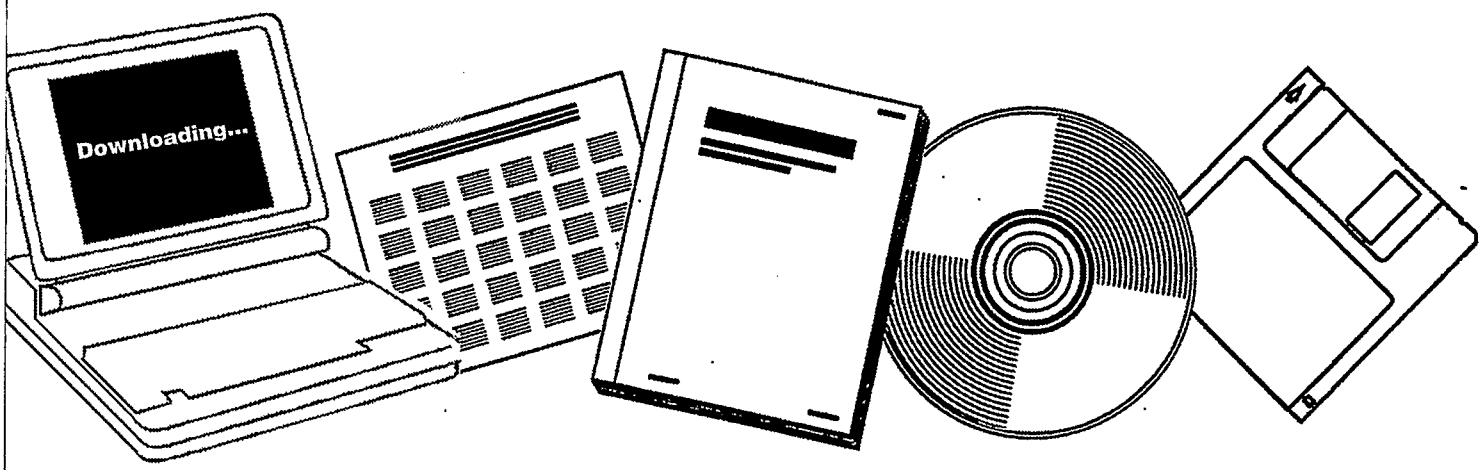
ASSESSMENT OF TRACE CONTAMINANTS FROM A MODEL INDIRECT LIQUEFACTION FACILITY. VOLUME IV. PUBLIC HEALTH HAZARDS OF LURGI/FISCHER-TROPSCH COAL LIQUEFACTION

ARGONNE NATIONAL LAB., IL

JAN 1982



DOE/EV/10291--T2-Vol.4

DE84013990

Report 1234-04-81-TR

Assessment of Trace Contaminants From a Model Indirect Liquefaction Facility

VOLUME IV — PUBLIC HEALTH HAZARDS OF LURGI/FISCHER-TROPSCH COAL LIQUEFACTION

DOE/EV/10291--T2-Vol.4

DE84 013990

Prepared By:

J.R. Gasper and S.E. Rosenberg

Integrated Assessments and Policy Evaluation Group
Energy and Environmental Systems Division
Argonne National Laboratory
Argonne, Illinois 60439

January 1982

MANAGEMENT SYSTEMS DIVISION

GENERAL RESEARCH CORPORATION

A SUBSIDIARY OF FLOW GENERAL INC.

7655 Old Springhouse Road, McLean, Virginia 22102

Acol-79EV10291.

MASTER

Prepared For:

Technology Assessment Division
Environmental Protection, Safety, and Emergency Preparedness Office
U.S. Department of Energy

Under Contract No. W-31-109-ENG-38

FOREWORD

Development and deployment of a commercial indirect liquefaction industry has been proposed as a means of reducing United States dependence on foreign sources of energy.

Deployment of a commercial industry on an environmentally acceptable basis requires identification and evaluation of potential environmental problems. This assessment is an attempt to anticipate potential environmental hazards that may be posed by commercial-scale facilities to provide an improved basis for planning and implementing environmental research.

The study comprises four major tasks: characterization of hazardous materials released from an indirect liquefaction facility; assessment of ecological hazards; assessment of public health hazards; and assessment of occupational health hazards. The report is organized in the same manner. Volume I is an overview and summary of the results; volume II presents stream characterization data; and volumes III, IV and V present assessments of ecological, public health and occupational health hazards, respectively.

The study was sponsored by the Technology Assessment Division of the Department of Energy. Organizations participating in the assessment were General Research Corporation, Oak Ridge National Laboratory, and Argonne National Laboratory.

ACKNOWLEDGMENTS

The authors wish to express their appreciation to Dr. Arnold Goldberg, President, INA Loss Control Services, Inc., formerly the DOE Project Officer, and Mr. Bipin Almaula, of the U.S. Department of Energy, for their assistance and guidance throughout the project.

CONTENTS

FIGURES

TABLES

TABLES (Cont'd)

EXECUTIVE SUMMARY

This analysis identifies the public health hazards of wastes from Lurgi/Fischer-Tropsch coal liquefaction. Because data on dose-response and synergism are not available for many of the waste chemicals, we evaluated hazards with a relative risk approach. This approach employs two measures of hazards. First, body burdens that result from exposure to Lurgi/Fischer-Tropsch wastes are compared to body burdens from other sources of the same chemicals. Second, ambient concentrations of pollutants from Lurgi/Fischer-Tropsch operations are projected and compared to various air and water quality standards.

Quantitative characterization of liquefaction wastes is limited to a few specific process streams. Consequently, quantifiable measures of risk are restricted to those streams. For waste streams that are not quantified, we used qualitative measures of risk.

Results of the quantitative assessment indicate:

- Trace-element body burdens from Lurgi/Fischer-Tropsch wastes will be similar to those from coal-fired power-plant wastes or from background air and water, and will be substantially below those from dietary exposure.

- Ambient atmospheric concentrations of ammonia, hydrogen sulfide, sulfur dioxide, nitrogen oxides, arsenic, nickel carbonyl, and mercury -- seven out of 73 substances for which risks were quantifiable -- are projected to violate or be within one order of magnitude of acceptable levels for public exposure. However, due to their toxicity and slow biodegradation, only the trace elements and nickel carbonyl are projected to be of concern to public health.

- Ambient aquatic concentrations of trace elements, phenols, polynuclear aromatic hydrocarbons, substituted benzenes, mercaptans, fatty acids, and aromatic amines will violate or be within one order of magnitude of acceptable levels for public exposure. Trace elements, polynuclear aromatic hydrocarbons, and phenols are projected to be of concern to public health.

- Unquantified waste streams currently release carcinogens, co-carcinogens, and toxic substances to the environment. All carry the potential for public health impact.

- The flaring of process gas during normal operations may release carcinogens and toxic chemicals to the atmosphere. The effect of flaring on gas composition needs to be assessed.

We also pinpoint the need for more in-depth research in the following areas:

- Waste-stream volume and composition during startup, shutdown, and upset operation,
- Changes in environmental controls in the reference design that affect waste stream volumes or components, and
- Synergistic, antagonistic, and dose-response relationships of Lurgi/Fischer-Tropsch wastes.

INTRODUCTION

Decreasing U.S. dependence on foreign oil is an energy policy priority underscored by legislation that appropriates funds to foster growth of the synthetic fuel industry. Indirect coal liquefaction is one synthetic fuel technology with near-term potential for commercial application. However, environmental effects of the technology need to be evaluated before commercial scale production can begin. This report assesses the impact of emissions, effluents, and solid waste by-products of liquefaction process streams on public health.

This assessment is part of a larger study of indirect coal liquefaction performed under the auspices of the U.S. Department of Energy, Office of Environmental Assessments, Technology Assessments Division. The study included four major efforts: (1) characterization of hazardous materials released from an indirect liquefaction facility, (2) occupational health assessment, (3) public health assessment, and (4) ecological assessment. Major contributors to the program include: Argonne National Laboratory, International Research and Technology, Oak Ridge National Laboratory, and U.S. Department of Energy.

No commercial-scale indirect liquefaction facilities currently exist in the United States. Pilot- and bench-scale operations, however, have identified the process' potential to release many toxic substances to the environment. Operating experience at commercial-scale facilities in South Africa, Yugoslavia, and Scotland have confirmed this potential. This report estimates the risk to the public from operation of a commercial-scale, indirect, coal-liquefaction facility that uses Lurgi gasification and Fischer-Tropsch liquefaction to produce fuels ranging from synthetic natural gas to heavy fuel oil.

Ideally, health hazards from the environmental release of toxic substances should be quantified in terms of deaths, illnesses, or some other absolute measure of exposure. Unfortunately, not enough data exist for Lurgi/Fischer-Tropsch by-products to allow such quantification. The lack of comprehensive data on commercial-scale U.S. operating conditions, dose-response relationships, and environmental transformation rates restricts the scale of the risk assessment. Enough information is available, however, for a relative risk analysis that compares the potential impacts of Lurgi/Fischer-Tropsch with impacts from other sources of toxic wastes. Such an analysis can identify those substances that present significant risk to public health. This assessment uses the relative risk approach.

This report assesses the public health risk of atmospheric emissions, liquid effluents, and solid wastes generated during steady-state operation of a commercial-scale (28,000 ton/day) Lurgi/Fischer-Tropsch coal liquefaction facility. The hypothetical plant is located in an area with meteorological, geographical, and hydrological characteristics that match average U.S. conditions.

The liquefaction process is a pressurized system under which process streams have little opportunity for release to the environment. However, during start-up, normal operation, shutdown, and upset conditions, process gases are flared to the atmosphere. These process gases include a variety of

toxic chemicals and possible carcinogens. Incomplete combustion of flare gas may expose the public to these species. The volume and components of liquid and solid waste streams will also vary between normal operating and upset conditions. However, the frequency and magnitude of upset operations, the completeness of flare-gas combustion, and waste-stream composition are too uncertain to adequately assess the resulting exposures during these conditions.

Additionally, this assessment does not consider the impacts of coal mining or plant construction and decommissioning. These impacts should be similar to those from existing commercial operations, such as oil refining, coal-fired electricity generation, and coke production, and thus were not addressed. The public health impacts from accidents, such as the explosion of a gasification vessel, were not quantified in this assessment. The potential impact from such an event is uncertain and extremely sensitive to site-specific considerations and as such was not discussed.

2 METHODOLOGY

This analysis of the public health hazards of Lurgi/Fischer-Tropsch indirect coal liquefaction determines relative health impact by measuring a number of surrogates. The surrogates (body burdens, ambient concentrations, substance toxicity) are required because data on dose-response, synergism, environmental transformation, and waste-streams are insufficient to quantify direct health impacts. Hazard is determined from the surrogate that most closely reflects actual impact, based on available data that correlate surrogates and human health effects. The method identifies which substances released through Lurgi/Fischer-Tropsch liquefaction are of significant public health concern and which have undetermined effects requiring further study.

The public health assessment is based on emission, effluent, and solid-waste data from Part 1 of the study, characterization of hazardous wastes. Although actual commercial facilities may vary in the use of specific process and waste flows (e.g., the use of deep well injection instead of reverse osmosis, or the substitution of Claus for Stretford sulfur-control units), the characterization provides state-of-the-art estimates of operating conditions in the United States. Figure 2.1 illustrates the process flow diagram for the Lurgi/Fischer-Tropsch liquefaction. Table 2.1 lists process streams with the potential for releasing environmental contaminants, and Table 2.2 lists the components of the assessment streams. Quantification of the streams varies from complete to nonexistent, but is the most up-to-date information available. As a result of the incomplete characterization, the quantification portion of the risk analysis reflects only those streams for which data or estimates are available.

Six streams have been quantified: two gaseous streams -- utility stack gas (28) and cooling tower drift (29); two liquid streams -- reverse osmosis waste (53) and combined ash leachate (69); and two solid wastes -- combined ash (36) and biosludge (70). Data on 14 of the streams listed in Table 2.1 were insufficient for quantitative assessment.

Lockhopper vent gas (72) is especially significant. The Lurgi gasification vessel requires purging of small amounts of process gas to relieve over-pressure during normal operation. Larger amounts of gas are purged during start-up, shutdown, and upset operation. This vent gas contains a rich mix of synthesis gas (H_2,CO), various toxic hydrocarbons, and trace elements and is flared to the atmosphere. Flaring reduces synthesis gas to water vapor and carbon dioxide and combusts the hydrocarbons while having little effect on the trace elements. The degree of organic degradation depends on factors such as the temperature and duration of the burn, which vary with operating conditions. Thus, even though unflared vent gas is characterized, the effect of the flared stream on public health has not been quantified due to the need for characterization of the flared gas. This type of characterization must be based on empirical data that are currently unavailable.

Figure 2.2 illustrates the methodology used to assess public health risk. Components of waste streams are first characterized by health indicators such as toxic effects, metabolites, bioaccumulation, environmental degradation, and acceptable exposure levels. Components are then screened by toxicity. Nontoxic substances are not dealt with further. Substances

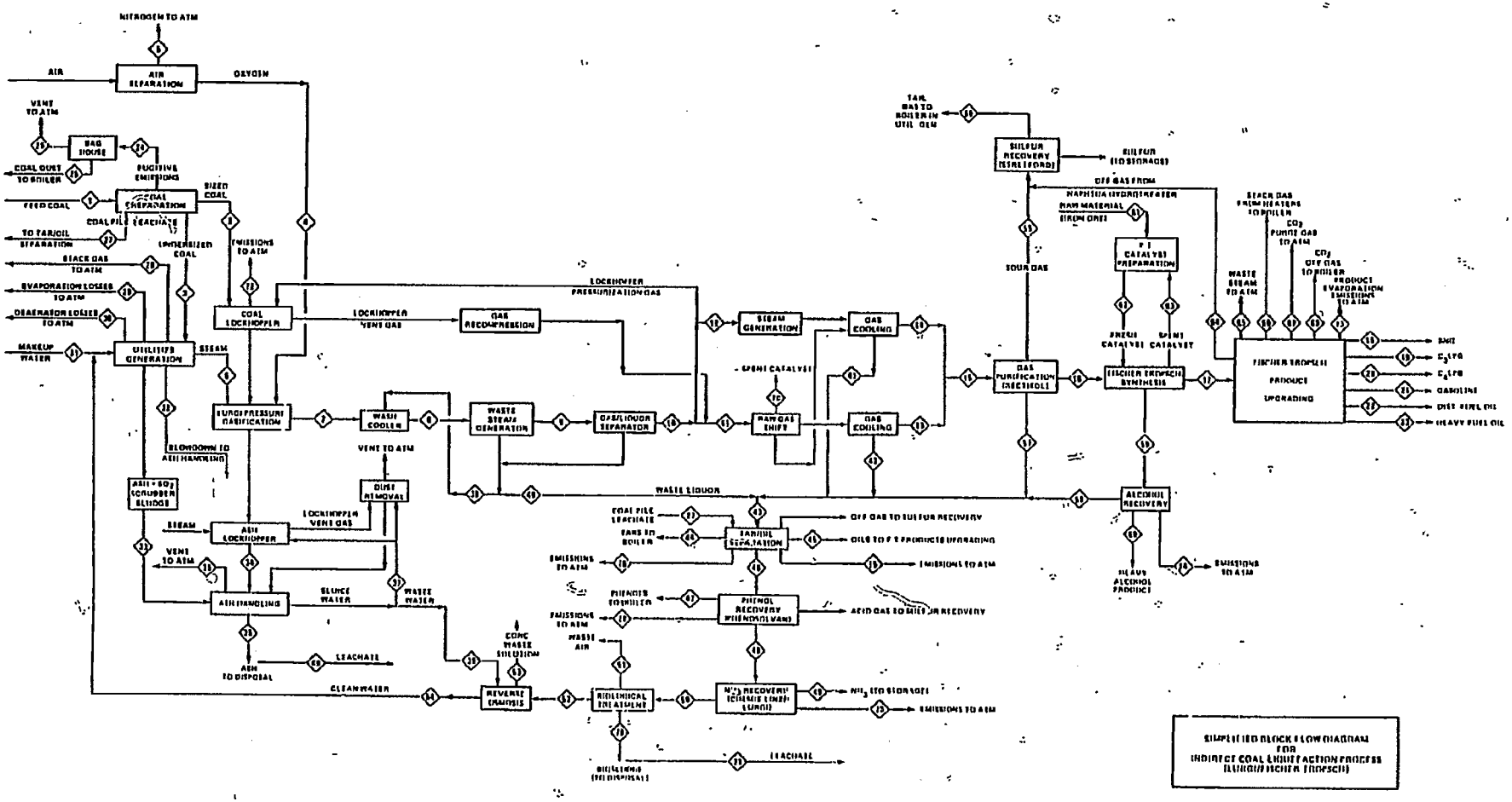


Fig. 2.1 Process Flow Diagram for Lurgi/Fischer-Tropsch Liquefaction

identified as possibly carcinogenic or toxic are flagged as potential limiters to development of the Lurgi/Fischer-Tropsch technology that require further assessment.

The screened substances are then categorized for further assessment. Four categories are established, based on the kinds of information found in Part 1 of the larger study, characterization of hazardous wastes. Category 1 substances are quantified based on data from commercial-scale Lurgi/Fischer-Tropsch facilities identical to the hypothetical reference facility. Category 2 substances are quantified based on data from processes similar to the reference facility. These may include bench-scale facilities, pilot plants, or high-Btu gasification using Lurgi or non-U.S. Lurgi/Fischer-Tropsch processes. Category 3 substances are probably present in the environmental streams, but information on concentrations or volumes is not available. Category 4 substances are probably present in the environmental streams, but their toxic potential is unknown.

Table 2.1 Process Streams with Potential for Environmental Release

Process Stream Type	Stream No.
Gas	
Utility stack gas	28
Evaporative emissions	29,73,74, 75,76,77
Deaeration emissions	30
Bag-house vent gas	26
Ash-handling emissions	35
Fischer-Tropsch purge gas, waste streams	65,67
Lockhopper vent gas	72
Liquid	
Reverse-osmosis waste solution	53
Biosludge leachate	71
Combined-ash leachate	69
Solid	
Combined utility, gasifier ash	36
Biosludge	70
Spent Fischer-Tropsch catalyst	63
Spent shift catalyst	79

A 2-tier approach is then used to determine the health hazards of Lurgi/Fischer-Tropsch wastes in Categories 1 and 2. First, the Source Analysis Model (SAM 1) was applied to quantified environmental release data to project maximum ambient concentrations. The model, developed by the U.S. Environmental Protection Agency (EPA), determines maximum ambient concentration of atmospheric and aquatic pollutants from point sources. It reflects average U.S. environmental conditions and was used in this assessment to estimate worst-case conditions for a site in Wyoming. Resulting concentrations were then compared to public health standards or levels suggested as acceptable by the EPA. Substances that violate or approach limits are identified.

The second tier projects the relative hazards of Lurgi/Fischer-Tropsch wastes to human health. Dose-response data are insufficient to calculate absolute impact (such as death or illness) from exposure to most process pollutants. However, for some pollutants (e.g., trace elements) enough data on emission and human exposure, absorption, and excretion are available to calculate the amount of substance that will accumulate in exposed humans (body burden). The Argonne body-burden model was used to project body burdens resulting from exposure to atmospheric, liquid, and solid wastes from Lurgi/Fischer-Tropsch liquefaction; from coal-fired electricity generation; from

Table 2.2 Components of Process Streams with Potential for Environmental Release

Compound	Found in Stream No.
Aliphatics, alicyclics, and fatty acids	
Acetic acid	29,72,53
Propanoic acid	29,72,53
Butanoic acid	29,72,53
2-Methylpropanoic acid	29,72,53
Pentanoic acid	29,72,53
3-Methylbutanoic acid	29,72,53
Hexanoic acid	29,72,53
Ethane	72
C_2-C_6 Aliphatics	72
Ethanol	74
C_{3+} Alcohols	74
Methane	72
Benzenes and substituted benzenes	
Benzene	75,72
Ethylbenzene	29,53,72
Toluene	29,53,75,72
Xylenes	72,75
O-Xylene	29,53
Biphenyl	29,72
Indan	29,72
Monohydric phenols	
Cresols	76,75,77,72
Phenol	29,53,72,75, 76,77
Alkyl phenols	75
2-Methylphenol	29,53,72
3-Methylphenol	29,53
Triethylphenol	72
O-Isopropylphenol	72
Xylenols	77,72
2,4-Xylenol	29,53
Dihydric phenols	
Catechol	29,53,77
Methylcatechol	77
3,6-Dimethylcatechol	29,53
Resorcinol	29,53,77
Methylresorcinol	77
5-Methylresorcinol	29,53
4-Methylresorcinol	29,53
Mercaptans	
Methanethiol	29,53.72
Ethanethiol	72
Sulfur heterocyclics	
Methylthiophene	72
Thiophene	72,75
Benzothiophene	75
Nitrogen heterocyclics	
Acridine	72
2,4-Dimethylpyridine	29,53
2,5-Dimethylpyridine	29,53
2-Methylpyridine	29,53
3-Methylpyridine	29,53
4-Methylpyridine	29,53
Pyridine	29,53
Quinoline	29,53,72
Oxygen heterocyclics	
Benzofuran	72
Dibenzofuran	29,53,72,76
Nitrosamines	
N-Nitrosamine	72
Polynuclear aromatic hydrocarbons	
Acenaphthalene	29,53,70
Anthracene	29,70,72,76
Benz(a)anthracene	29,70
Benzo(g,h,i)perylene	29,70
Benzo(a)pyrene	29,70
Benzo(e)pyrene	29,70
Chrysene	29,70,72
Fluoranthene	29,70,72,76
Fluorene	29,70,72,76
Indene	72
Naphthalene	29,70,72, 75,76
Perylene	29,72
Phenanthrene	29,70,72,76
Pyrene	29.70,76,72
Aromatic amines	
Aniline	29,53,72
Methylaniline	72
Dimethylaniline	72
Gases	
Sulfur oxides	28
Nitrogen oxides	28
Carbon monoxide	72
Carbon dioxide	28,72
Nickel carbonyl	72
Hydrogen cyanide	72
Carbonyl sulfide	72
Ammonia	72
Hydrogen sulfide	72
Particulates	28
Nitrogen	28,72
Oxygen	28
Water	28,36,72
Hydrogen	28,72
Trace Elements	
Aluminum	69
Arsenic	72,28,29,53, 36,69,70,72
Barium	28,72,36
Beryllium	72,28,29,53, 36,70
Boron	28,72,29,36, 53
Cadmium	28,29,36,53, 69,70,72
Carbon	79
Chromium	28,36,72
Cobalt	28,36,72,79
Copper	69,36,72
Fluorine	29,36,53,28
Lead	72,28,29,53, 36,69,70,79
Mercury	72,28,29,53, 36,69,70,79
Molybdenum	79
Iron	69,72
Manganese	28,72,36,53, 69
Selenium	28,72,36,69, 79
Vanadium	28,36,72,29, 53
Sulfur	36,79
Zinc	36,69,28,72
Tin	28,72,36
Uranium	28,72,36
Silver	72,36
Nickel	28,72,29,53, 36,69

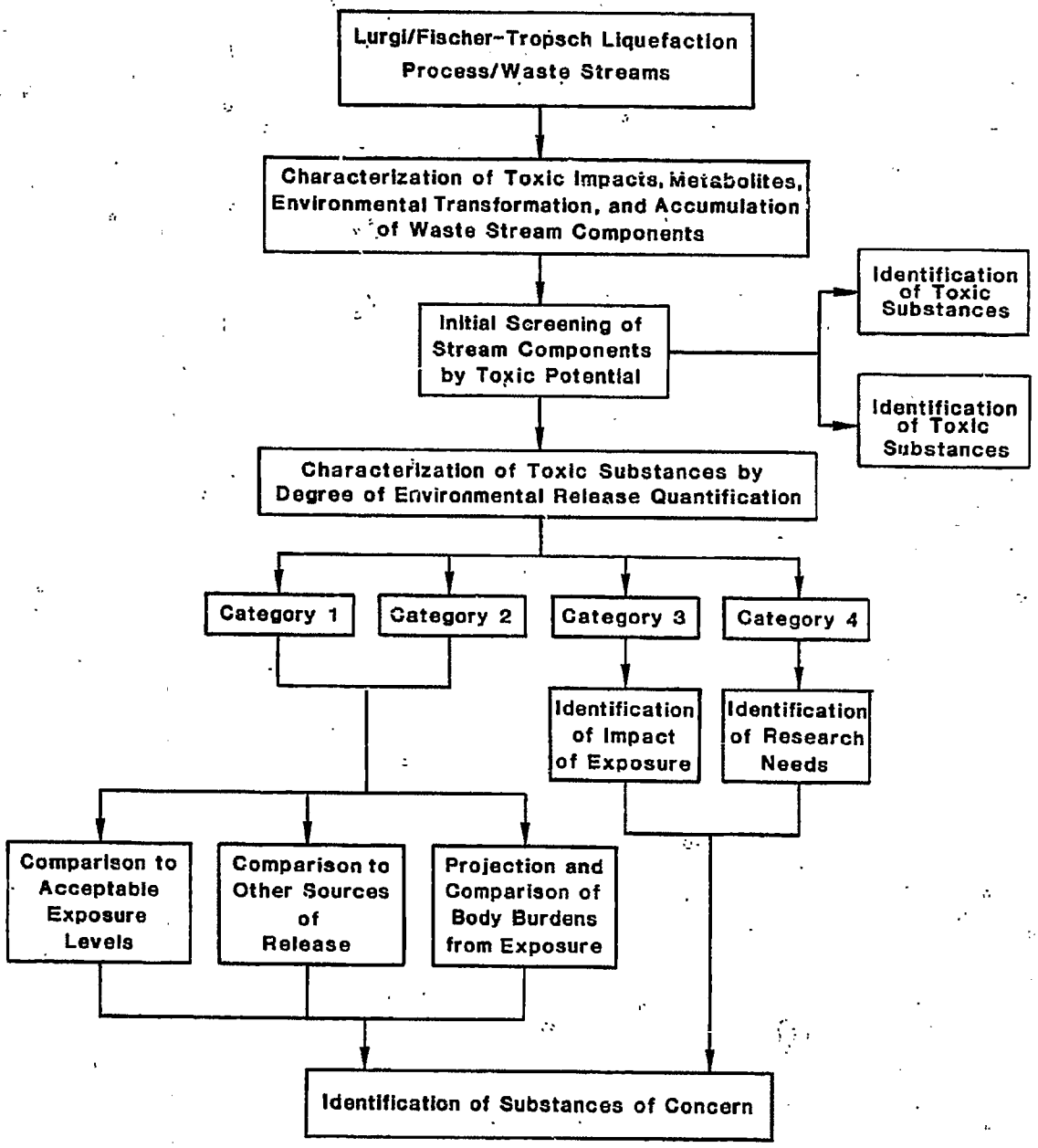


Fig. 2.2 Public Health Risk-Assessment Methodology

background air and water; and from diet. Comparing the body burdens from each source of exposure shows the relative risk to humans from Lurgi/Fischer-Tropsch wastes.

Category 3 substances are discussed in the context of potential impact if exposure occurs. Impacts of exposure such as toxic effect and metabolites are included in Sec. 3. Category 4 substances are identified as requiring further research.

3 RESULTS/DISCUSSION

3.1 INITIAL SCREENING

This section provides health data on chemicals and elements identified in Lurgi/Fischer-Tropsch effluent streams. The chemicals are screened on the basis of their toxicity, metabolic pathway, and potential to accumulate in the environment. Existing and proposed standards, regulations, or recommended exposure levels are also given.

The chemicals are grouped into the following families:

- trace elements
- polynuclear aromatic hydrocarbons
- nitrogen heterocyclics
- sulfur heterocyclics (thiophenes)
- oxygen heterocyclics
- phenols
- mercaptans (thiols)
- aromatic amines
- benzene and substituted benzenes
- aliphatics, alicyclics, and fatty acids
- nitrosamines
- gases

The type of waste stream (liquid, solid, or gas) in which the effluent has been identified is also listed. Table 2.1 lists the environmental waste streams that have been analyzed for toxic substances. In several instances, specific health data for each chemical in a particular category was unavailable or the health data for each effluent in the group was related so closely that a general discussion of the chemical group was adequate. For example, 3-methylcatechol, 4-methylcatechol, and 3,6-dimethylcatechol are not discussed separately but are grouped under the generic heading of dihydric phenols.

3.1.1 Trace Elements

Trace element emissions have been shown to have adverse effects on the health of exposed populations.[1] At high doses, many trace elements are toxic or carcinogenic.[2,3] Toxicity of a trace element depends on its chemical and physical state, and also on the size of particles transporting trace elements through the atmosphere. Elements adsorbed to respirable particles pose a much greater risk to health than those transported on nonrespirable particles. The fate of these elements in the environment will also determine human health effects. Trace elements may accumulate through food chains and may react synergistically. The following trace elements are significant wastes from coal gasification.

3.1.1.1 Arsenic

Present in streams: 27, 38, 43, 46, 48, 50, 53, 54, 31, 33, 34, 36, 69, 70, 79, 28, 72.

Waste stream types: liquid, solid, and gas.

Arsenic toxicity is related to the chemical state of the element; the pentavalent form is less toxic than the trivalent form. Acute arsenic poisoning by ingestion causes abdominal pain and vomiting, while acute poisoning by inhalation produces giddiness, headache, extreme weakness, and nausea. Chronic arsenic poisoning results in muscle weakness, loss of appetite, gastrointestinal pains, irritation of nasal and oral membranes, coughing, and dermatitis. Data on human exposure point to a causal relationship between skin cancer and high-level exposures to inorganic arsenic compounds.[4]

Arsenic readily combines with sulfhydryl groups, thus interfering with cellular enzymatic reactions. It is largely eliminated in the urine, with smaller amounts eliminated in the feces, hair, epithelium, nails, and possibly in the exhaled breath.

Arsenic has been shown to accumulate in freshwater and saltwater organisms. Although no animal experients have demonstrated carcinogenicity of arsenic, sodium arsenate induces developmental malformations in a variety of test animals, including chick embryos, hamsters, rats, and mice.[5]

The annual average concentration of atmospheric arsenic near major emission sources ranges from 3 mg/m^3 to 5,900 mg/m^3, with most below 290 mg/m^3. Daily inhalation levels in these areas may be as high as 6,148 mg to 125,080 mg for men and 3,219 mg to 65,490 mg for women.[5]

The American Conference of Governmental Industrial Hygienists (ACGIH) has set 0.5 mg/m^3 as the threshold limit value (TLV), time weighted average, for airborne arsenic. The Conference also issued a Notice of Intended Change (as of 1979) to reduce the TLV from 0.5 to 0.05 mg/m^3. The National Institute of Occupational Safety and Health (NIOSH) has recommended a ceiling level for airborne inorganic arsenic of 2 mg/m^3 for any 15-min period of the workday.[6] For the maximum protection of human health from the potential carcinogenic effects of arsenic exposure through ingestion of water or contaminated aquatic organisms, the EPA is considering setting criteria at an interim target risk level in the range of 10^{-5} $\mu g/L$, 10^{-6} $\mu g/L$, or 10^{-7} $\mu g/L$, with corresponding criteria of 0.02 $\mu g/L$, 0.002 $\mu g/L$, and 0.0002 $\mu g/L$, respectively. The drinking water standard for arsenic is 50 $\mu g/L$.

3.1.1.2 Beryllium

Present in streams: 27, 43, 46, 48, 50, 53, 54, 31, 33, 34, 36, 70, 28, 72.

Waste stream types: liquid, solid, and gas.

Beryllium is hazardous as an air contaminant. Berylliosis, a severe lung disease, develops from chronic exposure to airborne particles of soluble and insoluble beryllium compounds. The lowest toxic concentration for humans is reported as 0.1 mg/m^3.[7] In humans, short-term exposure to beryllium oxide in air at 4 mg/m^3 produced a high incidence of disease and some fatalities.[5] The onset of chronic toxicity may follow exposure by as long as 5 years. Symptoms include pneumonitis with coughing, chest pains, and general weakness.

In addition, lung and bone cancers are firmly associated with beryllium exposure.

Beryllium inhibits several enzyme systems, including alkaline phosphatase, phosphoglucomutase and potassium-activated ATPase. Reportedly, it interferes with DNA metabolism in the liver and may induce chromosomal and mitotic abnormalities. Beryllium increases misincorporation of nucleotides during polymerization of DNA.[2]

Marine organisms accumulate beryllium in levels up to 1,000 times the ambient concentration.[8] Intravenously administered beryllium is excreted in urine or deposited in kidney or bone; cows excrete little of it in their milk. The half-life ranges from 890 days in rats to 1,770 days in monkeys.

The National Air Sampling Network indicated that the average 24-h concentration of beryllium in air was less than 0.0005 $\mu g/m^3$. An average concentration of 0.0281 $\mu g/m^3$ was reported within 0.5 mi of a large beryllium plant in Reading, Pennsylvania.

The TLV for beryllium was set at 2 $\mu g/m^3$ by the ACGIH in 1979; the Occupational Safety and Health Administration (OSHA) has recommended that occupational exposure to the element and its compounds not exceed 1 $\mu g/m^3$.[9] The EPA National Emission Standard for Hazardous Air Pollutants limits emissions to not more than 10 g in 24 h or a maximum output concentration of 0.01 $\mu g/m^3$, 30-d average.[10]

3.1.1.3 Boron

Present in streams: 43, 46, 50, 52, 53, 54, 31, 34, 36, 72.

Waste stream types: liquid, solid, and gas.

Boron, a known constituent of coal, is not a highly toxic element. Serious human health effects are not reported under potential exposure conditions and the element does not accumulate significantly in body tissues.[11] The current TLV is 3.1 mg/m^3 for boron and 10 mg/m^3 for boron oxide. The EPA's estimated permissible concentration (EPC) in air is 7.4 $\mu g/m^3$.[12]

3.1.1.4 Cadmium

Present in streams: 27, 38, 43, 46, 50, 52, 53, 54, 31, 33, 34, 36, 69, 70, 28, 72.

Waste stream types: liquid, solid, and gas.

Cadmium is highly toxic. It can enter the body through the lungs or the gastrointestinal tract and is excreted through the kidneys, possibly causing proteinuria.[13] In humans, the threshold for kidney dysfunction is about 200 mg/kg in the renal cortex.[14] Inhalation or ingestion produces both acute and chronic effects. Workers who inhaled 0.01-0.27 mg/m^3 experienced pulmonary and renal damage[15] and ingestion of 13-15 parts per million (ppm)

was toxic to children.[16] Cadmium has been identified as a cause of the Japanese Itai-Itai disease, which is characterized by a degeneration of the bones.[17]

The active form of the metal, the Cd^{+2} ion, binds to the membranes of the mitochondria and inhibits enzymes required in cellular energy production.[18] Cadmium is a dangerous cumulative poison that tends to collect in the liver and kidneys of exposed organisms. Fish tissues have been reported with levels 2000 times as great as those of ambient water.[19] Over 90% of cadmium in the blood is found in the red cells, probably bound to hemoglobin. The biological half-life is estimated at 16-33 yr.[20] Cadmium and cadmium compounds are reported to produce oncogenic and teratogenic effects.

Numerous official and private agencies, both foreign and domestic, have suggested limits for cadmium in the environment. Table 3.1 lists some of the more pertinent recommendations.

3.1.1.5 Lead

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 33, 34, 36, 72.

Waste stream types: liquid, solid, and gas.

Lead can enter the body via inhalation of dust, fumes, mists, or vapors; via ingestion of food or from tobacco or fingers; and through the skin (especially organic lead compounds). Lead is teratogenic in animals and decreases reproductive ability in men and women.[21] Toxic effects include blood chemistry disturbances, neurological disorders, kidney damage, and cardiovascular dysfunction.

When ingested, some lead passes through the body unabsorbed and is eliminated in the feces. A greater portion is caught in the liver and excreted in part by bile. For this reason, large doses or long exposure times are usually necessary to produce symptoms. On the other hand, absorption takes place easily from the respiratory tract and symptoms develop more quickly.

Table 3.1 Cadmium Standards

Agency	Year	Standard in Air ($\mu g/m^3$)	Remarks
OSHA	1974	100	This standard is for cadmium fume. Limit for dust is 200 $\mu g/m^3$.
NIOSH	1977	40	
ACGIH	1977	40	ACGIH notes cadmium oxide as "industrial substance suspect of carcinogenic potential for man."
EPA	1977	0.12	

Lead is a cumulative poison with a biological half-life of 6 mo.[22] It accumulates in cattle tissue or milk and is known to cross the human placenta. The most recent action to regulate lead levels was taken by the Consumer Product Safety Commission, which in 1977 lowered the maximum allowable concentration of lead in house paint to 0.06%. The federal standard for lead and its inorganic compounds is 0.2 mg/m^3 as a time-weighted average. In November, 1978, OSHA set a final standard that gives industries 1-3 yr to reach a 0.1 mg/m^3 level, and from 1-10 yr to reach a final standard of 0.05 mg/m^3.

3.1.1.6 Mercury

Present in streams: 27, 38, 43, 46, 48, 50, 52, 53, 54, 31, 33, 34, 36, 69, 70, 79, 28, 72.

Waste stream types: liquid, solid, and gas.

Characteristic consequences of chronic exposure to inorganic mercury compounds include tremors, psychic disturbances, renal damage, and cardiovascular disease. Fetal effects have been observed in humans and animals.[23] Inhaled mercurials will damage the bronchial epithelium and, in freshwater invertebrates, interrupt respiratory function. Ingestion of 1.4 mg/kg of mercury has resulted in human death.[12]

Mercury, like most heavy metals, interferes with enzymes and proteins that function in metabolic reactions. In addition, it depresses active transport mechanisms involving sodium, potassium, and certain anions in the kidney. Brain cells are particularly sensitive to methylmercury.

The greatest risk for humans is mercury poisoning through food. Mercury and methylmercury accumulate in the food chain, especially in aquatic environments, but do not accumulate permanently in man. Ingested metallic mercury is not absorbed. The half-life of methylmercury, which is eliminated through the feces and urine, is 70 to 74 d; the biological half-life of mercury is 5 wk.[12]

The World Health Organization (WHO) recommends a drinking water standard of 1 μg/L.[24] The EPA recommends a drinking water standard of 2 μg/L and its EPC for air is 0.1 ppm by volume. In 1977, the ACGIH set a time-weighted average of 0.05 μg/m^3 for all forms of mercury except alkylmercury.

3.1.1.7 Manganese

Present in streams: 27, 38, 43, 46, 48, 50, 52, 53, 69, 72.

Waste stream types: liquid, solid, and gas.

Chronic exposure to manganese causes a wide range of toxic effects. Industrial manganese exposure has been correlated with respiratory dysfunction[25] and manganese salts are mutagenic. The strong oxidizing properties of manganese can irritate the skin. The lowest toxic concentration in humans is reported to be 11 mg/m^3 by inhalation, which affects the central nervous

system.[12] Manganese compounds may catalyze the oxidation of some air pollutants, producing more-undesirable pollutants. The OSHA standard is a ceiling limit of 5 mg/m^3.

3.1.1.8 Nickel

Present in streams: 27, 38, 43, 46, 48, 50, 52, 53, 54, 31, 34, 36, 69, 72.

Waste stream types: liquid, solid, and gas.

Exposure to nickel is usually by inhalation or dermal absorption. The various salts of the metal are toxic, especially the volatile nickel carbonyl [$Ni(CO)_4$], which is formed when carbon monoxide contacts nickel and nickel alloys and is usually the result of fossil-fuel combustion. Exposure to nickel produces dermatitis.

Inhaled nickel carbonyl decomposes to metallic nickel, which deposits on the epithelium of the lung. This finely divided nickel is rapidly absorbed and damages the lung and brain. One major problem posed by nickel in the United States is nickel hypersensitivity. The metal can alter defense mechanisms against xenobiotic agents in the respiratory tract, leading to enhanced risk of respiratory tract infections.[5]

The TLV for soluble nickel compounds (as Ni) is 0.1 mg/m^3, based on a time-weighted average, with a short-term exposure level of 0.3 mg/m^3. A time-weighted average of 1.0 mg/m^3 for metallic nickel was adopted by ACGIH in 1979.

3.1.1.9 Vanadium

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 72.

Waste stream types: liquid and gas.

Vanadium is toxic by all routes of exposure, with the pentavalent compounds exhibiting the highest degree of toxicity. Inhalation causes respiratory effects including tracheitis, bronchitis, pulmonary edema, and bronchial pneumonia.[12] Exposure of skin and mucous membranes causes dermatitis and conjunctivitis. Acute and chronic respiratory dysfunction has occurred in maintenance workers who clean fossil-fuel plants.

Studies have revealed that pharmacological levels of vanadium can affect tissue cholesterol levels.[26] Vanadium may also have a catalytic or enzymatic function in hard-tissue metabolism or formation. The OSHA standard for vanadium fume is a ceiling limit of 100 mg/m^3 and, for vanadium dust, a ceiling limit of 500 mg/m^3.[28]

3.1.1.10 Selenium

May be present in streams: 69, 79, 72.

Waste stream types: solid and gas.

Selenium poisoning in humans produces symptoms similar to those of arsenic. Chronic exposure may induce gastrointestinal disturbance, anemia, and damage to the nervous system, liver, and spleen. Normal human intake is estimated at 50-150 mg per day.[27] Selenium produces tumors in animals and is among the cancer-producing agents prohibited by the Delaney clause in the Food Additive Amendment of 1958.

Levels of selenium in municipal and community air range from 0.0025 $\mu g/m^3$ to 0.0097 mg/m^3.[5] The TLV for selenium is 0.2 mg/m^3, set as a time-weighted average for a normal 40-h/workweek. The drinking water standard is 0.01 mg/L and the EPC for air is 0.5 mg/m^3. Selenium is included in the EPA Consent Decree List as Priority 3.

3.1.1.11 Cobalt

May be present in streams: 79, 72.

Waste stream types: solid and gas.

Cobalt is an essential trace element associated with vitamin B_{12} in animals. Pulmonary effects, an allergic-type dermatitis, digestive changes, and liver and kidney damage have resulted from exposure to cobalt and its compounds.[12] Cobalt and its salts accumulate in food chains. The current TLV for cobalt (metal fume and dust) is 0.1 mg/m^3; ACGIH has issued a Notice of Intended Change to 0.05 mg/m^3. The EPC for air is 0.12 mg/m^3.

3.1.1.12 Copper

May be present in streams: 69, 72.

Waste stream types: solid and gas.

Copper is an essential trace element for humans as well as other biota. Although copper poisoning in humans is rare, ingestion of milligram quantities of ionic copper (usually from acidic waters or foods exposed to copper) can cause acute symptoms of nausea, vomiting, and diarrhea.[5] Chronic exposure may result in anemia. Exposure to metallic copper fume may cause respiratory, eye, and skin irritation. Damage to the liver, kidneys, and nervous system may result from acute exposure to copper.[15]

The TLV for metallic copper fume is 0.2 mg/m^3; for copper dust and mist it is 1 mg/m^3. Copper is included on the EPA Consent Decree List as Priority 3. The EPC for air is 0.5 $\mu g/m^3$.

3.1.1.13 Molybdenum

May be present in stream: 79.

Waste stream type: solid.

Molybdenum exhibits a low order of toxicity for exposed workers. Signs of molybdenum poisoning include loss of appetite, listlessness, diarrhea, and reduced growth rate.[12] The TLV for soluble molybdenum compounds is 5 mg/m^3; for insoluble compounds, 10 mg/m^3.

3.1.1.14 Iron

May be present in stream: 69.

Waste stream type: solid.

Iron has long been recognized as a nutritionally essential element. It is not likely to be present in any toxic concentration in effluent streams.

3.1.1.15 Sulfur

Sulfur may be present as a trace element in certain effluent streams. Chronic inhalation of sulfur dust can irritate mucous membranes. Sulfur and sulfide gases, on the other hand, such as sulfur dioxide, hydrogen sulfide, and carbonyl sulfide, may pose significant public health hazards and are discussed in Sec. 3.1.12.

3.1.1.16 Zinc

May be present in streams: 34, 36, 62.

Waste stream type: solid.

Zinc occurs in nature as a sulfide, oxide, or carbonate. It readily reacts with mineral acids and also reacts with alkali hydroxides to form alkali zincates. In humans, zinc ingestion produces no clinical symptoms at doses of 150 mg/d for as long as 6 mo.[29]

Inhalation of fumes can cause metal fume fever with symptoms of fever, nausea, vomiting, aching, cough, and weakness. Zinc fumes, if present in effluent streams, will not likely pose a health hazard to the general population. The TLV for zinc oxide fume is 5 mg/m^3; for zinc oxide dust, as a nuisance particulate, 10 mg/m^3. Zinc is included in the EPA Consent Decree List as Priority 3.

3.1.1.17 Aluminum

May be present in stream: 69.

Waste stream type: solid.

Aluminum is readily oxidized and is soluble in acid or alkali. Although not a highly toxic element, aluminum in large quantities may produce deleterious effects, such as pulmonary fibrosis from inhalation of aluminum powder.[12]

Aluminum has been reported to concentrate 10,000 times ambient levels in fish muscle, and 15,000 times in benthic algae.[12] The TLV for aluminum oxide is 10 mg/m^3.

3.1.2 Polynuclear Aromatic Hydrocarbons

The polynuclear aromatic hydrocarbons (PAHs) produced and emitted during coal conversion are important because several are known animal carcinogens. Among these are compounds such as benzo(a)pyrene and benz(a)anthracene. Polynuclear aromatic hydrocarbons are a diverse class of compounds that consist of substituted and unsubstituted polycyclic aromatic rings. They form as a result of incomplete combustion of organic compounds due to insufficient oxygen. The less efficient the combustion, the higher the PAH emission. Major sources of emission include power plants, refuse burning, and coking. Because of the many sources, most people are exposed to low levels of PAHs.

Exposure may be through skin contact, inhalation, or ingestion (although PAHs are poorly absorbed from the gastrointestinal tract). Regardless of their route of entry, the hepatobiliary system and the gastrointestinal tract are the main routes of elimination of both PAHs and their metabolites.

In the body, PAHs are metabolized to epoxides, dihydrodiols, phenols, and quinones. Several metabolites have been found to be more mutagenic, carcinogenic and teratogenic than their parent compounds. The enzyme responsible for PAH metabolism is arylhydrocarbon hydroxylase. Recent evidence indicates a relationship between inducibility of this enzyme in human lymphocytes and susceptibility to lung cancer.[18]

No published studies adequately compare the carcinogenic activity of the various PAHs under similar experimental conditions. Likewise, no data are available on human responses to individual compounds in the PAH class, since environmental exposure involves contact with complex, and usually unidentified, PAH mixtures.

The following discussion briefly describes: (1) the PAH compounds identified or believed to be present in Lurgi/Fischer-Tropsch effluent streams; (2) the stream numbers in which each have been identified; (3) current health data if available; and (4) existing or proposed standards, guidelines, or regulations. Few exposure standards for PAHs have been developed, either individually or as a class. A federal standard has been promulgated for coke-oven emissions, based primarily on the presumed carcinogenic effects of PAHs in the mixture and measured by the benzene-soluble fraction of total particulate matter.[12] The ACGIH recommends a workplace exposure limit for coal-tar-pitch volatiles of 0.2 mg/m^3. Its limit is based on the unstable composition of volatiles from coal tar pitch (which may contain both low- and high-molecular-weight PAHs), and on the assumption that high-molecular-weight PAHs have greater potential for carcinogenic behavior.

3.1.2.1 Acenaphthene

Present in streams: 43, 46, 50, 52, 53, 54, 31, 70, 72.

Waste stream types: liquid, solid, and gas.

Virtually no incidences of human acenaphthene toxicity have been reported. No bioaccumulation data are available. Laboratory experimentation, however, points out the possibility of limited metabolism of acenaphthene to potentially toxic naphthalic acid and naphthalic anhydride. Naphthalic anhydride appears to be metabolically inert.[30] No guidelines have been established for acenaphthene exposure.

3.1.2.2 Acridine

Present in stream: 72.

Waste stream type: gas.

Acridine is chemically classified as a nitrogen heterocyclic but is closely associated with particulate PAH. Acridine, which is present in soot, coal tar, and pitch, irritates the skin and mucous membranes. There is no evidence that acridine alone is carcinogenic to man or animals. The TLV for coal-tar-pitch volatiles is 0.2 mg/m^3.

3.1.2.3 Anthracene

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 70, 72, 76.

Waste stream types: liquid, solid, and gas.

Acute toxic properties of anthracene are unknown. Anthracene is on the NIOSH list of suspected carcinogens although other sources consider it inactive as a carcinogen.[31] The TLV for coal-tar-pitch volatiles is 0.2 mg/m^3.

3.1.2.4 Benz(a)anthracene (1,2 Benzanthracene)

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 70.

Waste stream types: liquid and solid.

Benz(a)anthracene is slightly carcinogenic, but its 9,10-dimethyl derivative is a definite rapid-acting carcinogen.[30] Both compounds, however, are metabolized in rats to the corresponding 4-hydroxy derivatives, which can be detected in animal feces. The TLV of 0.2 mg/m^3 for particulate PAHs applies to benz(a)anthracene.

3.1.2.5 Benzo(g,h,i)perylene

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 70.

Waste stream types: liquid and solid.

Benzo(g,h,i)perylene occurs in tar and in smoke-polluted atmospheres, but the urban concentration for this chemical alone is unavailable. It probably is not a highly active carcinogen.

3.1.2.6 Benzo(a)pyrene

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 70.

Waste stream types: liquid and solid.

Benzo(a)pyrene is the most widely recognized and extensively studied of all carcinogenic PAHs.[32] It is among the most potent animal carcinogens known, producing tumors in all species by all routes of administration. Toxicity data indicate that, as a class, PAHs can cross epithelial membranes. Accordingly, benzo(a)pyrene is transported readily across the intestinal mucosa and the respiratory membranes. It exhibits high lipid solubility and localizes primarily in body fat and fatty tissues.

Benzo(a)pyrene, like other PAHs, is metabolized by the microsomal mixed-function hydroxylase enzymes in mammals. One or more of the aromatic rings are metabolized to phenols and isomeric dihydrodiols by the intermediate formation of reactive epoxides. Dihydrodiols are further metabolized by microsomal mixed-function oxidases to yield diol epoxides, compounds which are known to be the ultimate carcinogen for certain PAHs.[33]

Generally, benzo(a)pyrene and related PAHs are rapidly excreted in the feces. This class of compounds is not likely to accumulate in the body as a result of chronic low-level exposure. The chemical half-life of benzo(a)-pyrene in the atmosphere is less than one day when exposed to solar radiation and several days without solar radiation.[12] The TLV for particulate PAHs, including benzo(a)pyrene, is 0.2 mg/m^3. The TLV takes into account the collective carcinogenic potential of the PAHs.

3.1.2.7 Benzo(e)pyrene

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 70.

Waste stream types: liquid and solid.

Benzo(e)pyrene is included on the NIOSH list of suspected carcinogens. The lowest dose to induce an oncogenic response is reported as 140 mg/kg. No TLV currently exists, pending determination of acceptable exposure levels.

3.1.2.8 Chrysene

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 70, 72.

Waste stream types: liquid, solid, and gas.

Chrysene is included in the NIOSH list of suspected carcinogens. The lowest dose to produce an oncogenic response is reported as 99 mg/kg.

On the EPA Consent Decree List it has an assigned priority of 1 and it is included in the TLV for particulate PAHs, 0.2 mg/m^3.

3.1.2.9 Fluoranthene

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 70.

Waste stream types: liquid and solid.

No evidence indicates that fluoranthene alone is carcinogenic. However, topical application of fluoranthene in an acetone mixture with phenanthrene, anthracene, pyrene, chrysene, benzo(a)pyrene, benzo(e)pyrene, and perylene induced tumors in 225 mice.[12] The TLV is 0.2 mg/m^3 (for particulate PAH). Fluoranthene appears on the EPA Consent Decree List with a priority of 2.

3.1.2.10 Fluorene

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 70, 72, 75, 76.

Waste stream types: liquid, solid, and gas.

No evidence indicates that fluorene alone is carcinogenic, although its 2-acetamido derivative is a potent carcinogen.

3.1.2.11 Indene

May be present in stream: 72.

Waste stream type: gas.

Serious systemic responses may result from inhalation of high concentrations of indene.[12] It is not highly toxic[30] and the epoxide is inactive as a skin carcinogen. The TLV for indene is 45 mg/m^3 (10 ppm).

3.1.2.12 Naphthalene (tar camphor, naphthaline, naphthene)

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 70, 72, 75, 76.

Waste stream types: liquid, solid, and gas.

The toxic effects of naphthalene include injury to the eye and irritation of the mucous membranes in concentrations of 15 ppm. It is inactive as a carcinogen and is moderately toxic to aquatic organisms. The TLV is 50 mg/m^3 (10 ppm); napthalene appears on the EPA Consent Decree List with a priority of 2.

3.1.2.13 Perylene

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 72.

Waste stream types: liquid and gas.

Exposure to perylene produces no oncogenic response in animals or humans.

3.1.2.14 Phenanthrene

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 70, 72, 77.

Waste stream types: liquid, solid, and gas.

Phenanthrene is among the lower-molecular-weight polycyclic hydrocarbons that make up the volatile, benzene-soluble fraction of coal tar. Phenanthrene is on the NIOSH list of suspected carcinogens. The lowest dose to induce an oncogenic response is reported as 71 mg/kg. The TLV for coal-tar-pitch volatiles is 0.2 mg/m^3.

3.1.2.15 Pyrene

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 70, 72, 76.

Waste stream types: liquid, solid, and gas.

Although not considered a highly active carcinogen alone, pyrene is included on the NIOSH list of suspected carcinogens. The lowest dose to produce an oncogenic response is 10 $\mu g/kg$. Pyrene has an assigned priority of 1 on the EPA Consent Decree List. The TLV (for particulate PAH) is 0.2 mg/m^3.

3.1.3 Nitrogen Heterocyclics

Nitrogen heterocyclics, or heterocyclic amines, are solids and liquids of moderate to high boiling point. Their significance as hazards in coal gasification is uncertain. Pyridines, methylpyridines and quinolines may act as potentiators of carcinogens rather than as direct toxicants. The one-ring pyridine and its derivatives are mildly irritant and narcotic; symptoms are reported from exposure to 10 ppm. Chronic poisoning may damage liver, kidneys, and bone marrow. Pyridines, picolines, and collidines are photolytically active. Picolines irritate the eyes and the respiratory tract. Collidines, found in low-temperature tar, may act as local irritants. Little information is available on the toxicity of quinoline, another mild irritant.

The TLV for pyridine is 15 mg/m^3 (5 ppm). There are currently no standards for picolines, collidines, or quinolines, but the EPCs for these compounds in air are:[12] picolines, 64 $\mu g/m^3$; collidines, 125 $\mu g/m^3$; and quinolines, 28.4 $\mu g/m^3$.

Nitrogen heterocyclics identified in Lurgi/Fischer-Tropsch waste streams include the following:

2,4-Dimethylpyridine

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31.

Waste stream types: liquid and gas.

2,5-Dimethylpyridine

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31.

Waste stream types: liquid and gas.

2-Methylpyridine

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31.

Waste stream types: liquid and gas.

3-Methylpyridine

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31.

Waste stream types: liquid and gas.

4-Methylpyridine

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31.

Waste stream types: liquid and gas.

Pyridine

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31.

Waste stream types: liquid and gas.

Quinoline

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31.

Waste stream types: liquid and gas.

3.1.4 Sulfur Heterocyclics (Thiophenes)

The sulfur heterocyclics methylthiophene and thiophene were identified in stream no. 72 in small quantities, and benzothiophene may be present in stream no. 75. The toxic effects and metabolism of thiophene and methylthiophene are unknown. Pyrolysis of thiophene at 800°F to 825°F yields methane, hydrogen sulfide, and hydrogen as the only gaseous products. The EPC in air is 8 $\mu g/m^3$ for thiophene and 4 $\mu g/m^3$ for methylthiophene.[12]

3.1.5 Oxygen Heterocyclics

Benzofuran is believed to be present in stream no. 72. Dibenzofuran (diphenylene oxide) was identified in stream nos. 43, 46, 48, 50, 52, 53, 54, and 31 and is believed to be present in stream nos. 72, 75, and 76. Mutagenic and other toxic effects are associated with oxygen heterocyclics.[23]

3.1.6 Phenols

In inhalation studies with mice, Tye and Stemmer found that coal tars with phenols removed were less potent carcinogens than coal tars containing phenols.[34] They attributed the cocarcinogenic potential of phenols to their irritant properties. Phenols and related aromatic hydroxy compounds are absorbed mainly through the skin, although some are sufficiently volatile to be possible respiratory hazards. In the animal body, phenols undergo two main reactions: conjugation of the hydroxyl group with glucuronic acid to form aryl glucosiduronic acids, and conjugation with sulfuric acid to form ethereal sulfates or monoesters of sulfuric acid.

3.1.6.1 Monohydric Phenols

Phenol is absorbed through the skin and the gastrointestinal and respiratory tracts. Chronic poisoning follows prolonged exposures to low concentrations of vapor or mist and results in digestive disturbances, nervous disorders, skin erruptions, and liver and kidney damage. Because phenol is a potent skin irritant and sensitizer, it is an industrial concern as a cause of dermatitis. The metabolites of phenol are known to be phenylglucuronide, phenylsulfuric acid, conjugated catechol, and quinol. Phenol is a normal constituent of human urine and is usually present in amounts up to 10 mg/L/24 h. Experiments with Drosophila (fruit fly) show that phenol is highly mutagenic.

The TLV for phenol is 19 mg/m^3 (5 ppm). It appears on the EPA Consent Decree List with a priority of 3, and NIOSH recommends exposure to phenol vapor, solid, or mist be limited to 20 mg/m^3 as a time-weighted average for a 10-h workday. The EPC for phenol in air is 45 $\mu g/m^3$ (0.01 ppm).

O-Isopropylphenol

Present in stream: 72.

Waste stream type: gas.

3.1.6.3 Cresols

Cresols do not present an acute inhalation hazard due to their disagreeable odor and low vapor pressure. They resist biological oxidation, are dermally absorbed, and damage the liver, kidneys, and nervous system. The TLV for all isomers of cresol is 22 mg/m^3 (5 ppm). The EPC for cresol in air is 52 $\mu g/m^3$ (0.1 ppm).

2-Methylphenol

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 72, 77.

Waste stream types: liquid and gas.

3-Methylphenol

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31.

Waste stream types: liquid and gas.

4-Methylphenol

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31.

Waste stream types: liquid and gas.

Trimethylphenol

Present in stream: 72.

Waste stream type: gas.

3.1.6.4 Dihydric Phenols

Dihydric phenols are of interest since many of their derivatives have been used as antiseptics. The metabolism of dihydric phenols is similar to that of phenol. Their main metabolites are monoglucuronides. In toxic doses, catechols cause convulsions and injure the blood. Resorcinol acts primarily as a skin irritant and can cause systemic blood and nerve poisoning. Symptoms include dermatitis and eye injury, particularly in sensitive individuals. The TLVs for catechol and resorcinol are 20 mg/m^3 (5 ppm) and 45 mg/m^3 (10 ppm), respectively. The EPCs for the compounds are set at 48 $\mu g/m^3$ and 110 $\mu g/m^3$, respectively.

3-Methylcatechol

Present in streams: 43, 46, 48, 50.

Waste stream type: liquid.

4-Methylcatechol

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 77.

Waste stream types: liquid and gas.

3,6-Dimethylcatechol

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31.

Waste stream type: liquid.

Resorcinol

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 77.

Waste stream types: liquid and gas.

5-Methylresorcinol

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 77.

Waste stream types: liquid and gas.

4-Methylresorcinol

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 77.

Waste stream types: liquid and gas.

3.1.6.5 Trihydric Phenols

Phloroglucinol (1,3,5,-benzene triol) may be present in gas from stream no. 72. Evidence suggests that trihydric phenols behave as typical phenols and undergo conjugation with glucuronic and sulfuric acids.

3.1.7 Mercaptans

3.1.7.1 Methanethiol (Methylmercaptan)

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 72.

Waste stream types: liquid and gas.

The toxicity of methanethiol is closely related to that of hydrogen sulfide. Methanethiol, like hydrogen sulfide, acts on the respiratory center to produce death by respiratory paralysis. At lower concentrations, it may cause pulmonary edema. Methanethiol has a strong, unpleasant odor and emits highly toxic fumes of sulfur oxides when oxidized. The TLV for methanethiol is 1 mg/m^3 (0.5 ppm) and the EPC in air is 2.4 $\mu g/m^3$ (0.001 ppm).

3.1.7.2 Ethanethiol (Ethylmercaptan)

May be present in stream: 72.

Waste stream type: gas.

Ethanethiol's odor is so intense that the odor threshold is reported at 1 part per billion (ppb). Animals exposed to single doses of ethanethiol by several routes indicate only slight acute toxicity; 4 ppm is the lowest toxic dose in humans. Ethanethiol reacts with water, steam, acid, or heat to produce toxic and flammable vapors. The TLV is 1 mg/m^3 (0.5 ppm), based on intense odor. The EPC in air, based on health effects, is 2.4 $\mu g/m^3$ (0.001 ppm).

3.1.8 Aromatic Amines

The toxicity of aromatic amines is considered by most authorities to be a more serious hazard than their flammability. They are poisonous by inhalation, ingestion, and by rapid absorption through skin. Aromatic amines can be absorbed when they are pure or part of a mixture, such as a dye. For example, infants have been poisoned by diaper laundry marks made with aniline-containing inks. The manufacture of aromatic amines usually entails health hazards.

3.1.8.1 Aniline (Phenylamine, Aminobenzene)

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 72.

Waste stream types: liquid and gas.

Aniline is a liquid, similar to ammonia, that impairs the oxygen-transporting ability of hemoglobin. Ingestion of 350 mg/kg has resulted in human death; chronic poisoning may damage the central nervous system. Aniline is oxidized in man and animals to *p*-aminophenol and *o*-aminophenol, which are then excreted. The biological half-life in man is 0.120 d.

Aniline has not been shown to be carcinogenic in man, although several aniline derivatives have produced oncogenic responses in test animals. The TLV for aniline is 19 mg/m^3 (5 ppm). The EPC in air is 45 μg/m^3 (0.02 ppm).

3.1.8.2 Methylaniline (Toluidines, Aminotoluidines)

Present in stream: 72.

Waste stream type: gas.

3.1.8.3 Dimethylaniline

Present in stream: 72.

Waste stream type: gas.

The toxicity of methylanilines, or toluidines, is similar to that of aniline. Workers handling toluidines and chlorotoluidines may experience hematuria and hemorrhagic cystitis with painful and frequent urination. These are symptoms of acute exposure and have no long-term significance if exposure ceases.[35] The lowest dose to produce an oncogenic response in animals is reported as 6,600 mg/kg. The TLV is 22 mg/m^3 (5 ppm) for 2-aminotoluene; the EPC is 45 μg/m^3 (0.01 ppm).

3.1.9 Benzene and Substituted Benzenes

3.1.9.1 Ethylbenzene

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 72.

Waste stream types: liquid and gas.

Relative to benzene, ethylbenzene is much less toxic but is a more severe skin irritant. A concentration of 200 ppm irritates mucous membranes and eyes and high concentrations are narcotic. It is absorbed dermally and through respiratory passageways. Ethylbenzene is metabolized by the liver and excreted by the kidneys. Consequently, these tissues may be susceptible to injury from ethylbenzene exposure. Chronic inhalation may exacerbate lung disease.[36]

The TLV for ethylbenzene is 435 mg/m^3 (100 ppm). The ambient atmosphere contains approximately 0.01 ppm,[5] and ethylbenzene has not been reported in food or potable waters. The EPC in air is 1,040 μg/m^3 (0.24 ppm).

3.1.9.2 Toluene

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 72, 75.

Waste stream types: liquid and gas.

Toluene may be inhaled, ingested, or dermally absorbed. It affects the central nervous system, liver, kidneys, and skin. Chronic inhalation of toluene vapor may cause decreased phagocytic activity of leukocytes, depression of the central nervous system, narcosis, addiction, and death. Most exposures to toluene are occupational or are due to deliberate inhalation for the compound's intoxicant effect.

Two major metabolites of toluene are benzaldehyde and benzoic acid. These substances occur naturally in foods and are intentionally added to commercially processed foods. Benzaldehyde is a flavoring agent; benzoic acid is a preservative.

In the atmosphere, toluene is photochemically degraded to benzaldehyde and traces of peroxybenzoyl nitrate. Toluene can reenter the hydrosphere in rain. Its biological half-life is 0.083 d.[12]

The TLV for toluene exposure is 100 ppm (375 mg/m^3), with a ceiling limit of 200 ppm.[28] This standard was based on subjective and objective signs of mucous membrane irritation and impairment of the central nervous system in human subjects who inhaled up to 200 ppm. The EPC for toluene is 890 $\mu g/m^3$.

3.1.9.4 O-Xylene

Present in streams: 43, 46, 48, 50, 51, 53, 54, 31, 72, 75.

Waste stream types: liquid and gas.

The effects of xylenes are similar to those of toluene, although xylene is more toxic. Effects of chronic toxicity are not well known. The TLV for xylene is 435 mg/m^3 (100 ppm). The EPC for xylene is 1 mg/m^3.

3.1.9.5 Biphenyl

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 72.

Waste stream types: liquid and gas.

Biphenyl exposure can irritate and injure respiratory passages; no chronic effects are documented. Inhalation of 4400 mg/m^3 caused irritation in a human.[7] The TLV is 1 mg/m^3 (0.2 ppm).

3.1.9.6 Indan

Present in streams: 43, 46, 50, 52, 53, 54, 31, 72, 76.

Waste stream types: liquid and gas.

No TLV currently exists for indan.

3.1.10 Aliphatics, Alicyclics, and Fatty Acids (Carboxylic Acids)

Alicyclic acids are characterized by the presence of at least one aliphatic cyclic skeleton with 3, 4, 5, 6, or more carbon atoms in the ring. Aliphatic acids react with chlorine or bromine in the presence of sunlight to form halogen-substituted acids. From a toxicological point of view, aliphatic compounds are not particularly active.

Aliphatics, alicyclics, and fatty acids are weak, organic acids and are usually emitted in large quantities to the atmosphere from coal-fired boiler plants. Monocarboxylic acids, such as acetic, propanoic, and butanoic, are widely distributed in nature. Most monocarboxylic acids are intermediates in normal metabolic pathways. Lower acids (one to five carbons) are found in many plant and animal fluids such as sap, perspiration, and urine. Intermediate and higher acids that contain an even number of carbon atoms occur in a wide variety of fats, oils, and waxes. Aliphatics that have been identified in Lurgi effluent streams are not highly toxic.

Table 3.2 lists the carboxylic acids found in Lurgi waste streams, their toxic effects, and applicable standards.

Acetic Acid

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 72.

Waste stream types: liquid and gas.

Propanoic Acid

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 72.

Waste stream types: liquid and gas.

Butanoic Acid

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 72.

Waste stream types: liquid and gas.

2-Methylpropanoic Acid

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 72.

Waste stream types: liquid and gas.

Pentanoic Acid

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 72.

Waste stream types: liquid and gas.

Table 3.2 Summary of Aliphatics, Alicyclics, and Fatty Acids Found in Lurgi/Fischer-Tropsch Waste Streams

Effluent	Toxic Effect	Standard or Classification[a]
Acetic acid	mild skin irritant	TLV = 10 ppm[37]
Propanoic acid	-	DOT:[b] Corrosive Material
Butanoic acid	-	DOT: Corrosive Material
Pentanoic acid	-	DOT: Corrosive Material
Hexanoic acid	-	DOT: Corrosive Material
2-Methylpropanoic acid	-	DOT: Corrosive Material.
3-Methylbutanoic acid	-	DOT: Corrosive Material
Pentane	central nervous system impairment [$LC_{1.0}$: 130,000 ppm] [$TC_{1.0}$: 90,000 ppm][c]	OSHA standard in air: 1,000 ppm (time-weighted average) DOT: Flammable Liquid NIOSH: Recommended Standard: 350 mg/m^3; Ceiling: 1800 mg/m^3 for 15 min
Methane	-	DOT: Flammable Gas
Ethane	-	DOT: Flammable Gas

[a]As reported in the EPA Toxic Substances Control Act Inventory in July 1979.

[b]DOT = U.S. Department of Transportation.

[c]$LC_{1.0}$ = lethal concentration in 1% of test population; $TC_{1.0}$ = toxic concentration in 1% of test population.

3-Methylbutanoic Acid

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 72.

Waste stream types: liquid and gas.

Hexanoic Acid

Present in streams: 43, 46, 48, 50, 52, 53, 54, 31, 72.

Waste stream types: liquid and gas.

Ethane

Present in stream: 72.

Waste stream type: gas.

Methane

Present in stream: 72.

Waste stream type: gas.

Pentane

Present in stream: 72.

Waste stream type: gas.

C_7^+ Hydrocarbons

Present in stream: 73.

Waste stream type: gas.

3.1.11 Nitrosamines

N-nitrosamines, which have been identified in stream no. 72 (lockhopper vent gas), are widespread in the environment, with man-made sources contributing a negligible amount. Carcinogenic and toxic properties of these compounds are well established. Seventy percent of all N-nitrosamines studied are carcinogenic in various laboratory animals, including aquatic organisms.[5] They induce tumors by all routes of administration in essentially all vital organs.[38] In the environment, nitrosamines are rapidly decomposed by photolysis and do not persist for a significant time in water exposed to sunlight.

The ACGIH states that, because of the extremely high toxicity and presumed carcinogenic potential of nitrosodimethylamine, contact should not be permitted by any route. In the Federal Register, EPA said it is considering an interim target risk level of 10^{-5} mg/L, 10^{-6} mg/L, or 10^{-7} mg/L.[5]

3.1.12 Gases

3.1.12.1 Carbon Monoxide

Present in stream: 28.

Carbon monoxide is a tasteless, odorless gas which, when absorbed, combines with hemoglobin and interferes with oxygenation of blood. Its affinity for hemoglobin is about 200-250 times that of oxygen.[12] Exposure to high concentrations of carbon monoxide impairs cardiovascular, brain, and skeletal-muscle function by compromising oxygen transport and delivery. This discussion, however, is limited to the toxic effects of low levels of carbon monoxide and to standards for ambient levels.

Exposure to low levels of carbon monoxide decreases exercise tolerance in patients with arteriosclerosis. Even in normal subjects, the ability to

exercise maximally appears to be limited by exposure to relatively low concentrations. Auditory and visual disturbances have been demonstrated at low concentrations. Repeated exposure to concentrations up to 100 ppm in air or a 1-h exposure to 400-500 ppm may be safely tolerated.[12] However, exposure to 650 ppm for 45 min caused toxic effects in man.[12]

The degree of harm from exposure to carbon monoxide is a product of concentration and length of exposure.[12] The public health effects of carbon monoxide are difficult to determine, however, since factors such as cigarette smoking, exercise, occupation, initial cardiovascular disease, and body burden must be taken into account.

Carbon monoxide is a product of incomplete combustion. Utility plants and coal-conversion facilities are not likely to contribute significantly to atmospheric carbon monoxide since combustion at these sites is usually efficient (excluding start-ups and shutdowns). Emissions of carbon monoxide, sulfur oxides, and nitrogen oxides from utility plants are usually within the same order of magnitude, and sulfur oxide and nitrogen oxide emissions are usually higher.

The standards set for carbon monoxide are less stringent than the standards for nitrogen and sulfur oxides. Therefore, if the carbon monoxide standard were violated, the nitrogen and sulfur oxide standards probably also would be violated. The National Primary and Secondary Ambient Air Quality Standard for carbon monoxide is 9 ppm maximum for 8-h average exposure or 35 ppm maximum for 1-h average exposure. The OSHA standard is 55 mg/m^3 (50 ppm).

3.1.12.2 Nitrogen Oxides

Present in stream: 28.

The principal man-made source of nitrogen oxides is combustion of coal, oil, natural gas, and motor-vehicle fuel. Of the seven oxides of nitrogen known to exist in ambient air, only two, nitric oxide and nitrogen dioxide, are thought to affect human health. Nitric oxide at its current ambient level has not been shown to be a health hazard. If nitric oxide is vented to the environment and cooled rapidly, a fraction is converted to nitrogen dioxide. Further complex reactions in the presence of hydrocarbons and sunlight form ozone, nitrogen dioxide, and other oxidants commonly known as photochemical smog.

Ozone and nitrogen dioxide are both human health hazards. Ozone is a strong irritant to the eyes and upper respiratory system. A concentration of 1 ppm has a disagreeable sulfur-like odor and may cause headaches and irritation of the upper respiratory tract. The TLV for ozone is 0.1 ppm (0.2 mg/m^3). The EPA National Primary and Secondary Ambient Air Quality Standard for photochemical oxidants, which include ozone, is 160 mg/m^3 (0.08 ppm), 1-h average.

Nitrogen dioxide oxidizes in the tracheobronchial tree and lungs to nitrous acid and nitric acid, which are both highly corrosive and irritate the mucous membranes of the lung. Nitrogen dioxide can penetrate the lung deeply without immediate severe discomfort and induce a delayed pulmonary

edema. Its association with carcinogenesis is a possible, but unproved, hazard.[23] Several epidemiological studies have linked high nitrogen-dioxide exposure to an increase in childhood respiratory illness and to a decrease in ventilatory performance.[39-41]

The EPA Ambient Air Quality Standard for nitrogen dioxide is 0.05 ppm or 100 $\mu g/m^3$. The TLV recommended by the ACGIH is 9 mg/m^3 (5 ppm) for nitrogen dioxide and 30 mg/m^3 (25 ppm) for nitric oxide.

3.1.12.3 Sulfur Oxides

Sulfur oxides in the air occur in three forms: sulfur dioxide, sulfuric acid, and inorganic sulfates. The major source of urban sulfur oxides is the combustion of fossil fuels. About 98% of sulfur released to the air is in the form of sulfur dioxide. Sulfur dioxide is slowly oxidized by sunlight to sulfur trioxide, which combines readily with water to form sulfuric acid. Sulfuric acid and sulfates account for from 5% to 29% of the total suspended particulates in urban air.[42] Of the urban atmospheric sulfates, 80% or more are associated with respirable particles less than 2 μm in diameter.

Sulfur dioxide is a mild respiratory irritant that causes bronchial narrowing and increases air-flow resistance in man and animals. It penetrates the lungs primarily when it is adsorbed on the surface of a particle or converted to sulfate in an aerosol.

Elevated ambient levels of sulfur dioxide and other pollutants are associated with increased mortality and morbidity. Those most affected are individuals suffering from chronic pulmonary disease or cardiovascular disorders, the very young, and the elderly. Long-term effects include increased prevalence of chronic bronchitis in adults, increased acute lower respiratory infections in children, and subtle decreases in ventilatory function of children. Short-term effects include symptoms of asthma. The OSHA standard for sulfur dioxide is a time-weighted average of 5 ppm (13 mg/m^3).

3.1.12.4 Hydrogen Sulfide

Present in stream: 72.

Hydrogen sulfide is an acute, rapid-acting poison at concentrations of about 400 ppm. In lower concentrations (50-500 ppm), it acts primarily as a respiratory irritant. Other symptoms of hydrogen-sulfide toxicity include conjunctivitis, psychic changes, disturbed equilibrium, nerve paralysis, spasms, and unconsciousness.[43] The odor theshold for hydrogen sulfide is between 1 mg/m^3 and 24 mg/m^3, much below the toxic level, and it is easily recognized by its characteristic odor of rotten eggs. The biological half-life is less than 20 min.[12]

The TLV for hydrogen sulfide is 15 mg/m^3 (10 ppm). The EPC in air is 36 $\mu g/m^3$ (0.024 ppm).

3.1.12.5 Carbonyl Sulfide

Present in stream: 72.

Carbonyl sulfide gas is a slight respiratory irritant that exerts its principal effects on the central nervous system. It may be narcotic in high concentrations and it decomposes to hydrogen sulfide. Toxicologically, it resembles hydrogen sulfide and carbon disulfide, but few data are available on its human health effects. It is less toxic than hydrogen sulfide. The EPC is 800 $\mu g/m^3$.

3.1.12.6 Hydrogen Cyanide

Present in stream: 72.

Hydrogen cyanide is a chemical asphyxiant that inhibits oxygen metabolism. It is absorbed rapidly by inhalation; at 0.3 mg/L, hydrogen cyanide is immediately fatal to man.[12] Chronic effects include weakness, vertigo, nausea, rapid pulse, headache, flushing of the face, and gastric disturbances. Hydrogen cyanide vapor can also be dangerous if absorbed dermally. The TLV for hydrogen cyanide is 11 mg/m^3 (10 ppm). The EPC in air is 26 $\mu g/m^3$ (0.024 ppm).

3.1.12.7 Carbon Dioxide

Present in streams: 28, 72.

Carbon dioxide makes up about 0.03% by volume of dry air (300 ppm). Its concentration in exhaled human breath may be as high as 5.6%.[12] High levels of carbon dioxide may contribute to oxygen deficiency, but the compound is not toxic and does not present a public health concern. The TLV is 9,000 mg/m^3 (5,000 ppm).[12] The EPC is 539,000 $\mu g/m^3$ (300 ppm).[12]

3.1.12.8 Ammonia

Present in streams: 29, 72.

Ammonia irritates the eyes, mucous membranes, and upper respiratory tract at concentrations of 20 ppm. It is too strong an irritant to be voluntarily inhaled. Low-level exposure has no chronic effects in humans. Some terrestrial and aquatic species may be sensitive to ammonia. The biological half-life is less than 20 min.[18] The TLV for ammonia is 18 mg/m^3 (25 ppm) and the EPC in air is 43 $\mu g/m^3$ (0.06 ppm).

3.1.12.9 Nickel Carbonyl

Present in stream: 72.

Nickel carbonyl is extremely toxic. Symptoms from inhalation are believed to be caused by both nickel and carbon monoxide when liberated

from the lungs. Inhalation of $Ni(CO)_4$ vapor affects the central nervous system and may induce acute chemical pneumonitis. Brief exposure to 0.15 ppm causes headache and chronic exposure has been associated with cancer of the lung and nasal sinuses. The lowest dose producing a carcinogenic response is 157 mg/kg. The TLV for $Ni(CO)_4$ is 0.35 mg/m^3 (0.05 ppm). Nickel is included in the National Cancer Institute list of carcinogens to man.

4 RELATIVE RISK ASSESSMENT

4.1 CATEGORIZATION

Of the toxic substances found in Lurgi/Fischer-Tropsch waste streams, carcinogens and promoters of carcinogenesis are major concerns to public health and their presence may slow the progress of gasification technology. Federal acts such as the Toxic Substances Control Act (TSCA) and the Resource Conservation and Recovery Act (RCRA) have been set up to regulate toxic substances wherever there is potential for public exposure. Table 4.1 lists the potential human carcinogens that have been identified in Lurgi/Fischer-Tropsch waste streams. These substances are based on the EPA Carcinogen Assessment Group's list of potential human carcinogens. The group classified chemicals as carcinogens if substantial evidence showed that they met one of the following criteria:

- Substance induces malignant tumors in one or more animal species, or
- Substance induces benign tumors that are generally recognized as early stages of malignancies, or
- Positive epidemiological studies indicate carcinogenesis.

Table 4.1 Potential Human Carcinogens Identified in Lurgi/Fischer-Tropsch Process Waste Streams

Element or Compound	Stream
Arsenic and arsenic compounds	28,29,36,53,69,70,72
Benz(a)anthracene	29,53,70
Benzene	72
Benz(a)pyrene	29,53,70
Beryllium and beryllium compounds	28,29,36,53,70,72
Cadmium and cadmium compounds	28,29,36,53,69,70,72
Chromium and chromium compounds	28,36,72
Chrysene	29,53,70,72
Nickel and nickel compounds	28,29,36,53,69,72
Nickel carbonyl	28,72
N-nitrosamine	72
Selenium	28,36,69,72,79
2,4-Dimethylphenol (2,4-Xylenol)	29,53

Source: Ref. 44.

Table 4.2 lists nontoxic substances identified in Lurgi wastes. Although these chemicals are unlikely to pose health hazards, several, such as the organic acids and hydrogen gas, may present potential safety hazards. Safety hazards, which include flammability and explosivity, are not analyzed in this report.

Table 4.2 Nontoxic Substances Identified in Selected Lurgi/Fischer-Tropsch Process Waste Streams

Substances	Waste Stream
Propanoic acid	29,53,72
2-Methylpropanoic acid	29,53,72
Pentanoic acid	29,53,72
3-Methylbutanoic acid	29,53,72
Ethane	72
Methane	72
C_2-C_6 aliphatics	72
Boron	28,29,36,53,72
Manganese	28,36,53,69,72
Copper	36,69,72
Iron	69,72
Aluminum	69
Tin	28,36,72
Carbon dioxide	28,72
Nitrogen gas	28,72
Oxygen gas	28
Hydrogen gas	28,72
Water	28,36,72
Ethanol	60

As outlined in Sec. 2, the screened substances were divided into four categories, based on the degree of available information on emissions, effluents, and solid wastes. Category 1 includes toxic substances that have been quantified in waste streams from a Lurgi/Fischer-Tropsch reference plant. Because empirical data are not available for a plant that matches the specifications of the U.S. commercial-scale plant chosen as a reference to this study, Category 1 is empty. Category 2 includes the toxic residues for which potential releases have been estimated from a variety of data sources. These sources include operating Lurgi/Fischer-Tropsch plants in Westfield, Scotland, and Sasolburg, South Africa; the Western Gasification Co. environmental impact statement; and unpublished EPA data and bench-scale tests with Montana rosebud or Wyoming subbituminous coal. Category 2 substances are listed in Table 4.3.

Category 3 substances, listed in Table 4.4, are of special concern. These substances are expected to be present in emission streams, but have not been quantified. Several are highly toxic or carcinogenic. Category 3 also contains chemicals which, although quantified in some streams, may be present but unquantifiable in others. If present, these substances may pose a larger hazard than that indicated by analysis of quantifiable streams alone.

Category 4 residues are listed in Table 4.5. Information on these substances is insufficient to evaluate their potential toxic effect.

4.2 COMPARISON OF RELATIVE RISKS

4.2.1 Ambient Concentrations and Environmental Standards

Table 4.6 summarizes ambient atmospheric and aquatic concentrations of pollutants projected for areas surrounding Lurgi/Fischer-Tropsch facilities.

Table 4.3 Category 2 Toxic Substances Estimated in Lurgi/Fischer-Tropsch Waste Streams

Substance	Stream
Benzene, Substituted Benzenes	
Ethylbenzene	29,53
Toluene	29,53
O-Xylene	29,53
Monohydric Phenols	
Phenol	72,29,53
2-Methylphenol	29,53
3-Methylphenol	29,53
4-Methylphenol	29,53
2,4-Xylenol	29,53
3,5-Xylenol	29,53
Dihydric Phenols	
Catechol	29,53
4-Methylcatechol	29
3,6-Dimethylcatechol	29,53
Resorcinol	29,53
4-Methylresorcinol	29,53
5-Methylresorcinol	29,53
Polynuclear Aromatic Hydrocarbons	
Acenaphthylene	29,53,70
Anthracene	29,53,70
Benz(a)anthracene	29,53,70
Benz(g,h,i)perylene	29,53,70
Benzo(a)pyrene	29,53,70
Benzo(e)pyrene	29,53,70
Biphenyl	29,53
Chrysene	29,53,70
Fluoranthene	29,53,70
Fluorene	29,53,70
Naphthalene	29,53,70
Phenanthrene	29,53,70
Pyrene	29,53,70
Sulfur Heterocyclics	
Thiophene	72
Nitrogen Heterocyclics	
2,4-Dimethylpyridine	29,53
2,5-Dimethylpyridine	29,53
2-Methylpyridine	29,53
3-Methylpyridine	29,53
4-Methylpyridine	29,53
Pyridine	29,53
Oxygen Heterocyclics	
Dibenzofuran	29,53
Mercaptans	
Ethanethiol	72
Methanethiol	29,53
Aromatic Amines	
Aniline	53,72
Methylaniline	53,72
Dimethylaniline	72
Methylaniline	29
Nitrosamines	72
Trace Elements	28,36,72
Arsenic	28,29,53,36, 69,70,79
Beryllium	28,29,36,53, 70
Cadmium	28,29,36,53, 69,70
Cobalt	79
Fluorine	29,36,53
Lead	28,29,36,53, 69,70,79
Mercury	28,29,36,53, 69,70,79
Nickel	29,36,53,69
Selenium	69,79
Vanadium	29,53
Molybdenum	79
Sulfur	36,79
Zinc	36,69
Gases	
Sulfur oxides	28
Nitrogen oxides	28
Carbon monoxide	28,72
Hydrogen cyanide	72
Carbonyl sulfide	72
Ammonia	29,72
Hydrogen sulfide	72

Table 4.4 Category 3 Toxic Substances in Non-Quantified Lurgi/Fischer-Tropsch Waste Streams

Substance	Stream	Substance	Stream
Aliphatics, Alicyclics, Fatty Acids		Polynuclear Aromatic Hydrocarbons (Cont'd)	
C_3^+ Alcohols	60	Fluoranthene[a]	76,72
Benzene, Substituted Benzenes		Fluorene[a]	76,72
Benzene	72,75	Naphthalene[a]	75,76,72
Ethylbenzene[a]	72	Phenanthrene[a]	76,72
Toluene[a]	72,75	Pyrene[a]	76,72
Xylene[a]	72,75	Oxygen Heterocyclics	
Monohydric Phenols		Dibenzofuran[a]	76,72
Cresols[a]	72,75,76,77	Benzofuran	72
Phenol[a]	72,75,76,77	Nitrogen Heterocyclics	
Alkyl phenols[a]	72,75	Acridine	72
2-Methylphenol[a]	72	Trace Elements	
Trimethylphenol	72	Arsenic[a]	72
O-Isopropylphenol	72	Beryllium[a]	72
Xylenols[a]	72,77	Cadmium[a]	72
Dihydric Phenols		Barium	28,36,72
Catechol[a]	77	Cobalt	28,36,72
Methylcatechol[a]	77	Chromium	28,36,72
Resorcinol[a]	77	Fluorine[a]	28,72
Methylresorcinol[a]	77	Nickel[a]	28,72
Polynuclear Aromatic Hydrocarbons		Selenium[a]	28,36,72
Anthracene[a]	76	Vanadium[a]	28,36,72
Biphenyl[a]	72	Zinc	28,72
Chrysene[a]	72	Uranium	28,36,72
Indene	72	Silver	36,72
		Lead[a]	72
		Mercury[a]	72

[a]Also found in quantified waste streams.

In addition, it contains two sets of atmospheric and water quality standards for each pollutant, when such standards have been established. By comparing the projected concentrations with these standards, researchers can estimate the potential public health risk.

Projected concentrations of pollutants in Table 4.6 were generated by applying the EPA Source Analysis Model 1 (SAM 1). This generic model estimates ambient concentrations by averaging meteorological, geological, and hydrological data for the entire country and applying worst-case assumptions. Worst-case assumptions may include conditions that do not reflect real situations. For example, SAM 1 dilution factors for aqueous wastes and solid-waste

leachates do not account for natural cleansing processes such as biodegradation and attenuation that occur in soils. As a result, concentrations obtained from SAM 1 provide a conservative estimate of exposure conditions.

Table 4.5 Category 4 - Residuals of Unknown Toxicity Found in Waste Streams

Substance	Stream
Perylene	72
Methylthiophene	72
Benzothiophene	72

Standards used for comparison with projected concentrations include: National Ambient Air Quality Standards (NAAQS), Estimated Permissible Concentrations for Health (EPC) from EPA's Multimedia Environmental Goals, and EPA Drinking Water Standards. More stringent federal or state levels may apply to specific sites, but levels used in this assessment are valid for the generic siting dictated by SAM 1 dispersion factors.

National Ambient Air Quality Standards have been set for sulfur dioxide, nitrogen oxides, hydrocarbons, carbon monoxide, particulates, ozone, and lead. No ambient air standards have been set for other Lurgi/Fischer-Tropsch emissions. The EPA developed Multimedia Environmental Goals to determine acceptable ambient concentrations for other substances. The goals include several sets of acceptable concentrations that vary according to exposed human population. The set used in this assessment, Estimated Permissible Concentrations for Human Health, is based on nonhuman data or on occupational rather than public exposures. The EPA also developed drinking water standards that address many aquatic pollutants, including a number that are released in Lurgi/Fischer-Tropsch effluents. Drinking water standards and EPCs in this report serve as a baseline; any ambient concentration above these levels is considered hazardous.

In the analysis that follows, only ambient concentrations of substances in quantified Lurgi/Fischer-Tropsch streams are evaluated. Many elements and compounds, though known to be present in Lurgi wastes, have not been quantified. Thus not all substances with the potential to violate pollution standards may be identified.

Substances are classified according to the ratio of projected concentration to the appropriate standard or EPC. Three levels are distinguished by the relative severity of potential exposures. Indirectly, the levels also address the uncertainty of process and dispersion characterizations due to engineering and modeling factors. Level 1 substances have projected ambient concentrations that exceed acceptable levels by a factor of 10 or more. Substances at this level will provide the greatest quantifiable potential hazard to public health from Lurgi/Fischer-Tropsch liquefaction.

Level 2 substances have projected ambient concentrations within a factor of ten above or below the appropriate standard or EPC. The potential public hazard associated with this category is more uncertain than with Level 1.

Level 3 contains substances whose ambient concentrations are more than a factor of ten below the appropriate standard or EPC. These substances have

Table 4.6 Projected Ambient Atmospheric and Aquatic Concentrations of Pollutants from Lurgi/Fisher-Tropsch Waste Streams

Compound/Element	Estimated Ambient Atmospheric Levels ($\mu g/m^3$)				Acceptable Ambient Atmospheric Levels ($\mu g/m^3$)		Estimated Ambient Aquatic Levels (mg/L)			Acceptable Ambient Aquatic Levels (mg/L)	
	Utility Stack Gas	Cooling Tower Evaporation	Lockhopper Vent Gas	Total	EPC[a]	NAAQS[b]	Reverse Osmosis	Ash Leachate	Total	EPC[a]	DWS[c]
Trace Elements											
Aluminum								2.0E-2	2.0E-2	7.3E-2	
Arsenic	5.8E-4	1.3E-2		1.4E-2	5.0E-3		8.9E-2	2.0E-3	9.1E-2		5.0E-2
Beryllium	6.1E-4	6.5E-3		7.1E-3	8.0E-1		4.6E-2		4.6E-2	4E-3	
Boron		4.3E-2		4.3E-2	7.4E 0		3.1E-1		3.1E-1	4.3E-3	
Cadmium	5.1E-4	7.5E-3		8.0E-3	1.2E-1		5.3E-2	6.4E-5	5.4E-3		1.0E-2
Copper								3.0E-3	3.0E-3	1.0E 0	
Fluorine		3.1E-1		3.1E-1			3.3E 0		3.3E 0		
Iron								1.6E-1	1.6E-1		
Lead	6.0E-3	1.5E-2		2.1E-2	3.6E-1	1.5E 0	1.7E-2	9.3E-3	2.6E-2		5.0E-2
Manganese							8.8E-2		8.8E-2		
Mercury	3.9E-2	1.1E-3		4.0E-2	1E-1		8.1E-3	3.0E-5	8.1E-3		2.0E-2
Nickel		4.0E-3		4.0E-3	2.4E-1		1.5E-2	3.5E-3	1.9E-2	1.4E-3	
Selenium					5E-1			6.0E-3	6.0E-3		1.0E-2
Vanadium		1.1E-4		1.1E-4	1.2E 0		1.8E-3		1.8E-3	7.0E-3	
Total trace elements			2.5E-2	2.5E-2							
Gases											
Ammonia		1.9E+1	9.8E+1	1.2E+2	4.3E+1						
Carbonyl sulfide			1.4E 0	1.4E 0	8.0E+2						
Hydrogen cyanide			9.6E-2	9.6E-2	2.6E+1						
Hydrogen sulfide			6.6E+0	6.6E+0	3.6E+1						
NO_x	1.3E+2			1.3E+2		1.0E+2					
SO_x	7.0E+2			7.0E+2		8.0E+1					
Nickel Carbonyl			7.4E-2	7.4E-2	1.0E-1						
Aliphatics, Alicyclics & Fatty Acids											
Acetic acid		1.6E 0		1.6E 0	6.0E+1		1.3E 0		1.3E 0	3.5E-1	
Butanoic acid		9.1E-2		9.1E-2	1.0E+1		1.2E-1		1.2E-1		
Hexanoic acid		6.9E-3		6.9E-3			9.4E-2		9.4E-3	5.1E-2	
3-Methylbutanoic acid		6.9E-3		6.9E-3			9.4E-3		9.4E-3		
2-Methylbutanoic acid		1.4E-2		1.4E-2			1.9E-2		1.9E-2		
Pentanoic acid		8.4E-2		8.4E-2	4.1E+1		1.1E-1		1.1E-1	2.0E-1	
Propanoic acid		1.7E-1		1.7E-2			2.5E-1		2.5E-1		
Total fatty acids			1.3E+1	1.3E+1							
Benzenes & Substituted Benzenes											
Ethylbenzene		2.1E-1		2.1E-1	1.0E+3		2.9E-1		2.9E-1	6.0E 0	
Toluene		6.9E-1		6.9E-1	8.9E+2		9.4E-1		9.4E-1	5.2E 0	

Table 4.6 (Cont'd)

Compound/Element	Estimated Ambient Atmospheric Levels (μg/m³)				Acceptable Ambient Atmospheric Levels (μg/m³)		Estimated Ambient Aquatic Levels (mg/L)			Acceptable Ambient Aquatic Levels (mg/L)	
	Utility Stack Gas	Cooling Tower Evaporation	Lockhopper Vent Gas	Total	EPC[a]	NAAQS[b]	Reverse Osmosis	Ash Leachate	Total	EPC[a]	DWS[c]
Benzenes & Substituted Benzenes (Cont'd)											
O-Xylene		2.4E-1		2.4E-1	1.0E+3		3.3E-1		3.3E-1	6.0E 0	
Biphenyl		7.6E-4		7.6E-5	2.4E 0		6.2E-2		6.2E-2	1.4E-2	
Indan		9.9E-3		9.9E-3	4.0E+2		7.9E-1		7.9E-1	2.0E 0	
Phenols											
Catechol		7.2E-1		7.2E-1	4.8E+1		1.7E 0		1.7E 0	2.8E-1	1.0E-3
3,6-Dimethylcatechol		5.9E-1		5.9E-1			1.4E-1		1.4E-1		1.0E-3
3-Methylcatechol		0.0E 0					0.0E 0				1.0E-3
4-Methylcatechol		5.1E-1		5.1E-1			1.2E 0		1.2E 0		1.0E-3
2-Methylphenol		5.9E-3		5.9E-3	5.2E+1		1.3E-2		1.3E-2	1.4E-1	1.0E-3
3-Methylphenol		3.6E-3		3.6E-3	2.4E+1		1.9E-2		1.9E-2	1.4E-1	1.0E-3
4-Methylphenol		4.9E-3		4.9E-3	2.4E+1		1.2E-2		1.2E-2	1.4E-1	1.0E-3
4-Methylresorcinol		4.7E-2		4.7E-2			1.1E-1		1.1E-1		1.0E-3
5-Methylresorcinol		8.4E-2		8.4E-2			2.1E-1		2.1E-1		1.0E-3
Resorcinol		2.4E-1		2.4E-1	1.1E+2		5.8E-1		5.8E-1	6.2E-1	1.0E-3
2,4-Xylenol		5.0E-3		5.0E-1	2.4E+1		3.6E-2		3.6E-2	1.2E-1	1.0E-3
3,5-Xylenol		5.0E-3		5.0E-3	2.4E+1		5.0E-2		5.0E-2	1.2E-1	1.0E-3
Total phenols											
Polynuclear Aromatic Hydrocarbons											
Acenaphthalene		1.5E-4		1.5E-4			1.2E-2		1.2E-2		
Acridine											
Anthracene		3.7E-6		3.7E-6			3.0E-3		3.0E-3		
Benz(a)anthracene		3.7E-6		3.7E-6	8.1E-1		3.0E-4		3.0E-4	4E-3	
Benzo(g,h,i)perylene		1.4E-2		1.4E-2			8.9E-6		8.9E-6		
Benzo(a)pyrene		1.5E-6		1.5E-6	4.0E 0		1.2E-4		1.2E-4	2E-2	
Benzo(e)pyrene		1.5E-6		1.5E-6			1.2E-4		1.2E-4		
Chrysene		7.5E-7		7.5E-7			5.9E-5		5.9E-5		
Fluoranthene		7.4E-5		7.4E-5	1.6E+2		5.9E-3		5.9E-3	8E-1	
Fluorene		7.4E-5		7.4E-5			5.9E-3		5.9E-3		
Indene											
Napthalene		3.6E-3		3.6E-3	1.2E+2		2.8E-1		2.8E-1	6.9E-1	
Perylene		1.5E-7		1.5E-7			1.2E-5		1.2E-5		
Phenanthrene		3.7E-5		3.7E-5	5.7E+1		3.0E-3		3.0E-3	2.8E-1	
Pyrene		7.4E-5		7.4E-5			5.9E-3		5.9E-3		
Total PAHs			2.5E-2	2.5E-2							

Table 4.6 (Cont'd)

Compound/Element	Estimated Ambient Atmospheric Levels ($\mu g/m^3$)				Acceptable Ambient Atmospheric Levels ($\mu g/m^3$)		Estimated Ambient Aquatic Levels (mg/L)			Acceptable Ambient Aquatic Levels (mg/L)	
	Utility Stack Gas	Cooling Tower Evaporation	Lockhopper Vent Gas	Total	EPC[a]	NAAQS[b]	Reverse Osmosis	Ash Leachate	Total	EPC[a]	DWS[c]
Sulfur Heterocyclics											
Methylthiophene					4.1E+1						
Thiophene					8.0E 0						
Total thiophenes			2.0E-1	2.0E-1							
Nitrogen Heterocyclics											
2,4-Dimethylpyridine		1.1E-4		1.1E-4	3.2E+1		2.6E-4		2.6E-4	1.6E-1	
2,5-Dimethylpyridine		1.1E-4		1.1E-4	3.2E+1		2.6E-4		2.6E-4	1.6E-1	
2-Methylpyridine		8.4E-3		8.4E-3	6.4E+1		1.8E-2		1.8E-2	3.2E-1	
3-Methylpyridine		2.0E-3		2.0E-3	6.4E+1		6.7E-3		6.7E-3	3.2E-1	
4-Methylpyridine		6.0E-4		6.0E-4	6.4E+1		1.6E-3		1.6E-3	3.6E-1	
Pyridine		1.4E-3		1.4E-3	3.6E+1		3.0E-3		3.0E-3	2.1E-1	
Quinoline		1.1E-3		1.1E-3	2.8E+1		2.5E-3		2.5E-3	1.4E+1	
Oxygen Heterocyclics											
Benzofuran											
Dibenzofuran		1.0E-3		1.0E-3			2.2E-3		2.2E-3		
Mercaptans											
Methanethiol		8.4E-2		8.4E-2	2.4E 0		5.9E-1		5.9E-1	1.4E-2	
Total mercaptans			2.5E-1	2.5E-1							
Aromatic Amines											
Aniline		1.0E-3		1.0E-3	4.5E+1		1.3E-3		1.3E-3	2.6E-3	
Total amines			4.9E-2	4.9E-2							
Nitrosamines			2.5E-2	2.5E-2							

[a]U.S. EPA MultiMedia Environmental Goal (Estimated Permissible Concentration for Health).

[b]U.S. EPA National Ambient Air Quality Standard

[c]U.S. EPA Drinking Water Standards

Columns may not total due to use of different data sources.

the lowest projected hazard level of the pollutants released by Lurgi/Fischer-Tropsch liquefaction.

Table 4.7 lists Lurgi atmospheric pollutants that qualify for Level 1 and Level 2 designations and the waste streams that are primary sources.

Of the quantifiable emission streams, only lockhopper vent gas is unique to the Lurgi/Fischer-Tropsch process. Utility stack gas and cooling tower evaporation streams are found in many other industrial applications, including electricity generation (although in the Lurgi process, treated gasifier waste water is cycled to the cooling tower). Three of the six substances classified in Level 1 or Level 2, hydrogen sulfide, ammonia, and nickel carbonyl, are released primarily in the lockhopper vent gas. The primary sources of arsenic, mercury, nitrogen oxices and sulfur dioxide are utility stack gas and cooling tower evaporation streams. Potential emissions from the Lurgi/Fischer-Tropsch operation that were not qualified include: fugitive emissions, non-cooling tower evaporative emissions, baghouse vent gas, deaeration, and Fischer-Tropsch vent gas.

Ammonia from the cooling tower and lockhopper vent may violate the atmospheric EPC by a factor of three. Ammonia irritates the eyes and respiratory tract, but low-level exposure has no chronic effects. Ammonia rapidly decomposes in the presence of oxygen, and consequently has a relatively short half-life in the atmosphere. Many commercially proven technologies

Table 4.7 Substances with Projected Level 1 or Level 2 Atmospheric Concentrations

Substance	Primary Emission Source	Standard
Level 1		
None		
Level 2		
Trace Elements		
Arsenic	cooling tower evaporation, utility stack gas	EPC
Mercury	utility stack gas	EPC
Gases		
Ammonia	cooling tower evaporation, lockhopper vent gas	EPC
Hydrogen sulfide	lockhopper vent gas	EPC
Nitrogen oxides	utility stack gas	NAAQS
Sulfur dioxide	utility stack gas	NAAQS
Nickel carbonyl	lockhopper vent gas	EPC

remove ammonia from aqueous streams, and it is economically viable to do so for small concentrations. Due to the nature of ammonia's toxicity, biodegradability, and controllability, it is not expected to be a major health concern from Lurgi/Fischer-Tropsch operations.

Two trace elements, arsenic and mercury, and four gases, hydrogen sulfide, sulfur dioxide, nitrogen oxides, and nickel carbonyl, are projected to have ambient atmospheric concentrations within a factor of ten of acceptable levels.

Arsenic and mercury are extremely toxic under acute and chronic exposure conditions. Arsenic is a suspected carcinogen that can damage the kidney and liver and inhibit enzyme activity. Mercury exposure causes neural, renal, and cardiovascular disorders. Both trace elements accumulate through food chains and have relatively long biological half-lives once they are absorbed into the body (see Sec. 4.1). They may be released in currently unquantifiable Lurgi/Fischer-Tropsch emission streams. As a result, both are considered a significant hazard to public health.

Trace element emissions from coal conversion are directly related to the trace element content of the coal being converted. Although the type of release (atmospheric, aquatic, or solid waste) may be affected by trace element characteristics such as volatility, the amount released depends on the concentrations in the makeup coal. Thus, while arsenic and mercury are important atmospheric emissions from Lurgi/Fischer-Tropsch liquefaction of Wyoming subbituminous coal, liquefaction of another coal could change the magnitude and type of trace elements emitted.

Hydrogen sulfide is acutely toxic in high concentrations and irritates the respiratory tract in low concentrations. Its primary quantifiable source in Lurgi/Fischer-Tropsch operations is lockhopper vent gas. Although not accounted for in this assessment, operating procedures call for flaring lockhopper gas that is vented. The effect of flaring has not been quantified but it will probably lower hydrogen-sulfide emissions. The biological half-life of hydrogen sulfide under ambient atmospheric conditions is short -- approximately 20 min. In addition, the odor threshold is far below the toxic threshold, which allows time to identify potential hazards and reduce the threat of exposure. As a result, hydrogen sulfide from Lurgi/Fischer-Tropsch liquefaction is not expected to present significant risk to public health.

Sulfur dioxide is a respiratory irritant that can instigate lung tissue dysfunction as well as exacerbate existing disorders. Because sulfur oxides from coal combustion are a recognized health threat, federal and state governments have set emission standards to limit ambient concentrations. Wyoming's sulfur-emission standards, for example, are more stringent than national standards. In this assessment, it is assumed that the Lurgi/Fischer-Tropsch facility will be located in and meet emission standards of Wyoming. Dispersion modeling indicates that ambient sulfur-oxide concentrations will exceed NAAQS of 80 $\mu g/m^3$. However, SAM I projects maximum instantaneous ground-level concentrations, whereas the NAAQS are set for annual average concentrations. The projected maximum sulfur-dioxide concentration of 700 $\mu g/m^3$ compares favorably with the 3-month NAAQS of 1200 $\mu g/m^3$ and indicates that the potential hazard is significantly lower.

Annual average NAAQS for nitrogen oxides are 100 $\mu g/m^3$. The SAM I projects a maximum ground-level concentration of 130 $\mu g/m^3$. No national standard with a shorter averaging time has been set, although some states, such as North Dakota, have set standards at 200 $\mu g/m^3$ for a 1-h average. Nitrogen-oxide levels may be made up of several compounds, of which nitrogen dioxide is the most important in terms of toxicity. Nitrogen dioxide is lipid soluble and easily reaches alveolar regions of the lung, where it forms nitrous and nitric acid. The acids irritate and corrode the mucous membranes in the lung. Acute exposure results in pulmonary edema; chronic exposure is associated with emphysema. Nitrogen oxides are also precursors of ozone, which is highly reactive and damages respiratory tissue. Because of the relationship between the projected maximum instantaneous concentrations and the annual average and 1-h maximum standards, nitrogen dioxide is not expected to be a major public health hazard.

Nickel carbonyl is a proved carcinogen under chronic exposure conditions in occupational environments. Acute exposure may induce chemical pneumonitis. The primary quantifiable source from Lurgi/Fischer-Tropsch operation is lockhopper vent gas, which is periodically flared to the atmosphere. The effect of flaring on nickel carbonyl concentrations is uncertain. In addition, the intermittent nature of the vent gas release increases the uncertainty of exposure impact. However, the proved carcinogenic effect of nickel carbonyl and the magnitude of potential releases underscore the possible public health risk from Lurgi/Fischer-Tropsch liquefaction.

Tables 4.8 and 4.9 list aquatic pollutants from Lurgi processes that qualify for Level 1 and Level 2 designations. The tables also indicate which standard applies and which effluent stream is the primary source of the pollutant.

Two primary effluent streams, reverse osmosis and combined ash leachate, have characteristics unique to the Lurgi/Fischer-Tropsch process. The effect of these streams on aquatic environment depends largely on the specific site. The SAM I dispersion factors used to estimate pollutant concentrations provide maximum projected values and do not account for such factors as sedimentation, which may significantly reduce aquatic concentrations. The third major Lurgi/Fischer-Tropsch effluent stream, biosludge leachate, was not quantified in this assessment.

Fourteen pollutants -- two trace elements, 11 phenols and one mercaptan -- qualify for Level I designation by exceeding acceptable ambient concentrations by more than a factor of ten. Seven more trace elements, three fatty acids, one substituted benzene, three polynuclear aromatic hydrocarbons, and one aromatic amine -- qualify for Level 2 designation.

Trace elements of concern include aluminum, arsenic, beryllium, cadmium, mercury, nickel, lead, selenium, and vanadium. The toxicity of trace elements varies significantly with chemical form of the element. In general, the free ions are most toxic. The degree to which trace elements attenuate in natural systems is also a function of chemical form. Again, the ionic form is most reactive and most likely to bind to particles or other substances in the solution or transfer medium, where it becomes less available for subsequent toxic impact. Biological systems may alter some trace elements to chemical forms that are more toxic than the original form (e.g., elemental

Table 4.8 Substances with Projected Level 1 Aquatic Concentrations

Substance	Primary Effluent	Standard[a]
Trace Elements		
Beryllium	Reverse osmosis	EPC
Nickel	Reverse osmosis, combined ash leachate	EPC
Phenols		
Catechol	Reverse osmosis	DWS
3,6-Dimethylcatechol	Reverse osmosis	DWS
4-Methylcatechol	Reverse osmosis	DWS
2-Methylphenol	Reverse osmosis	DWS
3-Methylphenol	Reverse osmosis	DWS
4-Methylphenol	Reverse osmosis	DWS
4-Methylresorcinol	Reverse osmosis	DWS
5-Methylresorcinol	Reverse osmosis	DWS
Resorcinol	Reverse osmosis	DWS
2,4-Xylenol	Reverse osmosis	DWS
3,5-Xylenol	Reverse osmosis	DWS
Mercaptans		
Methanethiol	Reverse osmosis	EPC

[a]EPC = EPA's Estimated Permissible Concentration for Health; DWS = EPA's Drinking Water Standard.

Table 4.9 Substances with Projected Level 2 Aquatic Concentrations

Substance	Primary Effluent	Standard[a]
Trace Elements		
Aluminum	Combined ash leachate	EPC
Arsenic	Reverse osmosis	DWS
Boron	Reverse osmosis	EPC
Cadmium	Reverse osmosis	DWS
Lead	Reverse osmosis, combined ash leachate	DWS
Mercury	Reverse osmosis	DWS
Selenium	Combined ash leachate	DWS
Vanadium	Reverse osmosis	EPC
Aliphatics, Alicyclics, and Fatty Acids		
Acetic acid	Reverse osmosis	EPC
Hexanoic acid	Reverse osmosis	EPC
Pentanoic acid	Reverse osmosis	EPC
Substituted Benzenes		
Toluene	Reverse osmosis	EPC
Polynuclear Aromatic Hydrocarbons		
Biphenyl	Reverse osmosis	EPC
Indan	Reverse osmosis	EPC
Napthalene	Reverse osmosis	EPC
Aromatic Amines		
Aniline	Reverse osmosis	EPC

[a]EPC = EPA's Estimated Permissible Concentration for Health; DWS = EPA's Drinking Water Standard.

mercury to methylmercury). The long biological half-life of most trace elements increases the potential for accumulation of toxic quantities in humans.

A long list of phenols have been projected to exceed EPA drinking water standards. Phenols, although not thought to be carcinogenic, are suspected to act synergistically with some carcinogens (e.g., coal tars) to increase the risk of cancer. Chronic exposure to phenols has been linked with kidney and liver damage. The phenol drinking water standard of 0.01 ppm is based on taste and odor considerations; the toxic threshold may not be reached until concentrations exceed the standard by several orders of magnitude. Additionally, a variety of commercially proved techniques can remove phenols from aqueous waste streams. However, all phenols projected to violate or approximate drinking water standards are also found in unquantified Lurgi/Fischer-Tropsch waste streams, a fact that underscores the potential human health risk.

Methanethiol is the mercaptan that is projected to exceed the aquatic EPC. Its primary toxic effects are respiratory paralysis and pulmonary edema. The aquatic EPC was established to minimize the additive or synergistic effects of combined exposure via respiratory and gastrointestinal routes of entry. Methanethiol is found in eight Lurgi/Fischer-Tropsch waste streams that are unquantified.

The aliphatics, alicyclics, and fatty acids projected to violate or nearly violate acceptable aquatic concentrations include acetic acid, hexanoic acid, and pentanoic acid. These weak organic acids are not active from a toxicological viewpoint. All are widely distributed in the environment and may be intermediate metabolites. They are projected to exceed EPCs based on impacts from chronic skin exposure, which is not a probable mode of public exposure. All three biodegrade in aquatic systems and can be removed from drinking water through conventional treatment methods. As a result, these acids are not important sources of public health risk.

One substituted benzene -- toluene -- is projected to nearly violate the EPC. It affects the central nervous system, liver, kidneys, and skin. Toluene's metabolites, benzaldehyde and benzoic acid, are nontoxic. Toluene is highly susceptible to photochemical degradation and has a short biological half-life of approximately 2 h. Consequently, toluene from Lurgi/Fischer-Tropsch operation is not expected to present a significant public health risk.

As a class, polynuclear aromatic hydrocarbons contain substances that cause cancer in humans [e.g., benzo(a)pyrene]. These compounds result from incomplete combustion of organic materials such as coal and are found in many Lurgi/Fischer-Tropsch waste streams, some of which are currently unquantifiable. Although evidence is inconclusive, the carcinogenic agent may be the PAH metabolite and not PAH itself. The primary risk from exposure to biphenyl, indan, and naphthalene is this potential for inducing cancer, since no evidence exists of additional chronic or acute exposure effects.

Aniline is the aromatic amine projected to have an ambient aquatic concentration within one order of magnitude of the EPC. Aniline is toxic via inhalation, ingestion, or dermal exposure. As a liquid, aniline behaves much

like ammonia. Acute ingestion has resulted in human death; chronic exposure causes neural dysfunction. Aniline is readily degraded in biological systems and, as such, is not considered a major public hazard from Lurgi/Fischer-Tropsch operation.

4.2.2 Body Burdens

Quantifying the effects of Lurgi/Fischer-Tropsch effluents on humans is the optimum measure of risk. Unfortunately, dose-response data for the pollutants are not available, which makes determination of the absolute number and type of effects impossible. Determining the relative impact of exposure is possible, however, for substances whose exposure, route of entry, absorption, and biological half-life are known. With these measures, the body burden resulting from a specific source of exposure can be calculated. Body burdens represent the amount of substance that accumulates within the body. Body burdens for different sources of exposure and for different routes of entry to the body can be compared to measure the relative risk of exposure.

Data on exposure, route of entry, absorption, and biological half-life are available for many trace elements released by the Lurgi/Fischer-Tropsch process. In this assessment, body burdens for the trace elements arsenic, cadmium, and lead were calculated using the Argonne body-burden model.[45] These three elements were chosen because they are all highly toxic and encompass the known range for absorption and biological half-life of other trace elements. Body burdens were calculated for four sources of exposure (background air and water, diet, a 1000-MWe coal-fired power plant, and the Lurgi/Fischer-Tropsch facility) and two routes of entry (respiratory and gastrointestinal). Body burdens represent amount of element per gram of tissue accumulating in an exposed 70-kg male who breathes 20 m^3 of air per day and drinks 2 L of water per day.

Trace-element exposure from Lurgi/Fischer-Tropsch wastes were based on environmental loading factors from Part 2 of the overall study (characterization of hazardous wastes) to which SAM I environmental dilution factors were applied. Contributions from the 1000-MWe coal-fired power plant were based on projected ambient concentrations for such a facility burning No. 6 Illinois coal and located in Fulton County, Illinois. Although Illinois and Wyoming coals vary in many respects, the arsenic, cadmium, and lead content of feed coals for these two technologies were similar. Contributions from background air and water were national averages determined by EPA monitoring programs. Contributions from diet represent average U.S. dietary characteristics.[45]

Waste streams from Lurgi/Fischer-Tropsch that have quantifiable amounts of trace elements include: utility stack gas, cooling tower evaporation, reverse osmosis, combined ash, and biosludge. Many other streams, while having the potential for trace-element release, were unquantifiable. Table 4.10 lists the results of the body-burden calculations and Figs. 4.1, 4.2, and 4.3 illustrate the results graphically.

As Fig. 4.1 shows, the body burden of arsenic from exposure to Lurgi/Fischer-Tropsch environmental wastes is approximately double that from exposure to coal-fired power-plant wastes or to background air and water. However, the Lurgi/Fischer-Tropsch body burden of arsenic is 45% of that

Table 4.10 Body Burdens

Elements	Source[a]	Via Respiratory Tract ($\mu g/g \times 10^{-2}$)	Via Gastrointestinal Tract ($\mu g/g \times 10^{-2}$)	Via Diet ($\mu g/g \times 10^{-2}$)
Arsenic	L/F-T[b]	0.02	3.1	6.9
	CFPP[b]	0.13	1.5	6.9
	background	0.004	1.7	6.9
Cadmium	L/F-T	0.5	62	4690
	CFPP	0.5	148	4690
	background	0.9	117	4690
Lead	L/F-T	0.3	31.3	325
	CFPP	9.5	60.1	325
	background	3.7	34	325

[a]L/F-T = Lurgi/Fischer-Tropsch plant; CFPP = coal-fired power plant.

[b]Lurgi/Fischer-Tropsch figures based on air and water concentrations from Table 4.6.

resulting from diet (total body burden equals the sum of burdens from all sources of exposure). Thus, the relative risk of exposure to arsenic from Lurgi/Fischer-Tropsch is twice that from coal-fired power plants but less than half of that from diet. The primary route of entry for all sources of arsenic is the gastrointestinal tract, which accounts for between 92% and 99% of the total absorbed from Lurgi/Fischer-Tropsch, coal-fired power generation, and background exposure. The importance of the gastrointestinal route of entry is underscored by the fact that only 3% of the arsenic passing through the tract is absorbed, compared to 50% absorption in the respiratory tract.

The Lurgi/Fischer-Tropsch contribution of cadmium to body burden is less than that from any other source of exposure. The projected burden from exposure to Lurgi/Fischer-Tropsch wastes is approximately 60% of that from background air and water, 40% of that from coal-fired power generation and 1% of that from diet. As with arsenic, the primary route of entry is the gastrointestinal tract. Although the health effects of cadmium will depend on the as-yet-undefined threshold of impact, the quantifiable contribution by Lurgi/Fischer-Tropsch wastes and the corresponding risk will be less than those from background air and water, coal-fired power generation, or diet.

Body burdens of lead from Lurgi/Fischer-Tropsch environmental wastes are projected to be less than from any other source of exposure in this assessment. The largest contributor to lead body burden is diet. Risk from diet is approximately ten times that from Lurgi/Fischer-Tropsch. Coal-fired power generation and background air and water also contribute more to potential risk -- 50% and 10% more, respectively -- than Lurgi/Fischer-Tropsch, based on the body burdens attributable to each. As with arsenic and cadmium, the gastrointestinal tract is the primary route of entry for lead from all sources of exposure.

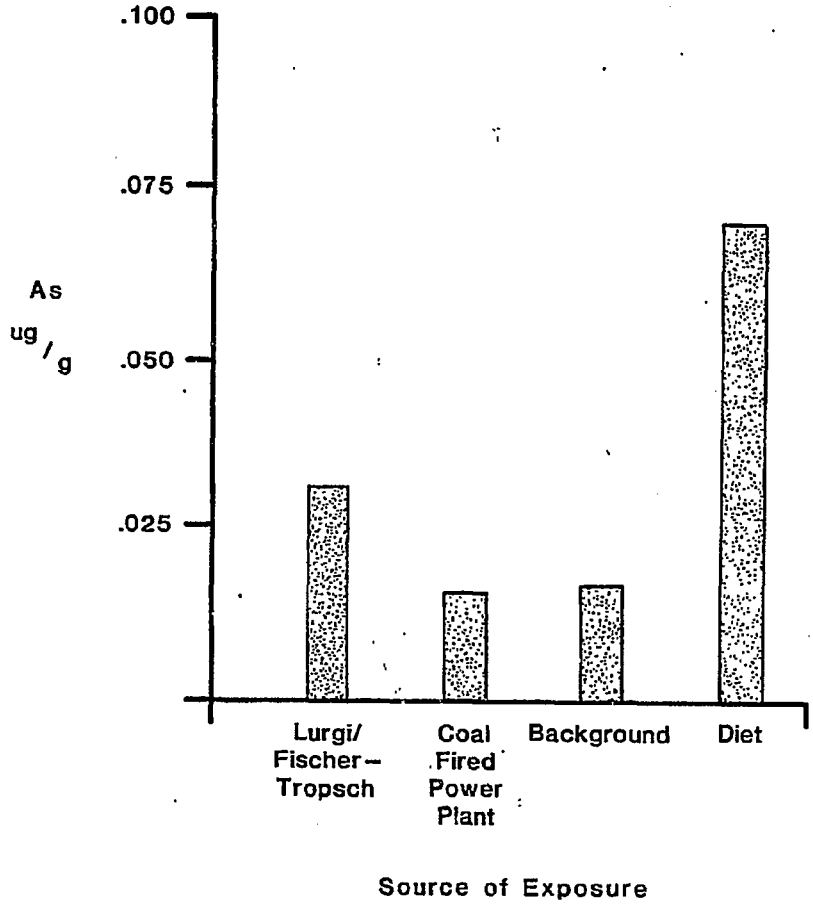


Fig. 4.1 Body Burdens of Arsenic from Four Sources of Exposure

The magnitude and severity of any adverse effect depends on the total body burden from all sources of exposure and a currently unquantifiable threshold level for effect. However, body burdens can be compared to estimate the relative risk from each source of exposure. The relative risk from Lurgi/Fischer-Tropsch environmental wastes is slightly less than that from coal-fired power generation for two trace elements and slightly greater for one. In all cases, the risk from diet is substantially greater than from any other source.

Two factors may affect these results. Several potential sources of trace element release from the Lurgi/Fischer-Tropsch process were not quantified in this assessment. Additionally, this analysis does not consider trace elements released to the environment when Lurgi/Fischer-Tropsch product liquid is burned. Both factors potentially would alter the ambient air and water trace-element concentrations and subsequently affect body-burden calculations.

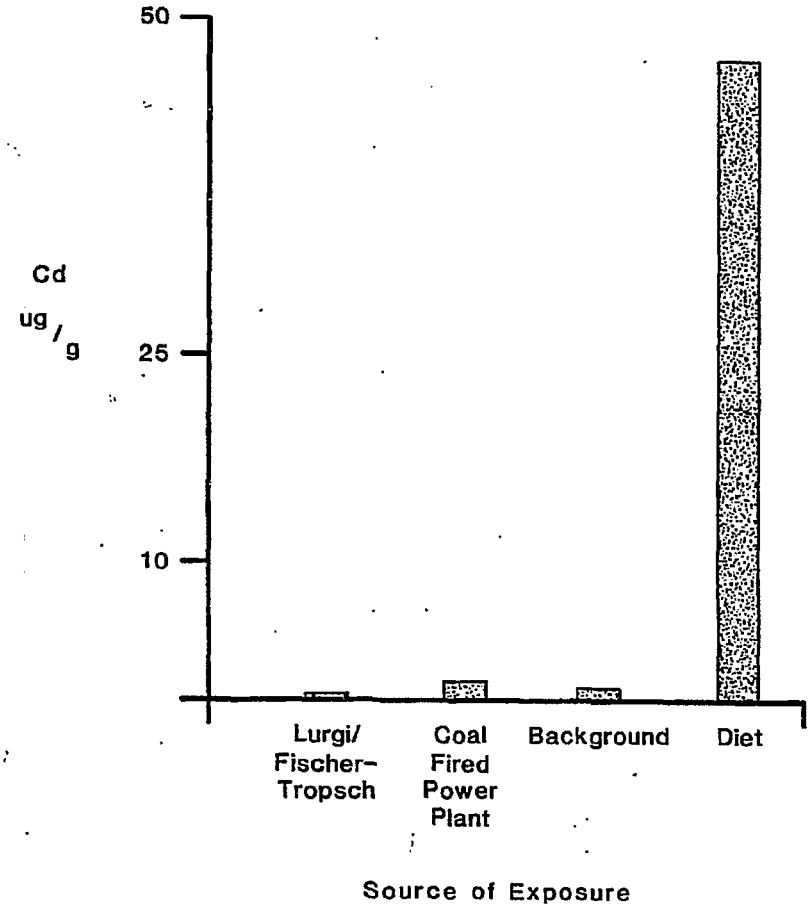


Fig. 4.2 Body Burdens of Cadmium from Four Sources of Exposure

4.2.3 Substances of Concern

To conclude this analysis, two lists of toxic substances from Lurgi/Fischer-Tropsch liquefaction are presented. The first list, Table 4.11, includes the substances that have been quantified in Lurgi effluents. Sufficient data for these substances are available to compare the health risks from coal liquefaction wastes to those from other sources of the same pollutants. Public health impacts from Lurgi/Fischer-Tropsch production of these substances are severe enough to warrant investigating environmental controls beyond those of the reference system. The second list, Table 4.12, is a qualitative assessment and includes substances that may be released in Lurgi effluents but have not been quantified and are highly toxic. These pollutants may be on the Cancer Assessment Group list of carcinogens or included in Category 3 (see Sec. 4.2). Thus the inherent potential for health impact of these substances, if released, is great enough to warrant further characterization.

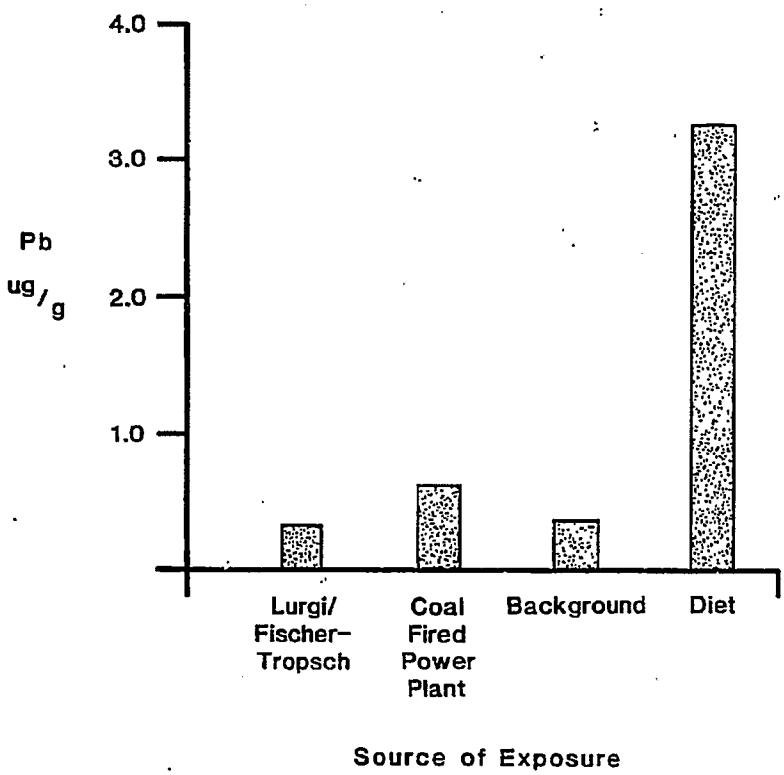


Fig. 4.3 Body Burdens of Lead from Four Sources of Exposure

Table 4.11 Substances of Concern -- Quantitative Analysis

Trace Elements	Phenols
Arsenic	Catechol
Boron	3,6-Dimethylcatechol
Beryllium	3-Methylcatechol
Cadmium	4-Methylcatechol
Mercury	2-Methylphenol
Nickel	3-Methylphenol
Lead	4-Methylphenol
Selenium	4-Methylresorcinol
Vanadium	5-Methylresorcinol
Polynuclear Aromatics	Phenol
	Resorcinol
Biphenyl	2,4-Xylenol
Napthalene	3,5-Xylenol
Indan	Sulfur Heterocyclics
Gases	Methanethiol
Nickel carbonyl	

Table 4.12 Substances of Concern -- Qualitative Analysis

Benzenes and Substituted Benzenes	Nitrosamines
Ethylbenzene	N-nitrosamine
Xylene	
Benzene[a]	Nitrogen Heterocyclics
Polynuclear Aromatic Hydrocarbons	Acridine
Anthracene[a]	Gases
Fluoranthene	Nickel carbonyl[a]
Fluorene	
Napthalene	Trace Elements
Phenanthrene	Arsenic
Pyrene	Beryllium[a]
Indene	Cadmium[a]
Biphenyl	Lead
Chrysene[a]	Mercury
Benzo(a)pyrene[a]	Chromium[a]
Phenols	Nickel[a]
Xylenol[a]	Sulfur[a]
	Vanadium
Oxygen Heterocyclics	Uranium
Benzofuran	Cobalt
Dibenzofuran	Barium

[a]Carcinogens.

REFERENCES

1. Comar, C.L., *Health Aspects*, Conf. Proceedings: Workshop on Health Effects of Fossil Fuel Combustion Products, Electric Power Research Institute, Menlo Park, Cal., pp. 52-55 (1975).

2. *Industrial Hygiene and Toxicology*, Vol. 2, Revised 2nd Ed., F.A. Patty, ed., Interscience Publishers, John Wiley and Sons Inc., New York (1967).

3. Driesbach, R.H., *Handbook of Poisoning: Diagnosis and Treatment*, 8th Ed., Lange Medical Publications, Los Altos, Cal. (1974).

4. Tseng, W.P., et al., *Prevalence of Skin Cancer in an Endemic Area of Chronic Arsenicism in Taiwan*, J. of National Cancer Institute, *40*:453 (1968).

5. Sittig, M., *Priority Toxic Pollutants: Health Impacts and Allowable Exposure Limits, Environmental Health Review No. 1*, Noyes Data Corp., N.J. (1980).

6. *Criteria for a Recommended Standard: Occupational Exposure to Inorganic Arsenic*, National Institute of Occupational Safety and Health, NIOSH Doc. 75-149 (Revised), Washington, D.C. (1975).

7. *Registry of Toxic Effects of Chemical Substances*, 1976 Ed., National Institute of Occupational Safety and Health, Washington, D.C.

8. *Quality Criteria for Water*, U.S. Environmental Protection Agency Report EPA-440/9-76-023 (1976).

9. *Proposed Standard for Occupational Exposure to Beryllium*, Occupational Safety and Health Administration, Washington, D.C. (1977).

10. *National Emission Standards for Hazardous Air Pollutants*, U.S. Environmental Protection Agency, Title 40 CFR, Part 61.

11. *Water Quality Criteria, 1972*, U.S. Environmental Protection Agency Report EPA-R3-73-033 (1973).

12. *Multimedia Environmental Goals for Environmental Assessment*, Vol. IV, MEG Charts and Background Information, U.S. Environmental Protection Agency Report EPA-600/7-79-176b (1979).

13. Friberg, L., *Proteinuria in Chronic Cadmium Dust*, American Medical Assn., Archives Industrial Health *16*:30-31 (1957).

14. Friberg, L., et al., *Cadmium in the Environment*, 2nd Ed., CRC Press, Cleveland, Ohio (1974).

15. *Documentation of Threshold Limit Values for Substances in the Workroom Air*, Supplements, 3rd Ed., American Conf. of Governmental Industrial Hygienists, Cincinnati, Ohio (1977).

16. Stokinger, H.E., *The Metals (Excluding Lead)*, Industrial Hygiene and Toxicology, Vol. 2, F.A. Patty, ed., Interscience Publishers, John Wiley and Sons Inc., New York (1963).

17. *Scientific Technical Assessment Report on Cadmium*, Star Series, U.S. Environmental Protection Agency Report EPA 600/6-75-003 (1975).

18. *Environmental Health and Control Aspects of Coal Conversion*, Oak Ridge National Laboratory Report ORNL/EIS-357, Chapter 10 (1978).

19. Spehar, R.L., *Cadmium and Zinc Toxicity to Flagfish, Jordanella Floridne*, Journal of Canadian Fisheries Research Board, *33*:1939 (1976).

20. World Health Organization, *Evaluation of Certain Food Additives and the Contaminants Mercury, Lead, and Cadmium*, 16th Report of the Joint FAD-WHO Expert Committee on Food Additives, WHO Technical Report 505, Geneva, Switzerland (1972).

21. *Dangerous Properties of Industrial Materials*, 5th Ed., N.I. Sax, ed., Van Nostrand Reinhold, New York (1979).

22. Roach, S.A., *A More Rational Basis for Air Sampling Programs*, J. of the American Industrial Hygienists Association, *27*:1012 (1966).

23. *Recommended Health and Safety Guidelines for Coal Gasification Pilot Plants*, interagency report, U.S. Environmental Protection Agency Report EPA-600/7-78-007 (Jan. 1978).

24. World Health Organization, *International Standards for Drinking Water*, 3rd Ed., Geneva, Switzerland (1971).

25. Nogawa, K.E., I. Kobyashi, and M. Sakamoto, *Studies of the Effects of Air Pollution Consisting of Dusts Mainly Composed of Manganese in the Respiratory Systems of Middle-School Pupils*, J. Public Health *20*:314-325, Japan (1973).

26. Sakurai, H., and T. Tsuchiya, *A Tentative Recommendation for the Maximum Daily Intake of Selenium*, Environmental Physiology and Biochemistry, *5*:107 (1975).

27. *Trace Elements in Human Health and Disease*, Vol. II, Essential and Toxic Elements, A.S. Prasad, ed., Academy Press, New York (1976).

28. *Threshold Limit Values for Chemical Substances in the Workroom Air*, American Conf. of Governmental Industrial Hygienists, Cincinnati, Ohio (1976).

29. Greaves, M.W., and A.W. Sillen, *Effects of Long-continued Ingestion of Zinc Sulfate in Patients with Venous Leg Ulceration*, Lancet (Oct. 31, 1970).

30. *Detoxication Mechanisms*, R.I. Williams, ed., John Wiley and Sons Inc., New York (1959).

31. *Chemical Carcinogens*, C.E. Searle, ed., American Chemical Society Monograph No. 173, Washington, D.C. (1976).

32. Wagoner, D., *Compilation of Ambient Trace Substances*, draft report for U.S. Environmental Protection Agency prepared by Research Triangle Institute under Contract No. 698-02-1325 (1979).

33. *Benz(a)pyrene: Hazard Profile*, Center for Hazard Assessment, Syracuse Research Corporation, Syracuse, N.Y.

34. Tye, R., and K.L. Stemmer, *Experimental Carcinogenesis of the Lung, Influence of Phenols in the Production of Carcinoma*, J. National Cancer Institute *39*:175-186 (1967).

35. Hamilton, A., and H.L. Hardy, *Industrial Toxicology*, 3rd Ed., Publishing Sciences Group, Cambridge, Mass. (1974).

36. 40 Federal Register 1910, 1034.

37. Texas Eastern Corporation, unpublished information (Aug. 1980).

38. Innes, J.R.M., et al., *Bioassay of Pesticides and Industrial Chemicals for Tumorigenicity in Mice: A Preliminary Note*, J. National Cancer Institute *42*:1101 (1969).

39. Shy, C.M., et al., *The Chattanooga School Children Study: Effects of Community Exposure to Nitrogen Dioxide, I.* Methods, description of pollutant exposure, and results of ventilatory function testing, J. Air Pollution Control Assn., *20*(9):539-45 (1970).

40. Shy, C.M., et al., *The Chattanooga School Children Study: Effects of Community Exposure to Nitrogen Dioxide, II.* Incidence of acute respiratory illness, J. Air Pollution Control Assn. *20*(9):582-88 (1970).

41. Pearlman, M.E., et al., *Nitrogen Dioxide and Lower Respiratory Illness*, Pediatrics *47*:291-95 (1971).

42. Stern, A.C., *Air Pollution*, Volume 2, 3rd Ed., Academy Press, N.Y. (1977).

43. *Health Effects of Environmental Pollutants*, G.L. Waldbott, ed., C.V. Mosby Co., St. Louis (1973).

44. Carcinogen Assessment Group, *List of Potential Human Carcinogens*, U.S. Environmental Protection Agency (July 1980).

45. Gasper, J.R., P.A. Dauzvardis, and T.G. Surles, *A Relative Risk Assessment of Trace Elements from Coal Emissions, Drinking Water, and Diet*, Argonne National Laboratory Report ANL/AA-23 (Oct. 1979).