); those exposed to straight MWF also reported more asthma (OR=1.4), but this difference was not statistically significant. In an analysis stratified by whether or not the asthma diagnosis predated employment as a machinist, Kriebel et al. [1994] indicated that the association was stronger for asthma with onset following employment than for asthma predating employment as a machinist. At the time of the questionnaire survey, machinists at this facility were exposed to either straight MWF aerosol (arithmetic mean of inhalable or "total" fraction = 0.24 mg/m^3) or soluble MWF aerosol (arithmetic mean inhalable or "total" fraction = 0.22 mg/m^3). Aerosol measurements were made using samplers with a seven-hole cassette inlet face, selected so as to approximate collection efficiencies of the ACGIH/ISO size selective criteria [Kriebel et al. 1994].

machining workers

Robins et al. [1994] provided relevant data from a study of ~~machinists~~ exposed to aerosols of soluble MWF and unexposed assembly workers for an automotive transmission plant. Among workers who ~~did not~~ report ed not having pre-existing asthma, development of new self-reported physician-diagnosed asthma after employment was reported by 3 of 84 machinists and by 1 of 44 assembly workers. Also, seven other machinists experienced clinically significant cross-shift FEV_1 decrements of at least 12%, compared to two of 46 assembly workers. Combining these indices of health effect, Robins et al. [1995b] suspected occupational asthma in 10/84 machinists who reported not having asthma prior to hire, compared to 3/44 assembly workers (OR=1.9; 95% CI: 0.4-11.0). Personal machinists' exposures associated with cross-shift decrements of at least 12% ranged from 0.17 mg/m^3 to 0.82 mg/m^3, with a median of just over 0.5 mg/m^3 (thoracic particulate).

Ameille et al. [1995] found no significant differences in bronchial responsiveness between workers exposed to MWFs and control workers, or among subgroups of workers exposed to the different MWF classes. However, the authors noted that self-selection away from exposure may have biased their findings, as 2/51 (4%) of those exposed only to soluble MWFs over the previous five years had been excluded from methacholine testing due to impairment of baseline lung function, compared to 15/118 (13%) of those not exposed to soluble MWFs over the same time (p=0.08). In a prospective study of non-specific bronchial responsiveness, Kennedy et al. [1995b; 1995c] followed apprentices in metalworking and other trades over two years. Study subjects were non-asthmatic at the beginning of the study, and MWF exposures (total aerosol) in machine shops ranged from nondetectable to 3.65 mg/m^3 (mean 0.46 mg/m^3). Compared to all others, workers who experienced at least 1800 hours of exposure to MWFs were more likely to develop a marked increase in methacholine responsiveness over the period of observation ($p<0.05$). In an analysis of all study subjects, increased bronchial responsiveness was positively associated with exposure to MWF [Kennedy et al. 1995b] and with development of work-related wheezing and chest tightness, and was negatively associated with wearing respiratory protection at least some of the time ($p<0.05$) [Kennedy et al. 1995c].

4.2.3.4 Asthma-Summary

The studies summarized above provide substantial evidence of an elevated risk of asthma among workers exposed to MWF aerosol exposure concentrations currently found in large MW shops and well below the OSHA PEL of 5 mg/m^3 for mineral oil mists. **Table 4-1, Risk estimates for association between MWF exposure and asthma morbidity,** summarizes risk estimates for asthma morbidity derived from these studies. Some evidence suggests a tendency for affected workers to transfer away from jobs with exposure toMWF. Not accounting for bias associated with such transfer, which is likely to result in underestimated risk, the overall evidence is indicative of approximately two-fold asthma prevalence risk among machinists exposed to soluble or synthetic MWFs at average exposures of 0.2 to 1 mg/m^3 (thoracic fraction). These studies provide much less clear-cut evidence for an increased risk of asthma associated with exposure to straight MWF. Clinical asthma induced by MWFs appears to involve specific sensitizers in some cases, but various other agents acting through irritant mechanisms may cause a high proportion of MWF-associated asthma.

Table 4-1, Risk estimates for association between MWF exposure and asthma morbidity

Study	Design	Population	Fluid Class	Aerosol Exposure Concentration	# Cases / # Exposed	Risk Estimate (95% CI or p value)
Gannon and Burge [1991]	Surveillance	Metal and Electrical Workers	----	----	36/million	>3-fold (relative to professional/clerical workers)
Rosenman et al. [1995]	Cross-Sectional	Automobile Parts Manufacture	straight soluble synthetic/semi-synthetic	generally <4.0 mg/m^3 generally <4.0 mg/m^3 generally <4.0 mg/m^3	13/105 (12%) 20/102 (20%) 104/407 (26%) (suspect OA)	-- OR=1.7 (0.8-4.0) OR=2.4 (1.3-4.9) (relative to straight MWF exposure)
Ameille et al. [1995]	Cross-Sectional	Automobile Parts Manufacture	straight soluble	current mean: 2.6 mg/m^3 (SD 1.8)	10/179 (6%) 10/198 (5%) (groups overlap)	OR=0.9 (0.3-3.3) OR=0.8 (0.2-3.1) (relative to assembly);(suggests transfer)
Greaves et al. [1995 b]	Cross-Sectional	Automobile Parts Manufacture	straight soluble synthetic	current mean: 0.43 mg/m^3 (SD 0.3) 0.55 mg/m^3 (SD 0.2) 0.41 mg/m^3 (SD 0.1) (thoracic fraction)	21/364 (6%) 25/452 (6%) 13/226 (6%)	OR=1.0 (p > 0.10) OR=0.8 (p > 0.10) OR=0.8 (p > 0.10) (evidence of transfer)
				cumulative straight soluble synthetic		at 1 mg/m^3 (thoracic fraction) for 45 years, approximately: OR=0.6 (p > 0.10) OR=2.4 (p < 0.05) OR=2.4 (p > 0.10)
Kriebel et al. [1994]	Cross-Sectional	Automobile Parts Manufacture	straight soluble	current mean: 0.24 mg/m^3 (SD 0.3) 0.22 mg/m^3 (SD 0.3) (inhalable fraction) (7-hole sampler)	6/74 (8%) 17/142 (12%)	OR=1.4 OR=2.1 (0.9-4.6) (relative to assembly exposure)
Robins et al. [1994]	Cross-Sectional	Automobile Parts Manufacture	soluble	current mean: 0.44 mg/m^3 (thoracic fraction)	10/84 (12%) (suspect OA)	OR=1.9 (0.4-11.0) (relative to assembly workers)

OR=odds ratio; SD=standard deviation; OA=occupational asthma

Oxhoj et al. [1982] studied 385 machine shop workers exposed to straight, soluble, semi-synthetic, or synthetic MWFs in 27 different facilities. Measured oil aerosol concentrations in these facilities ranged from 0.1 to 2.0 mg/m^3 (median 0.35 mg/m^3). Among smokers, workers with oil aerosol exposures exceeding 0.1 mg/m^3 had significantly higher prevalences of chronic cough (32% vs. 18%; $p<0.05$) and chronic phlegm (25% vs. 11%; $p<0.05$) than workers with lower exposure to oil aerosol. No symptom differences were reported for MWF classes.

Ameille et al. [1995] evaluated chronic respiratory symptoms among workers of a French automobile manufacturing plant. All exposed workers in a gear-box machining shop had at least one year of exposure to MWFs. Based on exposure during the most recent five years, three exposed groups and one unexposed group were defined: 40 workers with exposure only to straight MWFs; 51 with exposure only to soluble MWFs; 139 with mixed exposure to soluble and straight MWFs; and 78 assembly workers. In areas using straight MWFs, the arithmetic mean exposure was 2.6 mg/m^3 (SD 1.8; GM 2.2, GSD 1.9). No sampling was done in areas using soluble MWFs. The four groups were similar with respect to smoking habits. Symptoms assessed were chronic cough, chronic expectoration, and dyspnea. Those currently exposed to straight MWFs had a significantly higher prevalence of chronic cough and/or chronic phlegm (25.7% vs. 16.3%; $p<0.05$), as well as a higher prevalence of dyspnea (5.0% vs. 2.3%). A statistically significant increased risk of chronic cough was observed with increasing duration of exposure to straight MWFs after controlling for smoking (trend test $p=0.03$). Adjusted for smoking, the OR for chronic cough among those with more than 15 years of exposure to straight MWFs was 2.2 (95% CI: 1.01-4.85) relative to unexposed assembly workers. Although no statistically significant respiratory symptom findings were reported for workers currently exposed to soluble MWF, point estimates for odds ratios calculated from data presented in the report do exceed one for both dyspnea (OR=1.2; 95% CI: 0.22-12.8) and for chronic cough and phlegm (OR=1.34; 95% CI: 0.66-2.84), and Ameille et al. [1995] suggest that affected workers may have self-selected away from jobs with exposure to soluble MWF.

(transmission, chassis + steering gear)

Greaves et al. [1993a; 1995b] reported results from a cross-sectional respiratory morbidity study of 1,811 automobile manufacturing workers exposed to three classes of MWF (straight, soluble, and synthetic) in three plants. Prevalences of respiratory symptoms in 1042 machining and grinding operators were compared with those among 769 assemblers, only 239 of whom had no history of MWF exposures. Mean current exposures to aerosol (thoracic fraction) from straight, soluble, and synthetic MWFs were 0.43 (SD 0.26), 0.55 (SD 0.17), and 0.41 (SD 0.08) mg/m^3, respectively. Logistic regression analyses were controlled for smoking, race, and age, plant, and whether or not the worker was employed in grinding operations.

Compared with all assembly workers, machinists more frequently reported all previously described respiratory symptoms, including usual cough (OR=1.39; $p<0.05$), usual phlegm (OR=1.55; $p<0.001$), symptoms of chronic bronchitis (OR=1.45, $p<0.05$), and wheezing on most respiratory symptom prevalence, and current exposure concentrations appear to be the major determinants of respiratory symptoms. The Greaves et al. [1995b] study represents the most comprehensive assessment to date of respiratory symptoms among workers exposed to MWF aerosols. Nevertheless, findings from the study remain somewhat limited by the cross-sectional nature of the study.

In another cross-sectional study, Kriebel et al. [1994] studied 216 ~~automobile~~ truck transmission manufacturing workers exposed to straight and soluble MWFs from individual machine sumps in one machine shop compared to 170 assembly workers. Average aerosol exposures (inhalable fraction) were 0.24 mg/m^3 (SD 0.27) among workers exposed to straight MWFs, 0.22 mg/m^3 (SD 0.26) among workers exposed to soluble MWFs, 0.08 mg/m^3 (SD 0.05) among assembly workers, and 0.03 mg/m^3 (SD 0.03) among classroom/office workers. After controlling for age, race, gender, and smoking, the investigators found that machinists exposed to straight MWFs reported cough almost three times more often (OR=2.9; 95% CI: 1.2-6.7) and sinus problems almost twice as often (OR=1.7; 95% CI:0.96-3.0) as the comparison workers. Also, machinists exposed to soluble MWFs were more likely than those exposed to straight MWFs to report an increase in eye, nose, and throat irritation over the course of the workday ($p<0.01$). In addition, machinists whose MWF sump had not been refilled during the three weeks prior to the symptom survey were more likely to report cough than those whose sump had been changed within the last 3 days (OR=5.6; 95% CI: 4.3-6.9).

not published

Robins et al. [1994] compared acute respiratory effects among 85 machinists exposed to soluble MWF in an automobile ~~parts~~ transmission manufacturing plant to those among 46 assemblers working in an area that was physically isolated from the machining operations. Machinists, who had a mean personal aerosol exposure of 0.44 mg/m^3 (thoracic fraction), were significantly more likely than assemblers to report symptoms of chronic bronchitis (OR=6.8; p=0.04) and wheezing with dyspnea (OR=4.9; p=0.03) [Robins et al. 1995a]. In addition, machinists were about 3 times more likely to develop at least one respiratory symptom (dry cough, cough with phlegm, wheezing, chest tightness or dyspnea) during their Monday shift than were assemblers (p=0.012).

4.2.3.6 Symptoms of airways disorders-Summary

With the exception of one early study [Ely 1970], epidemiological studies of respiratory symptoms present generally consistent and, in the case of the more recent studies, compelling evidence indicating that occupational exposure to MWF aerosols causes symptoms consistent with airways irritation, chronic bronchitis, and asthma. The evidence suggests that each class of MWFs (straight, soluble, synthetic) is capable of inducing respiratory symptoms at MWF aerosol exposure concentrations that are currently typical of large metalworking shops and well below the current OSHA PEL for mineral oil mist of 5 mg/m^3. As summarized in **Table 4-2, Risk estimates for association between MWF exposures and respiratory symptoms**, a roughly 2- to 7-fold increased risk for various respiratory symptoms has been observed in studies of

Greaves et al. [1993b; 1995a] studied pulmonary function of 1745 automobile parts manufacturing workers employed in machining and grinding operations. Machinists, 352 of whom were currently exposed to straight, 441 to soluble, and 226 to synthetic, were compared with 726 assembly workers, 239 of whom had never been exposed in MW operations. Current exposures to aerosols (thoracic fraction) of straight MWFs (mean concentration 0.43 mg/m^3; SD 0.26), soluble MWFs (mean 0.54 mg/m^3; SD 0.17), or synthetic MWFs (mean 0.41 mg/m^3; SD 0.08) were measured. The relationships between pulmonary function and both current and cumulative exposures were evaluated. Previously exposed assemblers were not included in analyses that included current exposures only. Multivariate analyses controlled for age, height, race, smoking, grinding operation, and plant.

In terms of unadjusted mean lung function, Greaves et al. [1995a] found that approximately 18 percent of workers ever-exposed to any MWFs had abnormal (i.e., <85% of predicted) FEV_1 values, approximately 25% in excess over the 14% abnormal rate among never-exposed assemblers ($p<0.10$). Mean percent predicted and residual (observed minus predicted) FEV_1 values were significantly ($p<0.05$) reduced for the three groups of metal workers who were ever-exposed to straights, soluble, and synthetic MWFs , but not for the group of never-exposed assembly workers.

trichotomized

Analyzing the data tricotomized by current aerosol exposure, Greaves et al. [1995a] found trends

Oxhoj et al. [1982] communicated the results of a Danish study of 385 machine shop workers exposed to straight, soluble, semisynthetic, or synthetic MWFs in 27 different facilities. Measured oil aerosol concentrations in these facilities ranged from 0.1 to 2.0 mg/m^3 (median 0.35 mg/m^3). Controlling for age, height, and smoking, an analysis of spirometry data from 295 exposed male workers revealed no significant differences between four worker subgroups based on current exposure to straight, soluble, semisynthetic, or synthetic MWF. The authors summarized their rather limited spirometry findings by concluding that "if the four kinds of exposure influence ventilatory lung function, they do it to approximately the same degree" [Oxhoj et al. 1982].

In a study based in a French automobile manufacturing plant, Ameille et al. [1995] found no significant differences in baseline percent predicted lung function between four exposure groups (straight, soluble, mixed straight and soluble, and control), which did not differ by smoking habits. However, mean spirometry parameters were generally lower for the group of workers exposed to straight MWFs (current mean total airborne oil concentration was 2.6 mg/m^3) compared to the other groups, and the authors concluded that the study size was too small to detect significant difference in FEV$_1$ between exposure groups. After controlling for pack-years, linear regression analysis indicated that current smokers had significantly decreasing trends in FEV$_1$, FEF$_{25-75}$, and maximal flow rates at 50% and 75% of exhaled forced vital capacity with increasing duration of exposure to straight MWFs. This finding suggests a synergistic relationship between smoking and straight MWF exposure. No similar effects were observed for workers exposed to soluble MWF.

of declining function with increasing exposure for both straight and soluble MWFs. At the highest current exposure categories (>0.54 mg/m^3 for straight MWF and >0.65 mg/m^3 for soluble MWF), mean percent predicted FEV$_1$ (not adjusted for smoking) was significantly reduced ($p<0.01$). The exposure-related trend was inverted in a similar analysis of current exposure to synthetic MWF, the lowest exposure category (<0.18 mg/m^3) having the lowest mean percent predicted FEV$_1$ ($p=0.06$). Individual quantitative concentration of current aerosol exposures resulted in negative coefficients (adjusted for smoking) for percent predicted FEV$_1$ and percent predicted FVC ($p<0.05$) and for their residuals (observed value minus predicted value) ($p<0.10$) in relation to straight MWF and to soluble MWF aerosols. Adjusted for age, height, race, smoking, plant, and grinding, the coefficients for FEV$_1$ (-197 ml per mg/m3) and FVC (-229 ml per mg/m3) residuals were marginally significant ($p=0.06$) when compared to current straight MWF aerosol exposure concentrations. Coefficients for soluble and for synthetic MWF aerosol exposures were also negative, although they did not achieve statistical significance.

Analyzing the data tricotomized by cumulative aerosol exposure concentrations, Greaves et al. [1995a] found trends of declining mean percent predicted lung function with increasing exposure to straight and to soluble MWFs. At the highest exposure tertile (>1.71 mg/m^3-years for straight MWF and >3.41 mg/m^3-years for soluble MWF), mean percent predicted FEV$_1$ (unadjusted for smoking) was significantly reduced ($p<0.001$). Prevalence rates of abnormal FEV_1 among subgroups with highest cumulative aerosol exposures were greater than among never-exposed assembly workers for exposure to straight MWF (20.4% vs. 14.2%; $p=0.06$; rate ratio=1.4) and for exposure to soluble MWF (21.9% vs. 14.2%; $p<0.01$; rate ratio=1.5). Also, FEV_1 residuals were negative and statistically significant for the highest exposure categories of both straight MWFs (-117 ml; $p<0.001$) and soluble MWFs (-139 ml; $p<0.001$). Similar to the findings in relation to current exposures, there was an inverse trend among workers ever-exposed to synthetic MWF; prevalences of abnormal FEV_1 were 19.8%, 17.1%, and 13.6% for the lowest to highest cumulative exposure groups, respectively. The lowest exposure group (<0.18 mg/m^3-years) had an abnormality prevalence rate ratio of 1.4 relative to the never-exposed assemblers ($p=0.07$), representing a 40% excess.

In multiple linear regression analysis considering both current and past exposures simultaneously, Greaves et al. [1995a] found that accelerated decline in FEV_1 was significantly related to past exposures to aerosols from straight (FEV_1 residual = -5 ml per mg/m3-year; $p<0.05$) and from synthetic MWFs (FEV_1 residual = -7 ml per mg/m3-year; $p<0.10$), but not to past exposures to soluble MWFs (FEV_1 residual = -1 ml per mg/m3-year; $p>0.10$) or to current exposures.

The results of the Greaves et al. [1995a] study show that adverse pulmonary function effects are associated with cumulative occupational exposures to straight and synthetic MWFs. Cumulative exposure appeared to be more important than current aerosol exposure concentrations in predicting pulmonary function in this population. Greaves et al. [1995a] suggest that the increased impairment associated with lower current or lower cumulative exposures to synthetic MWF in the categorical exposure analysis may reflect a tendency for selective transfer of affected workers from jobs with higher MWF airborne exposure to jobs with lower exposures. The investigators expressed caution with respect to the lack of clear evidence of adverse effects of exposure to soluble MWFs. Among other reasons for this caution, they pointed out that most of the exposed workers in this study had at sometime also been exposed to soluble MWFs and that very few of the workers exposed to soluble MWFs had not also been exposed to straight or synthetic MWFs.

Kriebel et al. [1994] studied lung function in ~~automobile~~ truck transmission manufacturing workers exposed to soluble and straight MWFs in one machine shop compared to assembly (and office/classroom) workers. Average inhalable aerosol exposures were 0.24 mg/m^3 (SD 0.27) among workers exposed to straight MWFs, 0.22 mg/m^3 (SD 0.26) among workers exposed to soluble MWFs, 0.08 mg/m^3 (SD 0.05) among assembly workers, and 0.03 mg/m^3 (SD 0.03) among classroom/office workers. After adjustment for age, race, sex, height, and smoking, Kriebel et al. [1994] observed a statistically significant ($p<0.05$) deficit in baseline FEV$_1$ of 115 ml. (approximately 3%) among those exposed to soluble MWF.

4.2.4.2 Cross-sectional studies of lung function-Summary

Results of these cross-sectional studies of lung function generally parallel those from studies of respiratory symptoms among workers exposed to MWF aerosols. As summarized in **Table 4-3, Risk extimates for association between MWF exposure and chronic lung effects,** the findings indicate that occupational exposure to MWF aerosols is associated with reduced pulmonary function. Although the observed reductions in pulmonary function may, in part, be acute and reversible, their stronger relationship with past than with current exposures indicates that they are likely to be substantially chronic and irreversible. The observed adverse lung function effects are attributable to straight, soluble, and synthetic MWFs at exposures currently typical of large MW shops and well below the OSHA PEL for mineral oil mist of 5 mg/m^3. Moreover, they occur in a dose-related manner (at least for exposure to straight and synthetic MWFs aerosols). The respiratory symptoms and pulmonary function findings reported here suggest a differential ability of the MWF classes to effect respiratory capacity responses and physiological changes. Some pulmonary function evidence suggests possible synergism between smoking and exposure to MWF aerosol in reduced lung function. And finally, although the actual degree of self-selection away from MWF exposure by affected individuals is not known, such a phenomenon would bias cross-sectional studies towards underestimating the effects of exposure.

5% were consistently higher among exposed workers for all three Mondays on which testing was done. As mentioned above in the section regarding asthma, 6 of 85 exposed workers demonstrated clinically significant ($\geq$19%) cross-shift FEV_1 decrements, compared to none among the 46 workers in the assembly group (one tailed $p=0.07$). Using three exposure categories nearly equivalent to those used by Kennedy et al. [1989], Robins et al. [1994, 1995a] found evidence of a dose-related risk of cross-shift FEV_1 decrement of 5% or greater on Mondays. Aggregating observations from all three Mondays of the study and assuming independence of observations on the same individual on different Mondays, data from their report permit calculations of unadjusted relative risks for across-shift decrements greater than 5% of 1.34 (95% CI: 0.76-2.36) for "medium" exposure (0.16-0.47 mg/m^3, thoracic fraction) and 1.98 (95% CI: 1.11-3.52) for "high" exposure (>0.47 mg/m^3), relative to "low" exposure (<0.1.6 mg/m^3). The median personal MWF aerosol exposures of machinists experiencing across-shift FEV_1 decrements $\geq$12% ranged from 0.17 to 0.80 mg/m^3 (median: 0.5 mg/m^3) [Robins et al.1994].

The presence of chronic symptoms, as well as development of respiratory symptoms on Monday, were each associated with larger cross-shift FEV_1 decrements on Monday. Based on multiple regression analyses which excluded all workers who reported that they currently had asthma (and other influential outliers on a model-by-model basis), Robins et al. [1994] found that magnitude of cross-shift FEV_1 decrement was consistently related to higher airborne bacteria exposure among current smokers with lower baseline FEV_1/FVC ratio. Robins et al. [1995 b] reported that 25% of smokers with evidence of pulmonary obstruction at baseline experienced a cross-shift decrement of FEV1 of at least 10%, compared to only 5% of other study subjects. After controlling for other factors related to pulmonary function decrement, he found an increasing trend in risk of a 10% or greater across-shift FEV_1 decrement with increasing levels of exposure to both MWF aerosol and airborne bacteria among obstructed smokers, but not among other study subjects. Model-derived odds ratios for obstructed smokers were 3.1 (95% CI: 0.9-10.3), 6.0 (95% CI: 2.3-15.8), and 8.8 (95%: CI: 2.7-28.8) for exposure to MWF aerosol concentrations at 0.14, 0.34, and 0.57 mg/m³ (thoracic fraction), respectively, compared with other workers exposed at 0.14 mg/m³ (OR=1, by designation). Corresponding odds ratios for other study subjects were 1.0 (95% CI: 0.6-1.6) and 1.0 (95% CI: 0.4-2.2) for the medium and high levels of exposure, respectively. Model-derived odds ratios for obstructed smokers were 4.4 (95% CI: 1.8-10.7), 6.1 (95% CI: 2.4-15.3), and 7.5 (95% CI: 2.6-22.1) for exposure to airborne bacteria concentrations at 0.2, 1.0, and 3.0 bacteria/cc (thoracic fraction). Corresponding odds ratios among other study subjects were 1.0 (by designation), 0.5 (95% CI: 0.4-1.1), and 0.5 (95% CI: 0.2-1.1). These findings suggest important interactive effects of smoking, baseline airways obstruction, and exposure.

unobstructed smokers + nonsmokers?

Kenyon et al. [1993] reported exposures to TEA, DEA, and MEA in the same automotive parts manufacturing plants studied by Eisen et al. [1992] and Woskie et al. [1994]. The results are provided from one plant which used insoluble, soluble, synthetic, and semisynthetic fluids. Personal samples were collected from all operations using synthetic, semisynthetic and some soluble MWFs. TEA in particulate mass samples and TEA, MEA, and DEA in bulk fluid samples were collected and analyzed by gas chromatography. TEA did not account for more than 1% of the particulate mass except when the MWF contained more than 10% TEA in the bulk formulation. All three ethanolamines were found in bulk samples of synthetic and semisynthetic fluids. TEA and MEA were found in soluble fluids. There were no detectable concentration(s) of ethanolamines in mineral oil and only low concentrations of ethanolamines in soluble fluids. Higher airborne TEA concentrations were found with transfer operations (large complex machines that perform several operations) compared to other machining operations. The authors concluded that although airborne TEA concentrations generally increase with increasing TEA percent in the bulk fluids, the concentration is also operation-specific.

In 1994, the National Toxicology Program (NTP) released a Board Draft on two chronic experimental studies in which Fischer 344/N rats and B6C3F1 mice were dermally exposed to concentrations of TEA in acetone for 103 weeks [NTP 1994]. A final report has not been released as of the public review date of this CD.

The NTP stated that there was "equivocal evidence" of carcinogenic activity in the TEA treated male rats and concluded that the lack of both a clear dose-response and an increase in the total number of proliferative renal lesions in dosed male rats raises doubt that this result could have been attributed to TEA administration. Since there was no significant terminal increase in tumors for female rats in the treatment or control groups, the NTP concluded that there was "no evidence" of carcinogenic activity induced in these TEA treated females [NTP 1994].

The NTP [1994] also reported the experimental results of a significant increase (P=0.03) in hepatocellular adenomas in high dose male mice when compared to the concurrent controls. No differences in incidence of hepatocellular adenomas were observed for either the of the two lower dose males. When the terminal incidences for hepatocellular adenomas and carcinomas, and hepatoblastomas were combined for the high dose males, they also became statistically significant (P=0.018). However, these male mice were found to be infected with *Heliobacter hepaticus*, the presence of which has been associated with increased incidences of hepatocellular neoplasms in male mice and this may be a confounding factor in the interpretation of carcinogenicity studies [Ward et al. 1994a]. This infection in male mice was a significant factor in the N.T.P.'s final determination of "equivocal evidence" of carcinogenic activity in treated male mice based on the possibility that the increased numbers of hepatocellular adenomas induced by the *Heliobacter* infection.

discussed the association of some mineral oils with rectal cancer [Tolbert et al. 1992; Park et al. 1988]. Experimental animal bioassays demonstrated that the skin tumorigenicity of different refinement classes of mineral oils is related to their polycyclic aromatic content [IARC 1984].

The International Agency for Research on Cancer (IARC) has classified untreated and mildly treated oils as Group 1 human carcinogens; the evidence for carcinogenicity to humans is sufficient for untreated and mildly-treated oils and inadequate for highly refined oils. Untreated and mildly-treated oils have also been classified as Group 2 animal carcinogens; the evidence for carcinogenicity to animals is sufficient for untreated and mildly-treated oils and inadequate for highly-refined oils [IARC 1987a]. The Occupational Safety and Health Administration (OSHA) Hazard Communication Standard (HCS) [FR 1985] requires that employers report on material safety data sheets that a substance is a carcinogen, or potential carcinogen when (1) OSHA has regulated the substance as a carcinogen; (2) the National Toxicology Program NTP) lists the substance on its annual list of carcinogens; or (3) IARC has evaluated the substance, and found sufficient or limited evidence of carcinogenicity. Based on the IARC process parameters of mild hydrotreatment, an oil processed at a hydrogen pressure of 800 psi or less, at temperatures up to 800°F is subject to the HCS. Generally, the degree of mineral oil refinement has not been reported in epidemiology studies. Worker exposure to untreated and mildly treated mineral oils should be reduced to the extent technologically feasible

WHAT ABOUT RECLAIMED/REPROCESSED/RECYCLED OILS ?

5.3 Biocides

Biocides are incorporated as components in the formulated MWFs or added before and during use to MWFs to prevent microbial growth. Biocides are classified into two groups: formaldehyde release agents containing condensates of formaldehyde, and others. Formaldehyde releasers are usually soluble in water rather than oil and are more effective against bacteria than fungi. Examples of commonly used formaldehyde-releasing biocides are presented in **Table 5-1, Formaldehyde release biocides**.

Table 5-1, Formaldehyde release biocides

Trade name	Chemical name
Tris Nitro	Tris(hydroxymethyl)nitromethane
Onyxide 200	Hexahydro-1,3,5-tris(hydroxyethyl)-S-triazine
Grotan-Bk	Hexahydro-1,3,5, tris(2-hydroxyethyl) triazine
Vancide TH	Hexahydro-1,3,5-triethyl-S-triazine
Dowicil 75	1-(3-chloroallyl)-3,5,7-triaza-1-azonia-adamantane chloride
Bioban P-1487	4-(2-nitrobutyl)morpholine 4,4'-(2-ethyl-2-nitrotramethylene) Dimorpholine
Sodium Omadine	Sodium 2-pyridinethiol-1-oxide
Proxel CRL 1,2-BIT	1,2-Benzisothiazolin-3-one
Kathon 886	5-Chloro-2-methyl-4-isothiazolin-3-one 2-Methyl-4-isothiazolin-3-one
Dowicide-1	o-phenyl phenol

Staphylococcus aureus), *Escherichia coli, Proteus vulgaris, Aerobacter* (now *Enterobacter*) *aerogenes*, and members of the *Citrobacter* and *Achromobacter* genera [Tant and Bennett, 1956]. In a later study, Bennett [1972] again identified *Pseudomonas* and *Desulfavibrio* as the two most common genus isolated. Wort, et al. [1976] examined samples of soluble oil emulsions and also reported that *Pseudomonas* was the predominant genus isolated. *Cephalosporium* (*Acremonium*) was identified as the most common fungus isolated [Bennett, 1972]. Rossmore et al. found *Pseudomonas, Enterobacter, Moraxella, Aeromonas, Acinetobacter, Flavobacterium*, and *Alcaligenes* and, also, the fungi *Cephalosporium, Fusarium, Penicillium, Aspergillus, Cladosporium, Trichoderma, Candida, Botrytis, Saccharomyces, Trichosporon*, and *Cryptococcus* in MWFs from an automotive engine plant [Rossmore et al, 1987]. However, generalizations about bacterial contamination of all MWF classes cannot be made.

NOT IN REFERENCES

6.2.2 Potential Hazards

Although frankly pathogenic organisms, such as *Salmonella, Staphylococcus*, and *Legionella* have been isolated from MWFs, [Hill and Al-Zubaidy 1979; Herwaldt et al. 1984], most of the organisms associated with MWFs are characterized as either non-pathogens or "opportunistic" pathogens--those that primarily affect individuals with a major abnormality in their natural defenses. Conditions and situations that may result in compromised host defenses include: predisposing disease, such as diabetes, cancer (especially leukemia), or cystic fibrosis; alcoholism;

inherited or acquired immune deficiency; burns or other trauma; invasive medical procedures; and certain medications (e.g., some antibiotics; immunosuppressive drugs taken by organ transplant recipients).

The most common bacterial genus isolated from MWFs is *Pseudomonas*. Despite the frequency and severity of *Pseudomonas* infections in the susceptible host, healthy adults with intact immunity are rarely affected. One study of a worksite with a demonstrated viable count of 1 x 10^8 colony forming units (CFU) per ml of MWF showed no evidence of colonization of the respiratory tracts of the workers by *Pseudomonas*, even though the organisms were cultured from the MWF [Hill and Al-Zubaidy 1979], probably because the organisms are rapidly cleared from the lungs of healthy individuals. There have been no published reports of work-related *Pseudomonas* infections in MWF workers.

Infections are not the only health risks associated with occupational exposure to microorganisms. All microorganisms produce antigens -- molecules, often proteins or polysaccharide, that specifically stimulate the immune system. Exposure to an antigen may result in sensitization. If the sensitized individual is then re-exposed to the same antigen, there may be a hypersensitive or allergic response to an antigenic dose that would elicit little or no reaction from non-sensitized persons. Allergic reactions to inhaled antigens may be limited to the upper respiratory tract (e.g., allergic rhinitis), affect the airways (e.g., allergic asthma), or affect the distal portions of the lung (e.g., allergic alveolitis or HP). It is not entirely clear what role bacterial antigens play in MWF-related occupational effcts. Automotive workers and other workers exposed to MWFs were shown to have increased levels of antibodies to the *Pseudomonas* species found in the fluids, but no adverse health effects were noted [Mattsby-Baltzer et al. 1990]. Burge [1995] described an outbreak of HP possibly related to a "Gram-positive" bacterial antigen. A recent report by Bernstein et al. [1995] documented HP in machinists all of whom had serum antibodies to *Pseudomonas fluorescens*, which was inhaled from the MWF in use. The cause of HP in MWF exposed workers may not be limited to bacterial antigens (**4.2 Nonmalignant Respiratory Effects**). The role of microbial antigens in MWF related asthma is unknown..

Not in References

Microorganisms that contaminate MWFs also may produce toxins. Endotoxins (lipopolysaccharide) produced by Gram-negative bacteria have been found to contaminate MWFs [Mattsby-Baltzer et al. 1989], and aerosols of MWFs can contain endotoxin [Milton et al. 1990]. Endotoxins were first implicated in occupational disease in 1942 [Neal et al. 1942]. Subsequently, various animal, human and epidemiologic studies have established a link between exposure to airborne endotoxins and respiratory problems in a variety of workplace environments [Pernis et al. 1961; Cavagna et al. 1969; DeMaria and Burrell 1980; Snella MC 1981; Burrell et al. 1982; Brigham and Meyrick 1986; Castellan et al. 1987; Rylander et al. 1987; Jacobs, 1989; Burrell et al. 1990; Gordon 1991; Fogelmark et al. 1992; Rylander et al. 1994]. Animal exposure studies conducted by Gordan [1992] demonstrated that the endotoxin content of MWFs predicted respiratory toxicity in a guinea pig model of acute airways obstruction. Therefore, aerosolized endotoxins are suspect causative agents of occupationally related respiratory illnesses among workers exposed to MWF aerosols [Hill et al. 1979; Hill 1983; Kennedy et al. 1989; Mattsby-Baltzer et al. 1989; Milton 1992; Brosseau 1992; Gordon 1992; Gordon et al. 1992; Sprince et al. 1994]. As discussed in **4.2 Nonmalignant Respiratory Effects**, endotoxins may contribute to several of the respiratory effects found in MWF exposed workers (e.g., chronic bronchitis, abnormal cross-shift declines in pulmonary function, asthma, and other long term effects) [Castellan et al. 1987].

not References

Bacteria also secrete other toxins and extracellular enzymes that may present health hazards, although to date there is. is no evidence that exotoxins or other microbial enzymes have produced adverse health effects in MWF exposed workers. Theoretically, toxic metabolites and tissue-damaging enzymes may accumulate to concentrations that may constitute a threat to exposed workers. Other microbial metabolites may accumulate in MWFs or be evolved as gasses. Some of these include ammonia and hydrogen sulfide.

Fungi (yeasts and molds)also contaminate all water-based or water-contaminated MWFs. Generally the fungi isolated from MWFs are common saprophytic species, which live on decaying organic matter in the environment and are not usually the major microbial contaminant in MWFs.

However, given the opportunity, fungi may infect susceptible hosts, such as the immunocompromised individuals discussed earlier. *Cephalosporium*, a genus commonly isolated from MWFs, has reportedly caused HP, also known as extrinsic allergic alveolitis, in exposed individuals [Patterson et al. 1981]. *Penicillium* and *Aspergillus* species, likewise, have been implicated in HP and both are common contaminants of MWFs. In addition, several of the fungal species isolated from MWFs are know to cause allergic reactions, including allergic asthma, but the relationship between fungal contamination and occupational asthma associated with MWF exposures is uncertain at this time. There are no published reports of fungal-related diseases from contaminated MWF exposures.

Biocides are often used to control microbial growth in MWFs. They should be used to *prevent* growth and not for attempted remediation of fluids already contaminated, because disruption or killing of bacterial cells with biocidal agents can release large amounts of endotoxin and other microbial products. Currently, there is no feasible way to remove these substances. Careful fluid maintenance and prevention of microbial growth is the best approach for elimination or reduction of endotoxin or other hazardous biological substance build-up and preserving the MWFs quality and function.

[illegible]

7. Current Occupational Recommendations and Standards

In 1976, NIOSH published *Current Intelligence Bulletin 15: Nitrosamines in Cutting Fluids,* which identified the presence of potentially carcinogenic PAHs and nitrosamines in MWFs (**6. Potentially Hazardous Contaminants**), and recommended industrial hygiene practices to minimize dermal and respiratory exposure [NIOSH 1976]. OSHA has classified NDMA as a cancer-suspect agent [29 FR 1910.1016]; it is regulated as an occupational carcinogen. Exposure of workers to NDMA is controlled through the required use of engineering controls, work practices, and personal protective equipment, including respirators [29 CFR 1910.1003-1910.1016]. NIOSH has identified NDMA as a potential occupational carcinogen and recommends that occupational exposure to NDMA be limited to the lowest feasible concentration [NIOSH 1973]. The American Conference of Governmental Industrial Hygienists (The ACGIH) has designated NDMA with an A2 classification of "Suspected Human Carcinogen" [ACGIH 1993b].

Particularly in the past, petroleum-based mineral oils used in insoluble, soluble, and semisynthetic MWFs were be derived by limited refining by vacuum distillation or acid treatment, or severely- or mildly-treated by solvent-refinement or hydrotreatment [IARC 1987a]. As noted previously (5.2 Mineral Oil), the Occupational Safety and Health Administration (OSHA) Hazard Communication Standard (HCS) [FR 1985] requires that employers report on material safety

This 9. **Basis for a Recommended Exposure Limit (REL)** summarizes the studies used to develop the REL and other recommendations needed in a comprehensive occupational safety and health program. These studies provide the best available evidence of the association between adverse respiratory health effects and occupational exposure to MWFs.

9.1.1 Industry Trends

Major changes were introduced into the U.S. machine tool industry over the last several decades. The overall consumption of MWFs and specifically the use of synthetic MWFs increased as tool and cut speeds increased. Advances in automation enabled the machines to be partially enclosed which facilitated the application and use of local exhaust ventilation. During the 1970s and 1980s, many U.S. plants installed recirculating air cleaners, improved the recirculating air filtration systems, and renovated the factories. The improvements were prompted in some part by the ACGIH threshold limit value (TLV) of 5 mg/m^3 for mineral oil mist established in the 1960s, and its promulgation in 1970 by the Occupational Safety and Health Administration (OSHA) as a permissible exposure limit (PEL).

but cutting speeds, have increased

but poor maint + repair generators

The study by Hallock et al. [1994] describes the effectiveness these changes had in the automotive industry on reducing exposures to airborne MWFs. Airborne MWF concentrations were found to have significantly declined over a 30-year period (1958-1987) with an arithmetic

Workers in the MW industry who are exposed to MWFs have been reported to have increased risks of nonmalignant respiratory effects, cancer, and skin diseases. Although the reported epidemiologic studies do not represent workers employed in all types of MW operations, environments, and exposures, adequate evidence has been compiled to indicate that those exposed to MWFs are at risk of adverse health effects, which may vary according to the MWF class and length of exposure.

NIOSH primarily has used traditional, peer-reviewed, published articles to form conclusions about carcinogenic hazards from MWF exposures. In addition, NIOSH reviewed several recent, unpublished epidemiologic investigations of worker exposures to MWFs. These investigations were sponsored by the Occupational Health Advisory Board (OHAB) of the General Motors-International Union, United Automobile, Aerospace & Agricultural Implement Workers of America (GM-UAW) joint health and safety activities. OHAB is made up of 6-7 university scientists and solicits research proposals from academic institutions. Following peer-reviews, OHAB funds, monitors, and facilitates the progress of selected proposals. Final research reports are prepared by the investigators after non-binding, peer-review by OHAB. NIOSH included 3 such investigations [Kriebel et al, 1994; Greaves et al, 1995 a, b; and Robins et al, 1994], in addition to a traditionally peer-reviewed study by Kennedy et al. [1989], in its decision process for determining the need for a MWF recommended exposure limit (REL).

mean concentration of 5.42 mg/m^3 (total particulate) observed prior to 1970, and 1.82 mg/m^3 after 1980. The geometric mean for aerosol concentration after 1980 reported at the plants studied by Hallock is 0.56 mg/m^3.

too steep

Since 1987, these exposures in the automotive industry have continued to decline. In the most recent studies of automobile manufacturing workers exposed to straight, soluble, and synthetic MWFs, mean exposure concentrations in nongrinding operations were reported to be <1.0 mg/m^3 (total particulate) [Hallock et al. 1994; Kriebel et al, 1994; Greaves et al, 1995a,b; Robins et al, 1994]. Kriebel et al.[1994] reported mean exposure concentrations of 0.24 mg/m^3 (inhalable fraction or 'total') to aerosols of straight fluids and 0.22 mg/m^3 to soluble fluids. Similar concentrations were reported by Greaves et al. [1995 a,b] with mean concentrations ('thoracic' fraction) for several plant surveys ranging from 0.2-0.68 mg/m^3 (straight fluids), 0.35-0.65 mg/m^3 (soluble fluids), and 0.41 mg/m^3 for synthetic fluids. Likewise, Robins et al.[1994] reported exposure to soluble MWF aerosols for automotive parts manufacturing workers that ranged from 0.1 to 0.6 mg/m^3 (thoracic fraction). Hallock et al.[1994] reported that approximately two thirds of the total airborne particulate exposures in the three automotive industry plants studied was in the thoracic fraction of exposure.

The occupational exposure data compiled by NIOSH health hazard evaluation (HHE) program (1972-1993) also show that exposure to airborne MWFs has been generally decreasing over time.

These data indicate that the arithmetic mean personal exposure concentrations (total particulate) were 1.23 mg/m^3 (n=21 plants) in the 1970s, 0.57 mg/m^3 in the 1980s (n=15 plants), and increase to 1.0 mg/m^3 in the 1990's based on only two plants. The overall mean concentration for 38 plant-based HHEs was 0.96 mg/m^3.

This decline in airborne exposures has also been reported in the Integrated Management Information System (IMIS) of OSHA which compiles the environmental samples from OSHA inspectors. From 1979 to 1995 (**Table 9-1, OSHA-Integrated Management Information System (IMIS). Number of oil mist (MINERAL) Samples and % by year ranges** (1979-February 1995), OSHA has collected mineral oil mist exposure data for IMIS, which represent a substantial cross-section of industry. These exposure data demonstrate a steady decline in exposure concentrations from 1980 to the present. The arithmetic mean concentration for all samples collected during this time period was 0.92 mg/m^3 (total particulate); for the period 1989 to 1994, the arithmetic mean was 0.49 mg/m^3. The increasing percentage of samples with airborne concentrations below 0.5 mg/m^3 over time suggests that improvements in engineering controls and work practices have occurred. Airborne concentrations, prior to 1980, have declined from 37% of all samples with airborne concentrations less than 0.5 mg/m^3, to 42% of all samples at less than 0.5 mg/m^3 from 1980 to 1984, and to 73% after 1989.

But could also reflect
altered sampling plant selection

The review of the results from the studies of workers exposed to MWFs indicate an association between MWF exposure and the risk of developing cancer at several organ sites, including the stomach, pancreas, larynx, and rectum. Most of the mortality studies of MWF-exposed cohorts found that MWF-exposed workers had an elevated risk of cancer at each of these sites [Tolbert et al.1992 ; Eisen et al, 1992; Decoufle, 1978; Rotimi, 1993; Acquavella et al, 1993; Teta, 1988; Silverstein et al, 1988; Park, 1988; Vena et al, 1985], and several of these studies found significantly elevated risks for cancer at one or more of these sites [Tolbert et al, 1992; Eisen et al, 1992; Rotimi, 1993; Acquavella et al, 1993; Silverstein et al, 1988; Park, 1988; Vena et al, 1985; Mallin, 1986]. In addition, case-control studies found that MWF-exposed workers had elevated risks for cancer of the stomach [Park, 1988; Silverstein, 1988; Siemiatycki, 1987], bladder [Silverman et al., 1989; Siemiatycki et al., 1987; Claude et al., 1988; Steenland et al., 1987; Vineis and Magnani, 1985; Schifflers et al, 1987; Howe and Lindsay, 1980] and larynx [Eisen et al.1994; Wortley, 1992].

Findings from the largest study with the most statistical power [Tolbert et al, 1992] suggest that specific classes of MWFs are associated with cancer at certain sites. However, within these MWF classes, the specific formulations responsible for the elevated cancer risks remain to be determined. Straight oil exposure was found to be associated with an increased risk for laryngeal and rectal cancer [Tolbert et al, 1993]. Furthermore, those workers whose lifetime exposure to straight oils exceeded 0.5 mg/m^3 -years had a significantly elevated risk for laryngeal cancer

(OR=2.23, 95% CI=1.25, 3.98) [Eisen 1994]. In addition, synthetic fluid exposure was associated with an increased risk for pancreatic cancer [Tolbert et al. 1992]. The specific constituent(s), additive(s), and/or contaminant(s) of MWFs that are responsible for the elevated risk for the various site-specific cancers remains to be determined. Some of the components, additives, and contaminants found in certain MWFs are considered carcinogenic in humans and animals, including non-severely refined mineral oils, N-nitrosamines [IARC 1978], and PAHs [IARC 1983, 1987].

Information on important lifestyle factors, such as tobacco and alcohol consumption, are often not collected in mortality studies of workers. Smoking and alcohol are associated with some of the cancer sites observed to be associated with MWF exposure. The lack of association between lung cancer and MWF exposure in the majority of studies suggest that association between MWFs and specific cancer sites is not related to cigarette use. Laryngeal cancer, one of the cancers associated with straight MWFs, is associated with both cigarette and alcohol use in the general population. In the Tolbert et al.[1992] study which provides the strongest evidence for the association of straight MWF and laryngeal cancer; there was no association between straight MWF exposure and cirrhosis or lung cancer.

In summary, the substantial evidence suggests that MWF exposures prior to the mid-1970s are causally associated with several organ-specific types of cancer. It is unlikely these associations are due to non-MWF occupational exposures or to non-occupational factors. Over the last several decades, substantial changes have been made in the metalworking industry, including reductions in exposure concentrations, refinement technology to remove hazardous contaminants, changes in MWF formulations, and improved control of microbiologic contamination. Currently there is little epidemiologic data that examines the impact of these exposure changes on cancer risk. Because changes in MWF composition and reduced airborne MWF exposures may not be sufficient to eliminate the cancer risks associated with MWF exposures, the reductions in airborne MWF exposure recommended in this chapter are warranted.

9.2.2 Nonmalignant Respiratory Effects

Occupational exposure to MWF aerosols is associated with a variety of nonmalignant respiratory effects, including lipid pneumonia, asthma, acute airways irritation, chronic bronchitis, and impaired pulmonary function (**4.2 Nonmalignant Respiratory Effects**). Many thousands of metalworkers are exposed to MWF aerosols at concentrations now documented to have adverse effects on the respiratory system. The aggregate, potential respiratory health implications of occupational exposure to MWF aerosols is therefore very substantial.

Lipid pneumonia, hard metal disease, and hypersensitivity pneumonitis appear to be relatively unusual MW environments involving occupational exposure to MWF aerosols, although the large

not so unusual

numbers of workers exposed justify concern about these health effects [Blanc 1995]. Prevention relies on reduction and/or elimination of worker exposure to the causative agent(s). In the case of lipid pneumonia, the causative agent is respirable mineral oil. There are no reliable quantitative exposure-response data available, but the relative rarity of the disorder suggests that current airborne exposure concentrations are generally effective in preventing the disease. Prevention of hard metal disease largely depends upon controlling exposure to tungsten carbide cobalt-containing aerosol--to no more than the current REL of 50 μg/m³ [NIOSH 1988]. The prevention of Legionellosis associated with occupational exposure to MWF would be assured if contamination of MWFs by *Legionella* species were eliminated. Controlling microbial contamination of MWFs would also ~~may~~ prove effective in preventing a large proportion of HP associated with MWF aerosols, as would general reduction of MWF aerosol exposure concentrations.

.5 mg [illegible]

Not clear

Recent concerns regarding respiratory hazards of occupational exposure to MWF aerosols have focussed on airways disorders. A variety of components, additives, or contaminants of MWFs are sensitizers or irritants known to induce new-onset asthma, aggravate pre-existing asthma, and/or irritate airways of non-asthmatic individuals.

The results of many of the epidemiological studies of respiratory symptoms present compelling evidence that occupational exposure to MWF aerosols causes symptoms consistent with airways irritation, chronic bronchitis, and asthma. The evidence suggests that each class of MWF described may induce these symptoms at MWF aerosol exposure concentrations that are currently typical of large MW shops. Increased risks (ranging roughly from 2- to 7-fold) have been observed for various respiratory symptoms among occupationally-exposed machinists, and statistically-significant quantitative exposure-response relationships have been observed between respiratory symptoms and straight, soluble, and synthetic MWFs (**Table 4-2, Risk estimates for association between MWF exposures and respiratory symptoms**).

generally below .5 mg/m³.

The documented onset or worsening of many symptoms over a work-shift, as well as the apparent substantial symptomatic improvement when away from work in many affected workers [Greaves et al. 1995b], suggests opportunity (through reduction of MWF aerosol exposures) for reversing early effects of MWF-induced airways irritation and possibly also for reducing the frequency of asthma induction and/or severity of symptomatic episodes of asthma triggered by MWF aerosol exposure.

The existing literature provides substantial evidence for an elevated risk for asthma among workers exposed to MWF aerosol exposure concentrations well below the current PEL of 5 mg/m^3 for mineral oil mists. Clinical asthma induced by MWFs appears to involve specific sensitizers in some cases [Savonius et al. 1994; Hendy et al. 1985], but various other agents acting through irritant mechanisms may cause a high proportion of MWF-associated asthma. Some evidence indicates a tendency for effected workers to transfer away from jobs with high exposure to the causative MWF [Eisen 1994; Eisen and Greaves 1995, Greaves et al. 1995b]. Not accounting for bias associated with such transfer, which is likely to result in underestimated risk, the overall evidence from recent epidemiologic research is indicative of an approximate two-fold risk of asthma among machinists exposed to soluble or synthetic MWFs at average aerosol exposures of about 0.2 to 1 mg/m^3 (thoracic fraction) [Greaves et al. 1995b; Robins et al. 1995b; Kriebel et al 1994]. Exposure to straight MWF aerosol appears less likely than aerosol from soluble or synthetic to be associated with asthma (**Table 4-1, Risk estimates for association between MWF exposure and asthma morbidity**), but clear-cut occupational asthma related to aerosol from straight MWF has been observed [Robertson et al. 1988].

Three studies have evaluated acute (cross-shift) lung function decrements in workers exposed to MWF aerosols [Kennedy et al. 1989; Kreibel et al. 1994, 1995; Robins et al. 1994]. Each found that incidence of cross-shift FEV_1 decrement is associated with occupational exposure to MWF aerosol (**Table 4-4, Risk estimates for association between MWF exposure and cross-shift lung function**). The evidence indicates that straights, soluble, and synthetic MWFs cause acute reductions in ventilatory function at MWF aerosol exposure concentrations well below the current OSHA PEL for mineral oil mist [Kennedy et al. 1989]. These exposure-related acute airflow reductions are clearly attributable to MWF aerosol concentrations in excess of approximately 0.5 mg/m^3 (thoracic fraction)[Kennedy et al. 1989; Kreibel et al. 1994, 1995; Robins et al. 1994].

9.4 Technological Feasibility of Controlling Exposures to MWFs

The fourth consideration is that it is technologically feasible to control exposures to MWF aerosols to a concentration of 0.5 mg/m^3 (total particulate) for many MW operations. This is supported by the following observations: 1) the continued decrease in MWF exposure concentrations during the last several decades as indicated by the OSHA data set IMIS, NIOSH HHE reports, and environmental measurements reported in the scientific literature; 2) the increased automation of machining operations; and 3) the widespread implementation of engineering controls and adoption of good work practices. The most complete information available concerning the decrease in MWF exposures comes from the automotive manufacturing industry. Overall mean airborne MWF exposure concentrations (thoracic fraction) were found in most recent respiratory studies to be between 0.2 and 0.55 mg/m^3 suggesting that total particulate concentrations are approximately 0.3 to 0.8 mg/m^3 [Greaves et al. 1995 a,b; Robins et al. 1994; Kriebel et al. 1994]. A recent engineering study found that enclosed new machines had a median concentration of 0.21 mg/m^3, while older machines with retrofit enclosures had a median concentration of 0.44 mg/m^3, and without retrofit enclosures had a median concentration of 0.5 mg/m^3 [Hands, 1996].

Between 1990 and 1995, OSHA compliance data (**Table 3-3, OSHA-Integrated Management Information System (IMIS). Number of oil mist (mineral) samples and % by year ranges**

(February 1979-February 1995)) collected from a wide variety of industries, showed that 73% of the airborne concentrations for MWFs (oil mist) were less than 0.5 mg/m^3 and 78% of the concentrations were < 1.0 mg/m^3. These data indicate that MWF exposures are presently being controlled to below 0.5 mg/m^3 in many workplaces. However, not all work places may be able to control exposures to MWFs to 0.5 mg/m^3 for all operations. While the epidemiologic studies, the OSHA IMIS data set, and the HHEs provide some insight into the current distribution of MWF airborne exposure concentrations, there is less data on the feasibility of reducing existing mean exposures above 0.5 mg/m^3 (total particulate) to lower airborne concentrations. Worker exposures at non-automated MW operations may be more difficult to control. Many of non-automated machines involve worker-machine interactions-- thus placing the worker in close proximity to the MWF aerosol generation (i.e., arms length), and at risk for dermal exposure. If it is not possible to isolate the worker from the aerosol, it may be difficult to use local ventilation or enclosures to reduce the worker's exposure to the freshly generated aerosol.

Newer automated MW machines do not require operators to stand in close proximity to aerosol generation. Thus, work stations can be positioned to minimize exposures. Mist generating operations can be effectively enclosed, and the air can be exhausted from these enclosures and processed in air cleaners to remove the mist. While there may be a trend of increased mist production at the cutting surface due to higher production rates, generally exposures can effectively be controlled by machine enclosure, ventilation, and air cleaning.

VAPOR

In summary, NIOSH proposes a recommended exposure limit (REL) of 0.5 mg/m^3 MWF aerosol (mean total particulate) as an 8-hr time-weighted-average. This recommendation is based primarily on the prevention of the diverse respiratory effects causally associated with MWF exposure, although a reduction in aerosol exposure is also a prudent step because of the association between past MWF exposures and cancer at several sites, as observed in the reviewed epidemiologic studies. Total particulate measurements of the MWF aerosol are a practical and adequate surrogate for specific agents in MWFs which cause these adverse health effects. It is technologically feasible to control many MW operation using MWFs to the level of the REL or lower.

Although the control of airborne MWFs to the 0.5 mg/m^3 REL will significantly reduce the risk of adverse health effects, a comprehensive safety and health program that includes provisions for worker education and training, worksite analysis, hazard prevention and control, and medical monitoring should be implemented to further minimize any health risks.

10. Recommendations for an Occupational Safety and Health Program

NIOSH has long recognized the value of comprehensive occupational safety and health programs to prevent occupational deaths, injuries, and illnesses. To be effective, a safety and health program needs to be developed and implemented as part of the employer's management system.

Such a program must have strong management commitment and worker involvement. The major elements for a comprehensive, and effective safety and health program should include (1) safety and health training, (2) worksite analysis, (3) hazard prevention and control, and (4) medical monitoring of exposed workers. These elements are described in the following sections.

10.1 Safety and Health Training

Employers should establish a training program for all workers with MWF exposures. One of the important goals of training is *to enable workers to identify potential workplace hazards.* Instruction should be provided when changes occur in job duties, a new job is assigned, and when new MWFs or potentially hazardous chemicals are introduced. Workers, as well as contract workers employed to maintain the facility, should be informed about any hazardous chemicals in the work areas and the availability of information in the material safety data sheets (MSDS), and other available information sources.

Workers should be trained how to detect the presence or potential release of hazardous chemicals (e.g., appearance of bacterial overgrowth and degradation of MWFs). Instruction should include information about how workers can protect themselves from potentially hazardous exposures (e.g., the use of appropriate work practices, emergency procedures, and personal protective equipment). Workers should be encouraged to maintain good housekeeping practices to help procedures outlined in *Industrial Ventilation--A Manual of Recommended Practices* [ACGIH 1988]. Selection of appropriate air cleaning equipment for MW operations exhaust that is recirculated to the work environment, is based on the concentration and size distribution of the exhaust stream. General guidelines for the recirculation of exhaust air can be found in *Industrial Ventilation--A Manual of Recommended Practices* [ACGIH 1988]. If exhaust air is vented outside the work environment, local air pollution authorities should be contacted regarding the relevant regulations.

In addition to local ventilation of machining operations, general ventilation systems inside plants, manufacturing or processing enclosures, or buildings may be used to control worker exposures to airborne aerosols, vapors, mists, and dust. General ventilation systems are designed to maintain either heated or cooled airflow throughout the plant or building, and airborne hazards are controlled by dilution and/or removed by exhaust. Air quality is maintained by designing a general ventilation system that minimizes air stagnation, prevents short circuiting of the fresh air supply to the exhaust, and directs clean air across the workers to carry airborne contaminants to the exhaust.

Ventilation
maintenance

10.4.5 Containment Enclosure

Machine enclosures are one of the effective methods of reducing worker exposures. Johnston [1995] has described the features which are important in designing effective enclosures. Hands [1995] examined exposure data collected at an automobile parts manufacturing plant for the effect of enclosure upon MWF exposures. This study suggests that enclosures (particularly original equipment manufacturer enclosures) are effective in reducing exposures. Retrofitting containment structures may also reduce exposures.

10.5 Protective Clothing and Equipment

Engineering controls are used to reduce the airborne concentration of MWFs. But in some situations, the added protection of chemical protective clothing (CPC) and equipment (e.g., respirators) should be provided in the event of excessive airborne exposure concentrations over the NIOSH REL of 0.5 mg/m^3 or dermal contact with the MWFs. The operator and maintenance staff may also need CPC because the nature of the work requires coming in contact with the MWFs during specific operations. All workers should be trained in the proper use and care of the CPC. After all CPC has been in routine use, it should be examined periodically along with the

Housekeeping;
no mopping into flumes;
no floor drainage to flumes.

10.6 Sanitation and Hygiene

Workers should be trained to keep personal items such as food, drink, cosmetics, and tobacco separate from the work environment. Smoking, eating, drinking, and applying make-up should be discouraged in MW areas to prevent unnecessary additional exposures to MWFs. A no-smoking policy should be established, since cigarette smoking may exacerbate the respiratory effects of MWFs [NIOSH 1994].

Instruction in personal hygiene will reduce potential dermal MWF exposures. Workers should be encouraged to and allowed the time during the workshift to clean exposed skin contaminated with MWFs. If onsite shower facilities are available, workers should be encouraged to shower and change into clean clothes at the end of the workshift. If not, workers should change from contaminated work clothes into street clothes prior to leaving work.

10.8 Labeling and Posting

Workers should be trained to be aware of labeling practices in accordance with the OSHA Hazard Communication standards [29 CFR 1910.1200 and 29 CFR 1926.59], they must be informed of chemical exposure hazards, of their potentially adverse health effects, and of the appropriate methods for self-protection. Labels and signs posted on or near hazardous MW processes provide an initial warning to other workers who may not routinely work near processes. Depending on the process and exposure concentration, warning signs should state a need to wear protective clothing or an appropriate respirator for regular exposure to MWF aerosol in excess of the REL. Warning signs may be needed at the worksite to inform transient nonproduction workers of hazards. All labels and warning signs should be printed in both English and the predominant language of workers who do not read English.

11. Medical Monitoring of Exposed Workers

Because asthma and other adverse nonmalignant respiratory health effects are associated with MWF aerosol exposure, employers should provide preplacement examinations and medical monitoring for all workers exposed to MWF aerosols. Employers should also provide appropriate evaluation and management of workers with signs or symptoms suggesting respiratory effects associated with MWF exposure.

Medical monitoring represents secondary prevention and should not supplant primary prevention efforts aimed at controlling MWF aerosol exposures. One objective of the recommended medical monitoring is to enable early identification of workers who develop symptoms of MWF-related conditions such as asthma and dermatitis. Those with asthmatic symptoms can have their

- Studies should be conducted to determine whether contaminants are concentrated or removed during the refining and recycling of used MWFs. Potential health risks of using recycled or reprocessed MWFs should be examined.

- Dermal exposures to MWFs and absorption through normal or damaged skin should be accurately assessed.

- Additional study is required to determine differential risk for adverse respiratory effects due to aerosol exposures for the 4 classes of MWFs.

- Address issues relating to transfer of workers within and out of jobs with a specific range of exposures to MWFs.

- Additional toxicological research is needed for mid-chain and long-chain chlorinated paraffins. naphthenics.

REFERENCES

ACGIH [1988]. Industrial Ventilation-A Manual of Recommended Practices. 20th ed. Cincinnati, OH: American Conference of Governmental Industrial Hygienists, Committee on Industrial Ventilation. American Conference of Governmental Industrial Hygienists, Standard for Mineral Oil Mist, 1994.

ACGIH [1994-1995]. Threshold limit values for chemical substanes and physical agents and biological exposure indices. ACGIH, Cincinnati, OH.

ACGIH [1993a]. Notice of Intended Change -- Oil Mist, Mineral. Appl Occup Environ Hyg 8(5):494-501.

ACGIH [1993b]. 1993-1994 Threshold limit values for Chemical Substances and Physical Agents and Biological Exposure Indices, Cincinnati, OH: American Conference of Governmental Industrial Hygienists.

Acquavella J, Leet T, Johnson G [1993]. Occupational experience and mortality among a cohort of metal components manufacturing workers. Epidemiology 4:428-434.

Agarwal R, Gupta KP, Kumar S, Mehrotra NK [1986]. Assessment of some tumorigenic risks associated with fresh and used cutting oil. Ind J Exp Biol 24:508-510.

Ahrens W, Pohlabelin H, Bolm-Audorff U, Jahn I, Jockel K-H [1991a]. Occupational exposure to cooling lubricants and lung cancer. Preliminary results from a case-control study. [Abstract]. In: Book of Abstracts: 8th International Symposium. Epidemiology in Occupational Health, Paris, September 10-12.

Ahrens W, Jockel K, Patzak W, Elsner G [1991b]. Alcohol, smoking, and occupational factors in cancer of the larynx: a case-control study. Am J Industr Med 20:477-493.

Albert RE, Sellakumar AR, Laskin S, Kuschner M, Nelson A, Snyder CA [1982]. Nasal cancer in the rat induced by gaseous formaldehyde and hydrogen chloride. J Natl Cancer Inst *68*:597-603

Almich BP, and GA Carson [1974]. Some effects of Charging on 10-mm Nylon Cyclone Performance", Am Ind Hyg Assoc J. 35, 603-612

Alomar A, Conde-Salaar L, Romaguera C [1985]. Occupational dermatoses from cutting oils. Cont Derm *12*:129-138.

Alomar A [1994]. Occupational skin disease from cutting fluids. Dermatologic Clinics - Occupational Dermatoses *12*:537-546.

Ameille J, Wild P, Choudat D, et al. Respiratory symptoms, ventilatory impairment, and bronchial reactivity in oil mist-exposed automobile workers. Am J Ind Med 1995;27:247-256.

Greaves IA, Eisen EA, Smith TJ, Pothier LJ, Kriebel D, Woskie SR, Kennedy SM, Shalat S, Monson RR [1995b]. Respiratory health of automobile workers exposed to metal-working fluid aerosols. II. Respiratory Symptoms, Final Draft.

Guralnick L [1963]. Mortality by occupation and cause of death. Department of Health, Education, and Welfare, Washington, DC. (Vital Statistics Special Reports 53(3), DHEW (PHS)).

Hagopian and Bastress [1976]. Recommended industrial ventilation guidelines. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 76-162.

Haguenoer JM, Cordier S, Morel C, Lefebvre JL, Hemon D [1990]. Occupational risk factors for upper respiratory tract and upper digestive tract cancers. Brit J Industr Med 47:380-383.

Hallock MF, Smith TJ, Woskie SR, Hammond SK [1994]. Estimation of historical exposures to machining fluids in the automotive industry. Am J Industr Med 26:621-634.

Hankinson JL, Hodous TK. Short-term prospective spirometric study of new coal miners. Clinical Investigations Branch, NIOSH, Morgantown, WV. NTIS PB83-237-446.

Hands, Dave [1995]: Poster Presentation" Ford FIH 005 method" at the Symposium titled "The Industrial Metal Working Environment: Assessment and Control. November 15, Dearborn Michigan.

Harada T, Morita T, Iwanaga S, Nakamura S, Niwa M [1979]. A new chromogenic substrate method for assay of bacterial endotoxins using Limulus hemocyte lysate. In: Biomedical Applications of the Horseshoe Crab (Limulidae), pp. 209-220, Alan R. Liss, Inc., New York.

Hendy MS, Beattie BE, Surge PS [1985]. Occupational asthma due to an emulsified oil mist. Brit J Ind Med 42:51-54.

Henry SA [1947]. Occupational cutaneous cancer attributable to certain chemicals in industry. Brit Med Bul *4*:389-401.

Herwaldt LA, Gormann GW, McGrath T, Toma S, Brake B, Hightower AW, Jones J, Teingold AL, Boxer PA, Tang PW, Moss CW, Wilkinson H, Brenner DJ, Steigerwalt AG, Broome CV [1984]. A New *Legionella* species, *Legionella feeleii* species Nova, causes Pontiac fever in an automobile plant. Ann Int Med *100*(3):333-338.

Hilfrich J, Schmeltz I, Hoffmann D [1978]. Effects of N-nitrosodiethanolamine and 1,1-diethanolhydrazine in Syrian golden hamsters. Cancer Lett. 4:55-60.

Hill EC, Al-Zubaidy T [1979]. Some health aspects of infections in oil and emulsions. Tribology International. *8*:27-29.

Hill EC [1983]. Microbial aspects of health hazards form water based metalworking fluids. Tribology International 16(3):136-140.

Hoffmann D, Rivenson A, Adams JD, Juchatz A, Vinchkoski N, Hecht SS [1983]. Effects of route of administration and dose on the carcinogenicity of N-nitrosodiethanolamine in the Syrian golden hamster. Can Res *43*:251-2524.

Hollander A, Heederik D, Versloot P, Douwes J [1993]. Inhibition and enhancement in the analysis of airborne endotoxin levels in various occupational environments. Am Ind Hyg Assoc J 54:647-653.

Hoshino H, Tanooka H [1978]. Carcinogenicity of triethanolamine in mice and its mutagenicity after reaction with sodium nitrite in bacteria. Can Res *38*:3918-3921.

Howe GR, Lindsay JP [1983]. A follow-up of a ten-percent sample of the Canadian labor force.I. caner mortality in males, 1965-73. JNCI 70:37-44.

Howe GR, Burch JD, Miller AB, Cook GM, Esteve J, Morrison B, Gordon P, Chambers LW, Fodor G, Winsor GM [1980]. Tobacco use, occupation, coffee, various nutrients, and bladder cancer. JNCI 64:701-713.

Hrubec Z, Blair AE, Rogot E, Vaught J [1992]. Mortality risks by occupation among US veterans of known smoking status. US Government Printing Office, Washigton DC (NIH Publication no 92-3407).

L

Hunting K, Nessel-Stephens, Schiffman A. Summary of AOEC Database Case Reports--The First Three Years: 1991-1993. Division of Occupational and Environmental Medicine, George Washington University, September 1995.

IARC [1978a]. N-nitrosodiethanolamine. In: IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Some N-nitroso compounds, Volume 17. Lyon, France: International Agency for Research on Cancer, pp. 77-82.

IARC [1978b]. N-nitrosomorpholine. In: IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Some N-nitroso compounds, Volume 17. Lyon, France: International Agency for Research on Cancer, pp. 263-280.

IARC [1983]. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Polynuclear Aromatic Compounds, Part 1, Chemical, Environmental, and Experimental Data, Volume 32. Lyon, France: International Agency for Research on Cancer.

IARC [1984]. Mineral oils. In: IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Part 2, Volume 33. Lyon, France: International Agency for Research on Cancer, pp. 87-168.

Loeppky RN, Hansen TJ, Keefer LK [1983]. Reducing nitrosamine contamination in cutting fluids. Fd Chem Toxicol 21(5):607-613.

Lushbaugh CC, Green JW, Redemann CE. [1950]. Effects of prolonged inhalation of oil fogs on experimental animals. Arch Ind Hyg Occup Med 1:237-247.

Mack TM, Paganini-Hill A [1981]. Epidemiology of pancreas cancer in Los Angeles. Cancer 47:1474-1483.

Mackerer CR [1989]. Health effects of oil mists: A brief review. Toxicol Ind Health 5(3):439-440.

Maekawa A, Onodera H, Tanigawa H, Furuta K, Kanno J, Natsuoka C, Ogiu T, Hayashi Y [1986]. Lack of carcinogenicity of triethanolamine in F344 rats. J Tox Environ Hlt. 19:345-357.

Malker Hs, McLaughlin JK, Silverman DT, Ericsson JLE, Stone BJ, Weiner JA, Malker BK, Blot WJ [1987]. Occupational risks for bladder cancer among men in Sweden. Cancer Research *47*:6763-6766.

Mallin K, Berkeley L, Young Q [1986]. A proportional mortality ratio study of workers in a construction equipment and diesel engine manufacturing plant. Am J Industr Med 10:127-141.

Mansdorf SZ, Lubs PL [1994]. Role of the industrial hygienist:Evaluation and management of occupational skin disease. Dermatologic Clinics- Occupational Dermatoses 12:591-596.

Mastromatteo E [1955]. Cutting oils and squamous cell carcinoma. Part 1: Incidence in a plant with a report of six cases. Brit J Ind Med *12*:240.

Mattsby-Baltzer, I, L Edebo, B. Jarvholm, et al.[1990]. Subclass distribution of IgG and IgA antibody response to *Pseudomonas pseudoalcaligenes* in humans exposed to infected metal-working fluid. *86*:231-238.

Mattsby-Baltzer J, Edebo L, Jarvholm B, Lavenius B [1989]. Serum antibodies to *Pseudomonas pseudoalcaligenes* in metal workers exposed to infected netal-working fluids. International Archives of Allergy and Applied Immunology 88(3):304-311.

Mattsby-Baltzer I, Sandin M, Ahlstrom B, Allenmark S, Edebo M, Falsen E, Pedersen K, Rodin N, Thompson RA, Edebo L [1989]. Microbial growth and accumulation in industrial metal-working fluids. Appl Environ Microbiol55:2681-2689.

Matanoski GM, Elliott EA [1981]. Bladder cancer epidemiology. Epidemiol Rev 50:588-600.

Mausner JS and Kramer S [1985]. Mausner & Bahn Epidemiology-an introductory text. Philadelphia: WB Saunders

McKee RH, Scala RA, Chauzy C [1990]. An evaluation of the epidermal carcinogenic potential of cutting fluids. J Appl.Tox 10:251-256.

Menter P, Harrison W, Woodin WG [1975]. Patch testing of coolant fractions. J Occup Med *17*(9):565-568.

1994 ?

Merideth SK, McDonald JC. Work-related respiratory disease in the United Kingdom, 1989-1992: report on the SWORD project. Occup Med 1944;44:183-189.

Meredith SK, Taylor VM, McDonald JC. Occupational respiratory disease in the United Kingdom 1989: a report to the British Thoracic Society and the Society of Occupational Medicine by the SWORD project group. Brit J Ind Med 1991;48:292-298.

Milham S [1983]. Occupational mortality in Washington State, 1950-1979. National Institute for Occupational Safety and Health, Cincinnati, OH. (DHHS publication no (NIOSH) 83-116).

Mills AL and Alexander M [1976]. N-Nitrosamine formation by cultures of several microorganisms. Appl Environ Micro *31*(6):892-895.

Milton DK, Gere RJ, Feldman HA, Greaves IA [1990]. Endotoxin measurement: Aerosol sampling and application of a new Limulus method. Am Ind Hyg Assoc 51:331-337.

Milton DK [1992]. Endotoxin in metal working fluids. Report to United Auto Workers - General Motors Joint National Committee on Occupational Health and Safety. Harvard School of Public Health.

Milton DK, Feldman HA, Neuberg DS, Bruckner RJ, Greaves IA [1992]. Environmental endotoxin measurement: The kinetic Limulus assay with resistant-parallel-line estimation. Environ Res 57:212-230.

Monson R [1990] Occupational Epidemiology, 2nd Edition, CRC Press, Inc., Boca Raton, Florida.

Monson RR, Greaves IA, Eisen EA, Woskie SR, Smith TJ, Hallock M [1993]. Final report on health effects of machining fluids (respiratory morbidity cross-sectional study draft report),

Morgan MDL, Glass D, Tobertson AS, Boran A, Jones SH, Murray F, Burge PS [1988]. Prevalance of respiratory symptoms in an engineering factory with exposure to oil mists. Thorax 47:263.

Morris NM, Catalano E, Berni RJ [1988]. 3-hydroxymyristic acid as a measure of endotoxin incotton lint and dust. Am Ind Hyg Assoc J 49:81-88.

Morrison DC, Ulevitch, RJ [1978]. The effects of bacterial endotoxins on host mediation systems. Amer J Pathol 93:527-617