



Cornell University
ILR School

Cornell University ILR School
DigitalCommons@ILR

Manuals and User Guides

ILR Collection

12-1-1997

Health Hazard Manual: Wastewater Treatment Plant and Sewer Workers

Nellie J. Brown

Cornell University, njb7@cornell.edu

Follow this and additional works at: <http://digitalcommons.ilr.cornell.edu/manuals>



Part of the [Labor Relations Commons](#)

Thank you for downloading an article from DigitalCommons@ILR.

[Support this valuable resource today!](#)

This Article is brought to you for free and open access by the ILR Collection at DigitalCommons@ILR. It has been accepted for inclusion in Manuals and User Guides by an authorized administrator of DigitalCommons@ILR. For more information, please contact hlmdigital@cornell.edu.

Health Hazard Manual: Wastewater Treatment Plant and Sewer Workers

Abstract

[Excerpt] This manual examines how exposure occurs during the treatment processes; ways to reduce exposure by engineering controls, administrative controls, process control strategies, and protective equipment; and some suggested medical surveillance.

Keywords

ILR, Cornell University, chemical hazard information program, work environment, working conditions, employee, health, safe, contract, union, collective bargaining, work, member, labor, human resources, sewage, hazardous waste, chemicals, exposure, sewer, wastewater

Disciplines

Labor Relations

Comments

Suggested Citation

Brown, N. J. (1997). *Health hazards manual: Wastewater treatment plant and sewer workers*. Ithaca, NY: Cornell University, Chemical Hazard Information Program.
<http://digitalcommons.ilr.cornell.edu/manuals/2>

Required Publisher Statement

Copyright by Cornell University, Workplace Health and Safety Program (formerly Chemical Hazard Information Program).
Preparation of this manual was funded by New York State Department of Labor Grant #C005413.

Health Hazard Manual

WASTEWATER TREATMENT PLANT and SEWER WORKERS

Exposure to chemical hazards and biohazards

By Nellie J. Brown, M.S., C.I.H.

**Cornell University
Chemical Hazard Information Program
Dr. James Platner, Toxicologist/Director
New York State Department of Labor Grant #C005413
Revision 12/01/97**

ACKNOWLEDGEMENTS

Manuscript review has been provided by Dan Bentivogli and Dr. James Platner. Catherine M. Gawinski and Nona McQuay provided assistance in technical writing and format design in preparing this manual. Sheila Suggs and Jeanette O'Connor did the word processing to bring the manual to print.

Cornell University and the New York State Department of Labor assumes no responsibility for any actions or losses that might occur as a result of reading this manual. The opinions expressed are those of the author.

TABLE OF CONTENTS

INTRODUCTION	1
1. How are wastewater workers exposed to chemicals or diseases?	3
2. What kind of exposures occur for different kinds of treatment processes?	3
3. What about exposure to other materials in wastewater such as metals, radioactive compounds, or asbestos?	6
4. Do workers exposed to chemicals for short periods of time show any adverse health effects?	7
5. Do wastewater workers show adverse health effects from chemicals after years on the job?	9
6. Do wastewater workers experience any adverse effects upon their reproductive function or upon their offspring?	11
7. Are workers exposed to diseases by inhalation?	11
8. What kinds of diseases are wastewater workers exposed to?	13
9. Do treatment processes reduce or kill disease-causing organisms?	20
10. How can we find out whether wastewater workers are exposed to disease?	22
11. Do wastewater workers actually get sick from disease exposure at work?	25
12. What about the risk of contracting hepatitis B or AIDS?	27
13. Can a worker get a disease from an accidental exposure, such as sewage splashed into the mouth?	28
14. Is the disease risk really so low?	31
15. Do wastewater workers develop allergies, other than reactions to endotoxins or mycotoxins?	
16. How can exposure to chemicals and disease be reduced?	32

17. Should a wastewater worker be immunized to reduce the risk of infection?	36
18. Are there adverse health effects from chronic exposure to low levels of hydrogen sulfide gas?	37
19. What medical surveillance is recommended for the wastewater worker?	42

- *A list of complete source references is available upon request.*

Call

(716) 852-4191

or write to

Cornell University NYSSILR

Chemical Hazard Information Program

237 Main Street

Suite 1200

Buffalo, New York 14203

HEALTH HAZARD MANUAL FOR WATER AND WASTEWATER TREATMENT WORKERS

Introduction

Sewage is the used water of a community and can include domestic wastewater and industrial wastewater. Combined sewer systems will include storm water such as road runoff which carries oils, salts, metals, and asbestos. Many systems, especially older ones, will receive infiltration which can carry pesticides and herbicides from soil application.

For many years, work in the wastewater treatment field was considered the most hazardous, especially due to deaths involving confined space entry. This field is considered somewhat less hazardous today, but treatment plant workers still do experience health problems and deaths. These experiences occur in specific incidents involving chemicals in the sewer system and in regular work exposures throughout the plant and its processes.

Some chemically-related health complaints are acute in nature, involving short-term exposures and complaints such as irritations of the eyes, nose or throat. Other problems are chronic in which repeated exposures, sometimes over several years, have caused effects upon internal organs or have involved occupationally-related allergies.

Studies have shown that wastewater treatment may generate aerosols containing microbiological and chemical constituents. In fact, the primary route of exposure for workers is probably inhalation. The physical layouts of many sewage treatment plants involve open tanks and basins; plants typically are not designed to prevent aerial dispersion of wastewater during the treatment process. Volatile organics in wastewater may be vaporized or air-stripped during treatment. Many of the compounds are carcinogens and/or mutagens, so sewage workers may be at increased risk of cancer or adverse birth outcomes.

Infections from exposure to waterborne disease organisms may be subclinical or may appear as actual disease in wastewater workers.

Treatment personnel have reported nausea, vomiting, indigestion, diarrhea, and flu-like complaints. Studies of antibodies in the blood of workers have documented that disease exposures have occurred.

Although several years of exposure tends to produce eventual immunity for many workers to some organisms, new workers tend to be ill more often than experienced workers.

This manual examines how exposure occurs during the treatment processes; ways to reduce exposure by engineering controls, administrative controls, process control strategies, and protective equipment; and some suggested medical surveillance.

A single sewage treatment plant may service a hundred or more industries; therefore an enormous range of chemicals may be present in the influent and sludges. The presence of toxic chemicals and organisms in sewage, in sludge, and in the air at specific sites in sewage plants has raised suspicion regarding their possible effects on the health of the workers in these plants.

Wastewater treatment plant workers may be exposed to chemicals or organisms by direct contact with wastewater and sludges, or by inhalation of gases, particles, aerosols, vapors, or droplets. These hazards may enter the plant in soluble form or attached to suspended solids. Compounds reported from sludge analyses include chlorinated organic solvents and pesticides, PCBs, polycyclic aromatics, petroleum hydrocarbons, flame retardants, nitrosamines, heavy metals, asbestos, dioxins, and radioactive materials. The concentration of organics and metals in sludge is indicative of the areas' industries; for example, high concentrations of PCBs in Schenectady, NY, sludge was due to the manufacture of electrical equipment upstream from the treatment plant. There are also derivatives of chemicals formed by microbiological or other processes during the sewage treatment process; these may be more or less toxic than the original compound. Disease-causing organisms have been found in sewage sludge; therefore, sewage workers may be at increased risk of infection or diseases.

1. How are wastewater workers exposed to chemicals or diseases?

INHALATION appears to be a major route for chemicals or organisms to enter the body. Some chemicals are air-stripped from wastewater and workers working near weirs, aerated tanks, dewatering processes, and other sludge processes (drying, compacting, incineration). Aeration and dewatering processes also put droplets and particles into the air which can be inhaled. Much of the material inhaled into the throat or bronchial tubes is cleared from the lungs and swallowed. As a result, respiratory and gastrointestinal exposure can occur from inhaled chemicals and organisms. Wastewater workers have also been exposed to chemicals while attempting to remove these substances from treatment plant equipment.

SKIN CONTACT is also a route of entry for both chemicals and disease. Chemicals can be absorbed through the skin from contact with wastewater or sludge. Disease organisms can also enter the body through cuts or abrasions. There has also been a report of a wastewater worker who received a needlestick injury when removing screenings from a bar screen.

2. What kind of exposures occur for different kinds of treatment processes?

THE POTENTIAL FOR INHALATION IS DETERMINED BY THE WATER-SOLUBILITY OF A CHEMICAL.

When air is passed through water, or if that water is splashed into the air, some chemicals will be removed from the water and transferred into the air: this is called air-stripping. Chemicals which tend to be very soluble in water are more likely to be air-stripped from pure water or natural waters; as, for example, during aeration of drinking water. However air-stripping does not appear to occur quite so readily from wastewater because water insoluble compounds tend to attach to sewage solids. As a result, in sewage, compounds which are somewhat water-soluble appear to be air-stripped early in the process, such as from the grit chamber weir, primary clarifier weir, or during aeration in the grit chamber itself. Compounds which are very water-insoluble tend to be released to the atmosphere during

later treatment steps such as aeration in the activated sludge process. If a volatile chemical is biodegradable, its emission may be even higher from the primary clarifier weir than from the aeration basin where it is biodegraded rather than air-stripped. Emissions from the weir are higher than from the clarifier surface.

This is important to the wastewater worker because it means that the water-soluble compounds will stay in the plant longer since they are attached to sludge. If a wastewater treatment plant tends to receive periodic slugs of fairly water-soluble organic chemicals, then covering the grit chamber weir (with ventilation for worker entry) may significantly reduce water exposure. Other alternatives include the collection and treatment of the off-gas; the use of holding tanks for the hazardous influent and reserving it for further, more specialized treatment such as wet-oxidation; or the addition of activated carbon to the wastewater to absorb these chemicals and prevent their air-stripping.

The water-insoluble organic chemicals which attach to sludge solids are returned to the head of the aeration basin (return activated sludge); thus, these materials may actually spend a considerable time in the facility, depending upon the sludge age. So, when a slug flow of a hazardous chemical is received, one cannot simply assume that volatile chemicals will be released only during the time required for the slug flow in the main wastewater stream to pass through the plant. Absorption onto sludge solids keep these materials in the plant for a much longer period of time and thus prolongs their release to the atmosphere. In terms of process control, air-stripping may be increased by increasing the aeration rate. On the other hand, maintaining higher concentrations of suspended solids in the system may slow the stripping rate, and thus lower the concentration of volatiles above the aeration basin, but lengthens the duration of the air-stripping.

When air-stripping occurs, the volatile chemical moves from an area of higher concentration (the sewage) to one of lower concentration (the air) until the air becomes saturated (that is, when the air holds as much chemical as it can). In the activated sludge process, the air-stripping of volatile organic chemicals seems to be greater in surface aeration systems than in

bubble aeration systems. The reason seems to be that in the surface aeration system, there is so much air flow above the basin that the air stream doesn't get the chance to become saturated, so there is virtually no limit on the stripping rate. Pure oxygen plants with covered aeration basins tend to be rather inefficient for the stripping of volatile compounds. Trickling filters with forced aeration are quite efficient for air-stripping.

In the bubble aeration system, the stripping rate is limited since the air bubbles quickly become saturated. Even if volatile chemicals are still available in the sewage, no more can be stripped. For bubble-type systems, stripping is more efficient for coarse bubbles than for fine bubbles. During bubble aeration, only a few percent of the air dissolves in the mixed liquor, most of the air escapes to the atmosphere as bursting bubbles. These bursting bubbles cast droplets into the air which evaporate to produce solid particles. Aeration also causes rolling motions of the mixed liquor and this splashing is another source of aerosol droplets. Studies of bursting bubbles have shown that about 40% of the particles from aeration are less than 10 microns in diameter and so they can be inhaled.

Aerosol formation and air-stripping may also occur at the treatment plant outfall; these may include the release of chloroform. To estimate the risk of disease from the aerosols of the plant effluent, it may be possible to draw upon studies of workers exposure to aerosols at wastewater landspraying sites.

So far we have dealt with chemicals coming down the pipe to the treatment plant, but we can actually produce some volatile chemicals ourselves within the water or wastewater treatment process. For example, when chlorination is used for disinfection or for odor control, humic materials in water or wastewater can react with chlorine (especially hypochlorous acid, HOCl) to form chloroform. Although chloroform is the major product, other chlorinated and nonchlorinated breakdown products of humic acids can be generated as well. Chloroform generation can be eliminated by considering alternatives to chlorine. Odor can also be controlled by using hydrogen peroxide or potassium permanganate.

Aerosol droplets are produced by aeration processes through the breakup of tiny water jets formed by the collapse of bubbles. When a bubble breaks, it ejects a droplet that is about one-tenth the diameter of the bubble which produced it. This process causes bacteria and viruses, as well as chemicals, to be ejected into the air. If the wastewater surface has an oily film, then the aerosol droplets also will have an oil film.

Aerosols and air stripping could also occur during some dewatering processes.

3. *What about exposure to other materials in wastewater such as metals, radioactive compounds, or asbestos?*

Metals are generally not air-stripped into the air in sufficient quantities to be significant. Metals tend to either accumulate in sludge or pass-through into the receiving water. Metal removal by activated sludge reflects not only the metals behavior in solution, but also the sludge age and whether the plant is acclimated to that metal. Metals are absorbed by activated sludge because they are attracted to the active sites of the floc's biopolymers. As a result, the metals content of waste activated sludge tends to be approximately 2 - 5 times that of primary sludge.

• Case history: mercury emissions

Mercury is a possible exception that may be air-stripped if it is present in the wastewater in sufficient concentrations. Mercury in the air has been studied at wastewater treatment plants including Utica, New York. Air concentrations varied enormously in this study since the plants receive different kinds of mercury compounds; but some plants did have organic mercury emissions above EPA limits.

For radioactive materials, it is useful to compare the half-life of the element to the plant's sludge age to approximate the wastewater worker exposure. Radioactive elements with short half-lives may be more hazardous to the worker since the element will be undergoing decay while it is in the plant. For a material with a long half-life, relatively little decay will be occurring

while that material is present in the wastewater or sludge. Radium appears to be effectively removed by activated sludge as well as by fixed growth biological treatment systems such as trickling filters or RBCs. Volatile radioactive compounds may be air-stripped.

Some sources of radioactive materials to be aware of include:

- ❖ Medical and industrial wastes released to sanitary sewers.
- ❖ Releases from medical treatment and research, including excretion by patients (for example, iodine - 131; 8-day half-life)
- ❖ Unauthorized releases from manufacturers, including NRC licenses
- ❖ Water treatment sludges from drinking water containing elevated concentrations of radium. Where this drinking water is treated by ion exchange, backflushing of the columns may be discharged to sewers.

Asbestos has been found in sludge samples from treatment plants across the U.S. The health effects are difficult to assess, but are probably minimal while sludge is relatively wet. The risk of inhaling fibers may exist for handling and bagging of dried sludge or possibly compost used for horticultural work or sold as fertilizer.

4. *Do workers exposed to chemicals for short periods of time show any adverse health effects?*

Yes. As we saw above in air-stripping, the fate of inhaled materials also depends to a great deal on their solubility in water. Highly soluble gases and substances dissolve in the mucous membrane lining of the nose and throat resulting in irritation of the upper respiratory tract. In high concentrations, this may be noticed by the worker almost immediately. Upper respiratory tract irritation is almost always accompanied by eye irritation as the materials dissolve in the fluid coating the conjunctival membrane of the eye. Less soluble gases result in irritation of the deeper structures of the lungs. Very poorly soluble substances may pass directly from the inhaled air into the blood stream and result in systemic poisoning or anesthetic effects if the agent can cause central nervous system

depression. As a result, the insoluble gases have little or no effect on the organs of respiration but may produce severe systemic toxicity. So, materials which might not cause irritation would not alert the worker to their presence, but could cause serious health effects.

Wastewater workers have been exposed to chemicals in acute (short-term) episodes. In fact, in some cases, it was the workers' reports of health effects which alerted the plant that something unexpected was being discharged to the treatment facility.

- **Case history:** Morris Forman WWTP, Louisville, KY. Acute exposure of wastewater workers to an unauthorized discharge to the municipal sewer of hexachlorocyclopentadiene (HCCPD), an intermediate in pesticide manufacture. When an unidentified material entered the plant, workers noticed an objectionable and odoriferous, sticky, viscous material collected on the bar screens and grit collectors. When workers tried to remove it with steam, a blue haze formed which spread throughout the primary treatment area and sent 20 of the workers at the primary plant receiving industrial sewage to the hospital. Of these workers, 74% experienced central nervous system symptoms such as headache, lightheadedness, fatigue, and increased need for sleep from exposure to benzene, toluene, and other organic solvents.

- **Case history:** North Wastewater Treatment Plant; Memphis, Tennessee. Similar exposure to the Morris Forman plant (above). Industrial discharger producing and using chlorinated organic chemicals for synthesis of flame retardants and pesticides (especially isodrin, endrin, chlordane, and heptachlor). Workers at the treatment plant complained of respiratory distress, dizziness, headache, and irritation of the eyes, nose, throat, lungs, and skin when an intense chemical odor was present. One night a pesticide spill occurred and six workers experienced severe headaches, nausea and loss of equilibrium. Air samples at the wet well and grit chamber confirmed the presence of these chemicals. Urine specimens of plant employees showed that they had inhaled them.

5. *Do wastewater workers show adverse health effects from chemicals after years on the job.*

There is little information on this so far.

■ **Case history:** Water and wastewater worker exposure has also been studied by testing worker urine for the presence of mutagenic substances. In one such study, the frequency of urinary mutagens was measured in water and wastewater workers employed in 14 plants in NYS processing 3 - 10 mgd. The results indicated that a higher frequency of mutagens was present in wastewater workers as compared to water treatment workers, suggesting a higher frequency of exposure to toxic chemicals. This may mean a higher risk of adverse health effects (e.g., cancer and birth defects), but it is difficult to assess the health significance. The measurement of mutagens in urine at one point in time may not reflect worker exposure on a daily basis or overall. The presence of mutagens as determined by a bacterial assay does not establish that mutations are taking place in human cells or that cancer might ultimately result.

■ **Case history:** Bloomington, Indiana, municipal sewage treatment plant. For example, in a case study of a trickling filter plant, when appreciable quantities of PCBs are present in raw sewage, significant quantities can pass through the plant into the effluent. But, because PCBs are not very soluble in water, they mostly tend to concentrate in sludge and in the sediment of the receiving stream. They can bioaccumulate in fish making it inedible and can accumulate in soil fertilized with PCB-contaminated sludge. PCBs adsorb readily onto silt particles and solids, therefore, bottom materials in sewers may be expected to contain appreciable quantities of adsorbed PCBs. Workers without gloves and handling PCB-contaminated sludge, have absorbed enough PCBs through their skin that their blood levels of PCBs resembled the levels found in people working in the PCB manufacturing facility which was sending the PCBs to a treatment plant.

Sewage sludge and sewer lines contaminated with waste PCBs from electrical capacitor manufacturing plant. Gardeners, farmers, and wastewater workers exposed to PCB-contaminated sludge showed that

blood serum PCB levels increased with the percentage of garden care performed by the sludge user and decreased with the wearing of gloves while gardening. Six workers at the sewage treatment plant had levels of 6ppm (lipid basis) compared to national surveys in which 5% of the population show levels higher than 2 ppm.

■ **Case history:** Study of mutagens in municipal sludges from 34 American cities (including Buffalo, NY). Thirty-three exhibited positive mutagenic response. Seventy-six percent of the positive responses required metabolic activation, indicating that the sludge materials are converted to mutagens when taken into the body.

■ **Case history:** Study of mutagens in municipal sludge from Chicago, Illinois. Three species of organisms showed mutagenic responses when exposed to the sludge.

■ **Mortality study:** Copenhagen, Denmark. Wastewater workers showed increased mortality in first year of retirement from cancer of pancreas as most common form of death. The wastewater treatment plants studied serviced chemical manufacturing plants. This was, however, a small study of the population.

■ **Mortality Study:** Metropolitan Sanitary District of Greater Chicago, Illinois. This wastewater treatment plant worker study showed no increased occurrence of cancer overall or other particular causes of death; but there were too few workers in the study to evaluate trends for specific cancers.

■ **Mortality study:** Buffalo Sewer Authority, Buffalo, New York. This wastewater treatment plant worker study indicated that there may be some increased cancer risk overall, but no specific cancer sites were pinpointed.

6. *Do wastewater workers experience any adverse effects upon their reproductive function or upon their offspring?*

Little is known about reproductive effects in wastewater workers. A study was conducted for an oil company treatment plant which looked at miscarriage rates among the wives of wastewater workers to see if there was any work-related effect due to the father's exposure. Pregnancy outcomes were studied in 101 wives of sewage workers and suggest risk of fetal loss increased among sewage workers exposed around the time of conception; but these conclusions have been challenged since higher fetal loss normally occurs with increasing age of mothers under 40 years of age. Also, recall bias is a problem since the worker-exposed birth outcomes occurred from 1976-80, whereas the unexposed group was 1934-80. This study also involved sperm count and sperm morphology among men working throughout the refinery which showed no differences from the wastewater plant workers. A recent study published in 1991 looked at reproductive outcomes in fertility in male wastewater treatment plant workers and their wives, as well as evaluating sperm and semen in the workers. The results were compared to water workers and their wives. Spontaneous early fetal loss did not appear to be related to wastewater exposures.

7. *Are workers exposed to diseases by inhalation?*

Inhalation itself may lead to a respiratory infection or the respiratory mucous laden with trapped pathogens may be swallowed so that the infection actually occurs in the digestive tract. Aerosols might contaminate food or water and lead infection in the digestive system. Organisms which can infect the lungs include *Mycobacterium tuberculosis* and some of the enteric viruses. It is difficult to study health effects from aerosols since the treatment plant is located within the area it serves: the ultimate sources of the pathogens in the aerosol are infected individuals in the service area. It is difficult to determine if the route of transmission for a disease was wastewater contact or contact with other people.

The amount and survival of microorganisms in wastewater aerosols depends upon the amount of the organism in the wastewater, aeration basin, or

sludge; the amount of material aerosolized; what happens to the aerosol while in the air (such as drying of moisture or impact with a surface); and the die-off of the organisms with distance in the downwind direction. Even when these circumstances are known, interpreting the health risk from the information is difficult since it is necessary to consider what is the quantity of a particular pathogen required to start an infection in people.

Bacteria and viruses are not necessarily evenly distributed throughout a liquid, but can concentrate in the surface microlayer. This affects how many organisms can be put into the air. During aeration, when air bubbles break at the air-water interface, microorganisms are ejected into the atmosphere. The bacterial concentration in the ejected drops from bubbles may, depending upon the drop size, be from 1 to 1000 times that of the water from which the bubbles burst.

Wind is the most important environmental factor that determines the aerosol spread of the pathogens. When in an aerosol, the survival of the organisms depends upon relative humidity, temperature and sunlight. These factors cause a die-off of microorganisms: the indicator organisms appear to be affected by this shock of aerosolization than are the pathogens, (*Pseudomonas*, *Streptococcus*, *Clostridium perfringens*, *Mycobacterium*, and enteric viruses).

Typically, indicator organisms are used to indicate the possible presence of associated pathogenic bacteria and human viruses because the pathogens are difficult to assay and seldom occur at readily detectable concentrations. Studies have shown that there can be a very poor correlation between pathogens and indicator organisms. Also, changes in indicator organisms may not relate to changes in pathogen concentrations. Fecal streptococcus may be a better indicator for aerosolized wastewater. The measure of the spread of pathogens by aerosols can be done rapidly and inexpensively by monitoring cyanophages, viruses that attack the blue-green bacteria (cyanobacteria). The LLP-cyanophages are not fecal organisms but may be good indicators of water pollution since they appear in polluted water when pathogens are present, survive longer than pathogens (more resistant to chlorination than coliforms), and are detected by a simple test. Diffused

aeration activated sludge causes little aerosol release of cyanophages in comparison to the mechanical aeration.

A trickling filter also produces aerosols while the filter is in operation; the more filters a plant has, the higher the bacterial content of the air downwind of the filter. A study showed that both coliform bacteria and total organisms are higher in the air at night than during the day, showing the effect on bacterial death. Higher airborne levels of organisms were present when the relative humidity was above 35% or during high wind speeds.

8. *What kinds of diseases are wastewater workers exposed to?*

The waterborne diseases include viruses, bacteria and protozoa (parasites) causing a variety of conditions including:

Disease

Agents

1. VIRAL

Gastroenteritis ("24-hour flu")	Enteroviruses (67 types), Rotaviruses, Parvoviruses, Reoviruses, Astrovirus, Calcivirus, Norwalk agent
Infectious Hepatitis	Hepatitis A Hepatitis B (see text for case history)
Serum Hepatitis (cirrhosis, liver cancer)	
Aseptic Meningitis	Coxsackiviruses, Echoviruses
Respiratory Disease	Adenoviruses (31 types), Reoviruses, Coronavirus
Poliomyelitis	Polioviruses

II. BACTERIAL

Salmonellosis, Typhoid Fever	Salmonellae (approx. 1700 types)
Shigellosis	Shigellae (4 spp.)
Cholera	<i>Vibrio cholerae</i>
Gastroenteritis	Escherichia coli (enteropathogenic types)

III. PROTOZOAN

Amoebic Dysentery, Ameobiasis	<i>Entamoeba histolytica</i>
Giardiasis	<i>Giardia lamblia</i>
Balantidiasis	<i>Balantidium coli</i>
Meningoencephalitis	<i>Naegleria fowleri</i>
	<i>Acanthamoeba</i>

BACTERIAL diseases are common to most countries; differences tend to be in the form of additional types of bacteria and a higher incidence in wastewaters in developing or tropical countries. Those bacteria sufficiently prevalent to be of concern in developed nations are *Salmonella*, enteropathogenic *Escherichia coli*, *Shigella*, and *Yersinia*; and to a lesser extent *Legionella* and *Leptospira*. *Salmonella* and *E. coli* are always found in sewage and sludge. *Salmonella* is present in domestic animals; only *Salmonella typhi* (typhoid) lives in man only. *Shigellae* (dysentery) is essentially exclusively a human disease. *Vibrio cholerae* (cholera) survives in water for only a short time; epidemics associated with contaminated drinking water show recent contamination. Shigellosis is the second most common enteric bacterial infection observed in the U.S.A. In 1976, the incidence rate for shigellosis was 6 per 100,000; salmonellosis at 11 per 100,000.

LEGIONELLA has been isolated from the water in the cooling towers of air conditioning units in association with disease outbreaks, suggesting that disease in man results from exposure to aerosols containing the organism. Wastewater-exposed workers and the neighbors of treatment plants have not shown that exposure posed a risk of infection.

SALMONELLA: The Cincinnati-Chicago-Memphis study showed higher levels in wastewater workers; especially among inexperienced workers in Memphis.

YERSINIA: A study of sewage workers in Manitoba did not reveal a wastewater-exposure effect.

BACTERIAL ENDOTOXINS: Toxins released from the cell walls of gram-negative bacteria after their death can produce fever and chest-tightness in exposed individuals. This appears to be a problem during sludge heat-treatment operations for sludge drying, at land application sites, and at composting operations. Individuals exposed to composted sludge in Philadelphia and Washington, D.C., showed higher antibody levels to compost-derived endotoxin.

HEPATITIS: Hepatitis A requires serological (antibody) analysis since most infections are subclinical and are not manifested as overt disease. Hepatitis B is a major unconquered disease: some 200 million people are chronic carriers of the virus and a significantly minority of these go on to develop cirrhosis or cancer of the liver. There are reliable diagnostic procedures and a vaccine. Hepatitis B is transmitted via blood or blood-contaminated materials; blood from skin and wounds or sores; tattooing acupuncture, or ear piercing without rigorous sterilization of equipment. Recent evidence indicates that sexual transmission is possible; semen and other genital secretions can transmit the virus; and saliva also contains the virus. There are strict disposal procedures for contaminated materials.

AIDS: The Aids virus does not appear to survive outside the body. For infection to occur, considerable numbers of viruses must be involved. For example, of 1100 hospital workers who have experienced needle punctures with AIDS-contaminated materials, only 2 have developed antibodies and these involved deep intramuscular puncture wounds. For AIDS to be transmitted via sewage would involve blood in the urine or feces of the infected individual to be discharged in the sewer. Infection would have to involve contact of this material with cuts or broken skin. The AIDS virus may live for an hour in blood specimens kept at room temperatures which are close to body temperatures; otherwise, the virus seems to die off at cooler temperatures. This would seem to mean a low chance of survival in the sewer system. As with hepatitis B, there are strict disposal procedures for contaminated materials.

NORWALK VIRUS is largely associated with epidemics of gastroenteritis in older children or adults. Infections are infrequent (<5%) in children under 10 years; 50 - 60% of adults have serological evidence of infections.

- **Case history:** Manitoba wastewater treatment plant workers showed a higher level of antibody to reovirus, an enteric virus.
- **Case history:** Cincinnati, Ohio; Chicago, Illinois; and Memphis, Tennessee (Cincinnati group included sewer maintenance workers). Antibody levels detected were less than those expected: 9 of the 10 significant differences in antibody levels were higher for the more exposed workers for both contact with wastewater and/or sludge and exposure to bacterial aerosols. The levels of Norwalk agent antibody were higher in workers with high and medium aerosol exposure than in low aerosol exposure. Black workers showed higher levels of antibody to Norwalk agent and to hepatitis-A than white workers. Although studies showed little risk of viral infections in wastewater workers; when the results were analyzed by degree of exposure within worker groups, antibody levels for enteroviruses and Norwalk agent suggested a wastewater effect; and antibody for echoviruses 3 and 6 were higher in exposed workers.
- **Case history:** Anchorage wastewater workers showed a prevalence of antibodies to three respiratory viruses (adenovirus, parainfluenza type 1, and influenza type A); a possible sewage exposure-related effect.
- **Case history:** Bucharest wastewater workers showed a prevalence of antibodies to three respiratory viruses (adenovirus, parainfluenza type 1, and influenza type A); a possible sewage exposure-related effect.
- **Case history:** Copenhagen sewer workers: hepatitis-A antibody found more often than among other workers; limited risk of enteric infection due to municipal sewage exposure.

PARASITES: The major threats of disease transmission through sewage are *Entamoeba histolytica* (dysentery) and *Giardia lamblia* (giardiasis). Amebiasis is probably the most common fatal parasitic infection in the USA; 1.35 cases per 100,000 in 1976. The most common protozoan parasitic disease in USA is *Giardia lamblia*. Waterborne epidemics tend to occur via contamination of the local water supply by sewage infiltration into water distribution system. Antibody studies may not be the best approach

for studying parasitic infections in populations since antibodies may not appear in the blood of individuals whose stools indicate infection.

- **Case history:** Rennes, France. Higher *Entamoeba histolytica* and *Giardia intestinalis* (*G. lamblia*) concentrations were found in stools from sewer workers.
- **Case history:** Hamburg, W. Germany. Sewage workers showed an increased infection rate for *Entamoeba coli*, *Endolimax nana*, *G. lamblia*, and all protozoa combined. An incident occurred in Hamburg in which a wastewater worker developed an amebic liver abscess attributed to swallowing wastewater. The authors of these studies feel that consideration should be given to the recognition of amebiasis and giardiasis as occupational diseases for wastewater workers.

FUNGI: Aside from the composting risk discussed below, fungi are not potentially pathogenic to man in the same context as bacteria, viruses, and parasites. Some species have been implicated as secondary agent of disease, as agents of hypersensitive reactions, and as producers of mycotoxins from composting or sludge heat-treatment operations or at land application sites. The potential risk for sludge composting workers is for fungal spores of *Aspergillus fumigatus* inhaled into the lungs. Compost-exposed workers from Washington, D.C., and Philadelphia showed a consistent increase in antibody to this mold.

EFFECT OF TREATMENT ON PATHOGEN CONCENTRATIONS AND SURVIVAL.

As effluents become cleaner, the volume of treatment by-product has increased in direct proportion. Since sludge is composed of the materials removed from liquid waste as it progresses through various treatment processes: the cleaner the effluent, the more contaminated the sludge.

Fate of Pathogens:

WORKER EXPOSURE AND INFECTION/DISEASE

Workers may be exposed by (1) inhalation of wastewater aerosols, by (2) direct contact with wastewater or with sludge, by (3) ingestion of food or water contaminated with wastewater or sludge (or accidental ingestion of wastewater or sludge itself), or (4) cuts, punctures, etc. contaminated with wastewater or sludge. Infection with an enteric organism can be **CONFIRMED** by the worker's medical history or by showing that more of the disease organism is shed in the feces than was originally received by the worker; or infection can be **INFERRED** if the worker begins to produce antibodies against the disease.

There is little data that indicates that workers or nearby residents to a plant have actually become ill because of inhalation of pathogens in wastewater aerosols or from other wastewater/sludge contact, even though waterborne diseases are certainly transmitted via the contamination of drinking water with sewage. Some studies are controversial as to whether sewage workers are at a higher risk of various infections/diseases than the general population. Moreover, exposure to wastewater aerosols occurs at home, since the household toilet generates aerosols when flushed. Workers engaged in sewer maintenance and wastewater treatment are exposed to a wide variety of routinely found disease-producing microorganisms, but, in spite of this exposure literature searches have revealed little evidence of occupational health problems associated with wastewater pathogens. Most studies show that risk of infection from exposure to wastewater or sludge is minimal. There is evidence that exposure carries a slight risk of viral infection, particularly in workers with the highest exposure levels. Overall, researchers tend to show surprise at having encountered so little disease among wastewater workers.

Beside examining the medical history of wastewater workers, past exposure to disease organisms can be determined by measuring antibody levels. Measurements of serum antibody levels have been used in many studies

during the past 10 years to see if workers have a history of prior infection with pathogenic viruses, bacteria, and certain parasites and fungi. Higher antibody levels frequently occur in inexperienced workers (<5yrs.) than experienced workers.

- **Case history:** North Side Sewage Treatment Works; Skokie, Illinois. Study in which particulates from sewage and air samples collected at the plant were inoculated in guinea pigs. In one experiment using

undiluted sewage, *Legionella pneumophila* was identified in spleen cells 6-7 days later. Infections were not detected in animals inoculated with aerosol samples.

- **Case history:** Cincinnati, Ohio; Chicago, Illinois; Memphis, Tennessee. To determine if the prevalence and level of antibodies were higher in wastewater-exposed people and whether the number of infections as indicated by increases in antibody concentration were different among the various study groups. Study showed that wastewater workers are not at significantly greater risk of disease nor source of viral infections to family members. In a few instances, levels of antibody to certain viruses appeared related to level of exposure to wastewater aerosols. Bacterial aerosol levels higher in sludge handling buildings than adjacent to aeration tanks at the same plant.

HELMINTHS (worms, cysts, etc): The most common parasitic helminths in the USA are *Ascaris lumbricoides*, *Trichuris trichiura* (whipworm), *Necator americanus* (hookworm), and *Taenia saginata* (beef tapeworm). The potential sources of parasites in municipal sludge are untreated wastes from slaughterhouses and meat and poultry plants. *Ascaris* ova (eggs) are extremely resistant to treatment processes and are used as indicator organisms of parasite contamination and the survival of parasites in sludge. The number present in the human host is dependent upon the number of ova ingested, because the worms do not reproduce in the body. Tapeworm infections can spread by livestock grazing on soils containing sludge in which eggs are present; or man can ingest the eggs.

9. Do treatment processes reduce or kill disease-causing organisms?

The disease-causing organisms found in sewage are almost entirely of human origin and are mostly of enteric forms; that is, organisms which infect the human intestinal tract, although a few cause respiratory infections. The microorganismal content of raw sewage reflects the type and number of infections experienced by the community served by the sewage system; this will vary from community to community and country to country. It is not necessary for people in the community to show disease symptoms; viruses, for example, are shed in large quantities in feces of even healthy carriers. The types and numbers of pathogens decrease drastically in the course of conventional sewage treatment, so the health risks will vary throughout the process and will depend upon the type of treatment.

The disease organisms which require a host in which to reproduce are not going to be able to reproduce in the sewer system. This means that when these organisms are shed into the water by the infected person, the number of organisms may decrease (die-off as well as dilution) or remain constant during passage down the pipe to the treatment plant, but will not increase. Also, the risk of disease, especially by the skin contact route, may depend upon the concentration of disease organisms in the sewage. The closer to the source, the higher the concentration since little dilution will have occurred. Also, some organisms, including the viruses, may adsorb to sewer films or settled solids and be of risk to sewer workers. The dilution factor for feces in household or community sewage is in the range of 1000 to 10,000. (For example, a pathogen present in the feces of 1-10% of the members of a community at a level of $10^8/\text{g}$ might occur in raw sewage at levels of 10^5 - $10^7/\text{liter}$.)

Overall, total fecal coliform analyses probably do not provide an accurate microbiological profile for the survival of pathogens in sludge.

In general, some pathogens such as *Salmonella typhosa* (salmonellosis) have a short survival time in wastewater; *Mycobacterium spp.* (tuberculosis and others), *Ascaris ova* (roundworms), and certain enteric viruses appear highly resistant to treatment processes.

PRIMARY TREATMENT does not significantly reduce pathogens, although primary sludges may contain large numbers of parasite eggs.

Most parasites present in raw sewage are found in primary sludge, including pathogenic bacteria and viruses due to affinity with settleable particles. It is disputed whether viruses are removed almost exclusively in primary sludge; there is too much variation and uncertainty in the methodologies used to study viruses. Primary treatment tends to remove 80 - 90% of Salmonella; 50% of Mycobacterium; and coliform removal varies from 27 - 96%.

SECONDARY TREATMENT: As previously discussed, biological treatment may have a destructive effect on the viruses; removals in activated sludge range from 50 - 90%. Activated sludge has a low removal for eggs and cysts; removals range from 85 - 99% for pathogenic bacteria. Waste solids do contain surviving pathogens. Secondary sludge may be expected to contain a major portion of the microbial population which was removed from the raw sewage. In activated sludge, viruses are removed by adsorption to flocs; however some sewage microflora are capable of virus inactivation. This may be due to metabolic products released by sewage organisms or to toxic substances present in wastewater. Some bacterial enzymes break down the viral protein coat of viruses such as Cocksackie, Flavobacterium, Aerobacter; and Klebsiella bacteria can inactivate poliovirus in activated sludge.

ANAEROBIC DIGESTION appears to reduce coliform populations considerably, but other pathogens such as Mycobacterium and Ascaris ova can withstand prolonged digestion. For viruses, inactivation rates ranged from 74.9%/day for Echovirus 11 to 90%/day for poliovirus to 97%/ day for Cocksackie virus A-9. This appears to be due to bacterial digestion of the virus' protein coat. Although significant pathogen reductions can be achieved by anaerobic digestion in laboratory studies, actual plant digesters appear less efficient - this may be due to the continuous input of contaminated raw sludges along with possible short-circuiting and incomplete mixing. After digestion for sludge subsequently dried in lagoons; research from Ottawa, Canada, has shown that even after eight months of lagoon-drying, sludge still contained detectable amounts of viruses.

OXIDATION PONDS: Pathogens entering oxidation ponds may be removed by flocculation, inactivated by sunlight, or inactivated by pond microflora, especially protozoa, rotifers and nematodes. Sunlight is probably the significant factor due to ultraviolet rays. The lethal effect of ultraviolet light is due to the absorption of UV by viral proteins and nucleic acids (DNA or RNA) which leads to changes in the protein structure and to breakage of the nucleic acids. (For example, for raw sewage exposed to sunlight for 5.5 hours during a four-day period, poliovirus 1 was reduced to zero in the upper 1 inch of sewage. At lower depths, the virus was more stable.) Ponds remove a significant portion of indicator and pathogenic bacteria, but viruses are not completely removed.

DISINFECTION, specifically chlorination, of effluents has yielded studies which lack specific information on initial chlorine dosage, reaction temperature, pH, and the level of organic or inorganic nitrogenous compounds present. This makes it difficult to compare published results of chlorination studies. However, some microorganisms, such as species of *Mycobacterium*, amoebic cysts, and certain enteric viruses (including hepatitis A), are reported to be more chlorine-resistant than indicator coliform organisms. For example, chlorinated secondary effluent has been found to contain 50 viruses/l. However, without chlorination, 1-10% of *Salmonella*, *Mycobacterium*, and some enteric viruses are probably surviving in the effluent after secondary treatment.

SANITARY LANDFILLS and LEACHATE: Diapers are a major source of enteric viruses in sanitary landfills. Viruses may be adsorbed to solid waste components or the landfill may have some inactivating property (e.g., heavy metals) or high temperatures during decomposition of waste (60°C). Whatever the reason, virus inactivation increases with increasing ages of landfill. As a result, landfill leachate tends to have little or no viruses.

10. How can we find out whether wastewater workers are exposed to diseases?

Organisms have been found in workers themselves using throat or rectal swab samples or examination of feces. This kind of evidence confirms that infection has actually occurred.

VIRUSES: All viruses that infect the enteric tract are shed in the feces. Although large numbers of viruses which infect the respiratory tract are swallowed, they tend to be inactivated by stomach acid or by the bile salts encountered in the intestine. A number of viruses are shed in the urine, such as mumps and measles, cytomegalovirus, and congenital rubella. Viruses may be shed by individuals who never develop disease (subclinical infection), or during the last day or so of the incubation period, or form chronic carriers whose infection persists long after evidence of the disease disappeared.

Enteric viruses do not grow in the external environment and appear generally to have a limited life span in soil and water. Although enteroviruses have been infrequently identified in sewage aerosols so far, they have been found in abundance in sewage. Primary and trickling filter treatments have only a modest effect on virus levels in sewage; while activated sludge can remove 90-98%, there is concern that some viruses may be aerosolized. The respirable particles may become trapped in the nasopharynx of exposed persons, an appropriate site to initiate an infection.

Poliovirus was once the most feared of all viral diseases, but the impact of the Salk and Sabin vaccines has been so effective that it has been virtually abolished in Western countries. Poliovirus is spread by direct fecal contamination of hands or contamination of water supplies by sewage. Modern standards of hygiene and sanitation have restricted its spread; wild viral strains are rarely found in human sewage, but the vaccine strains are common.

Other enteroviruses (Coxsackie virus and Echo virus) are common viruses causing a variety of diseases such as upper respiratory tract infections, gastroenteritis, meningitis, paralysis, pancreatitis, myocarditis, endocarditis, and hepatitis A. They enter through the mouth and are present first in the throat and then in the feces for some days.

A study of stool samples of sewer workers in Rennes, France, found the protozoan *Entamoeba histolytica* in 11% of sewer workers, but only 2% of non-sewer workers. In the same study, 16.5% of the sewer workers had

Giardia Intestinalis (now called *G. lamblia*). Hamburg, Germany, sewer workers have found to have increased infection rates with *Entamoeba coli*, *Endolimax nana*, and *Giardia lamblia*. Antibodies to these organisms have been found in the blood of workers. A study of workers at wastewater treatment plants in Cincinnati, Chicago, and Memphis showed higher levels of antibodies to Norwalk agent, rotavirus, and echovirus 3 and 6 than in unexposed people. Further, the inexperienced workers showed even higher levels than experienced workers.

Antibodies to adenovirus, influenza type A, and parainfluenza type A were studied in wastewater workers in Bucharest. The more exposure they had to wastewater and sludge, the higher their antibody levels to respiratory viruses. Wastewater workers in Copenhagen have shown higher levels of antibodies to hepatitis A when compared to nonsewage workers. Studies of workers at sludge or wastewater land application projects indicated seroconversions (antibody levels) to enteroviruses, Cocksackie B, echoviruses, and polioviruses for Ohio; Lubbock, Texas; Muskegon, Michigan; Sweden; and Israeli kibbutzim. However, the results were not clinically significant except in workers with high wastewater exposure who cleaned the irrigation nozzles. Workers in Memphis had higher antibody levels to Salmonella than workers used as controls. Workers at facilities in Philadelphia, Camden, Washington, D.C. and Beltsville were involved in heat-drying of sludge or sludge composting have high antibody levels to endotoxins released from the cell walls of Gram-negative rod-shaped bacteria. Workers at sludge composting sites may have some risk from inhalation of spores of the fungus *aspergillus fumigatus*. Some fungal species may cause hypersensitivity reactions or produce mycotoxins (similar to endotoxins).

However, it is possible for exposed workers to have protozoa in their stools and yet not have detectable antibody concentrations for that organism. For example, this was the case for some Hamburg, Germany, sewer workers infected with *Entamoeba histolytica*. Researchers have recommended that serological (antibody) tests may not be the best way to study parasitic infections.

Studies undertaken to test for disease exposure can include:

- Skin tests for tuberculosis and fungal infections
- Liver function tests for hepatitis
- White blood cell (leukocyte) counts
- Urinalysis for fibrinogen degradation product (FDP) concentration
- Air samples taken at treatment plants to show the presence of wastewater organisms in the air at and near the facilities.

One example of the last kind of study was conducted at the John Egan Plant, Schaumburg, Illinois, prior to and after the initial operation of this new plant. The study examined pathogenic bacteria, viruses, parasites in clinical specimens, viral antibodies in serum, specimen trace metal levels, and the reported incidence of relevant disease and symptoms. A household health survey was conducted for persons living within 5 kilometers of the plant. Air and wastewater were sampled for pathogens and metals. The levels of microorganisms and metals in the neighboring residential areas were not distinguishable from background levels. The nearby residents did report a higher incidence of skin disease and several gastrointestinal symptoms after the plant became operational, but antibody tests and attempted isolations of pathogenic organisms showed virtually no clinical evidence of disease associated with wastewater aerosol. In fact, some surveys have shown that people who live near wastewater treatment plants show higher levels of some of the same diseases, the closer they live to the wastewater treatment plant.

11. Do wastewater workers actually get sick from disease exposure at work?

Yes, occasionally they do. They need to take in an infectious dose by inhalation or ingestion; few organisms can penetrate the skin. Many illnesses and infections are subclinical and discovered by antibody levels or other tests, but actual diseases have occurred. These can be verified by looking at shedding of the organisms in the feces.

LEPOSPIRA bacteria enter the body through cuts and abrasions of the skin or by contact with the mucous membranes of the nose, mouth, or eyes. It has been considered an occupational health risk of wastewater workers in the British Isles and Germany. This has been especially true for sewer maintenance workers; the route of entry appears to be direct contact of cuts and skin abrasions with urine infected rats. The incidence among sewer workers declined from 8% (1933-1948) to 2% (1978-1983); this has been attributed to modern pest control measures, the use of protective clothing, and the presence of detergents in wastewater which rapidly destroy *Leptospira*. The Cincinnati-Chicago-Memphis study (cited earlier) showed some antibodies present in wastewater workers.

Since *Leptospiral* antibody levels fall rapidly after the infection is over, the detection of the antibody depends upon how close the study is done to the time of infection. Failure to detect *Leptospiral* infection in sewage workers may be due to lower background levels of antibody, lower exposure to wastewater, or both. The recent studies in general suggest that although some risk remains, *Leptospirosis* is no longer a major problem in sewage workers.

Wastewater workers in Cincinnati, Ohio; Anchorage, Alaska; and Ontario, Canada; have contracted hepatitis A. The Ontario workers worked in a primary purging station, grit chambers, and maintenance on sewer cleaning machines. The Ohio workers included construction workers at the wastewater treatment plant and a chemist. Researchers in the Hamburg, Germany, wastewater workers study recommended that amebiasis and giardiasis be considered as occupational diseases for wastewater workers. Endotoxin exposure at sludge-drying composting facilities has produced fever and chest-tightness reactions in exposed workers.

Parasitic worms have not been studied yet in wastewater workers. Overall, researchers have tended to conclude that the risk of viral diseases, based upon antibody studies, appears low. However, many of the infections studied in wastewater workers have involved sporadic epidemics which are more difficult to investigate.

Bacterial diseases appear to also show a low risk for wastewater treatment generally, but heat-drying and composting of sludge appear to carry some risk of endotoxin exposure and its effects. Workers at a wastewater treatment plant in Gothenburg, Sweden, a heat-drying operation where sludge was converted to dust, experienced episodes of chills, fever, and malaise. Also white blood cells moved into the eyes to fight eye inflammation. All these are typical symptoms of endotoxin exposure.

Workers during their first years of employment, especially the first 5 years, may experience increased rates of gastrointestinal or upper respiratory illnesses. This may be because time is needed for their immunity to become established.

12. What about the risk of contracting hepatitis B or AIDS?

There have been few studies of these diseases; but, so far, studies of antibody levels in wastewater workers have shown hepatitis B does not appear to be effectively transmitted via exposure to wastewater itself. However, in the 1983 annual Safety Survey conducted by the Water Pollution Control Federation, one utility reported that a worker had received Workers' Compensation for a hepatitis B infection. During the 1990 hearings on OSHA's proposed standard on bloodborne disease (due to become a final rule in 1991 or early 1992), a sewage worker from New York City told of contracting hepatitis B from a needlestick while screening debris from raw sewage in 1987.

The Center for Disease Control (CDC) allows Human Immunodeficiency Virus (HIV) contaminated blood and body fluids to be carefully poured down a drain leading to a sanitary sewer because they believe that wastewater workers are not at increased risk of contracting bloodborne diseases. Wastewater temperature, pH shifts, chemicals, and dilution itself are believed to quickly inactivate the HIV virus. From 1990 to 1993, several studies have been published which suggest that this conclusion is probably correct. HIV needs an intact outer envelope to be infectious; its outer membrane is susceptible to disruption under conditions of unfavorable

osmotic pressure (such as the virus coming into contact with tap water or wastewater, which are so different from human blood or body fluids). An experiment in which blood from State IV AIDS patients was mixed with dechlorinated tap water indicated that the osmotic balance was so severely affected that virally infected cells were no longer detectable after 5 minutes. Once membrane rupture occurs, noninfectious HIV can still be detected by using techniques to recover viral RNA or proviral DNA. The use of the polymerase chain reaction (PCR) technique enables very tiny amounts of RNA or DNA to be recovered from wastewater, amplified, and then detected. PCR techniques have needed to amplify the virus a millionfold or more to be able to detect it (even at picogram levels); this supports the conclusion that the wastewater concentration of HIV is exceedingly low.

But does this indicate that infectious virus is present? The results of one such study appear to indicate that, although the PCR method can detect the presence of the virus's RNA and related DNA, this does not necessarily indicate the presence of infectious viral particles. While recoverable RNA can persist for a few hours, HIV appears to have lost its infectivity within minutes. The presence of blood serum in water can only briefly slow the inactivation of HIV. Further, it would also appear that wastewater components inhibit the action of the viral enzymes reverse transcriptase and DNA polymerase, also needed for viral infectivity.

It is important to note that HIV is quite different from the traditional waterborne viral diseases discussed in this manual. These viruses can replicate in high numbers in the gastrointestinal tract, enter and be stable in wastewater, and thus be transmitted to the wastewater worker via the (usually) respiratory route of entry and (for a few types) via breaks in the skin. However, the replicative cycle of HIV does not introduce it directly into wastewater.

Thus, the research so far still appears to indicate that the wastewater worker is not at increased risk of occupationally-related HIV.

13. Can a worker get a disease from an accidental exposure, such as sewage splashed into the mouth?

Yes, this is possible. A sewer worker in Hamburg, Germany, developed an amoebic liver abscess due to an accident involving the swallowing of wastewater.

For organisms which cause infection by way of the digestive tract, some may remain there and cause disease. But others must work their way to other parts of the body to cause disease. So, the outcome of an infection may be determined by how effectively the organisms are confined to the intestines, rather than by how rapidly the body suppresses the infection entirely. The enteric bacteria cause intestinal disease by associating with the inner surface of the intestines.

Some organisms (such as *Vibrio cholerae* which causes cholera) cause diarrhea by producing and excreting toxins that stimulate the intestinal cavity. Others induce diarrhea by entering, multiplying in, and destroying the epithelial cells which line the intestine. Infectious *Escherichia coli* can behave by either of the preceding mechanisms. Other microorganisms can penetrate through and around epithelial cells and then travel by way of the lymphatics and the blood to other organs in the body where they cause systematic disorders. In some of these cases, the bacteria colonize (multiply on) the intestinal surface. To survive there, bacterial pathogens must resist or bypass the body's defenses. An important component of these defenses is the intestine's normal bacterial inhabitants.

The digestive system is able to defend itself using the low pH of the stomach and the regular rhythmic movement of the muscular wall of the bowel. The bile acids are particularly toxic to bacteria, but some pathogens are relatively resistant to bile acids (such as *E. coli*); some bacteria colonize the upper small bowel where the concentrations of bile acids are low. Mucous works well to trap microorganisms in the respiratory tract, but in the bowel, certain pathogens may colonize mucous and use it as a food source. White blood cells (phagocytes) inhabit the intestinal lining and appear effective against pathogens. The normal bacteria of the human intestine interfere with pathogenic microorganisms, but this defense does not function well in humans whose digestive systems are disturbed by

antimicrobial or other drugs or starvation or emotional stress. For example, *Vibrio cholerae* can only colonize the small bowel if the normal microflora have been upset in some way (such as antimicrobial drugs), or substances have been eaten that slow bowel mobility, or in children before their normal flora have been successfully established. But, if a high concentration of pathogens has been taken into the body, an infection might occur even in people with normal intestinal flora.

In addition to these defenses, there are some specific mechanisms such as secretory antibodies (particularly IgA). There is evidence to suggest that the normal intestinal microflora prevent the destruction of the IgA antibodies by a person's own digestive enzymes and therefore help the body to defend itself. However, disease organisms such as *Neisseria gonorrhoeae* and *Streptococcus mutans* produce enzymes that specifically digest IgA antibodies.

It is possible for infection and disease to follow the ingestion of fairly small numbers of Salmonella; higher numbers may simply decrease the incubation period. For example, healthy adults and children developed *Salmonella gastroenteritis* after eating chocolate Easter eggs and rabbits contaminated with levels of *S. eastbourne* of <1-100 organisms/100g. Infection and disease (dysentery) may follow ingestion of very small numbers of Shigella; about 25% of volunteers became ill after ingesting 180 cells. Approximately 30% of the volunteers were not susceptible to Shigella at all, although their serum antibodies levels did not indicate a high level of preexisting antibody. Other defense mechanisms, as yet unknown, appear to be at work.

Similar human volunteers studies have been done with viruses, but it is difficult to determine what doses were used since the studies often did not involve counting the number of viruses per dose. For example, a study using vaccine polioviruses showed that infections resulted after ingesting 1-20 tissue culture doses; others have reported infections only after 103.5 or more doses were ingested by large numbers of subjects. It is difficult to determine the pathogenic dose because a tissue culture dose of an enterovirus may be equivalent to 10 to 1000 virus particles.

Human volunteer studies with cysts of *Entamoeba coli* showed that an individual who received a single cyst became infected; when the dosages increased, the proportion of subjects infected increased. A study with

Giardia cysts required at least doses of 10 cysts; however, some persons given high doses (106) did not develop an illness. Cyst doses were easier to count since the cyst content of a dose can be verified directly by microscopic inspection of the dose. A 2-year old child who ate dirt, which had been spread with sludge several days previously, contracted enteritis from *Salmonella typhi*.

It would appear that, although a single infectious organism may be capable of causing an infection, a single unit almost never does produce infection. Usually a substantial probability of infection is associated only with substantial numbers of infectious organisms. More work needs to be done in studying how infections begin due to ingestion of small doses and to determine the human factors that determine whether infection leads to disease.

14. Is the disease risk really so low?

Although no significant increase in human disease appears to be attributable to aerosols from wastewater treatment plants, sludge application, or spray irrigation sites, these studies are considered inconclusive by those who believe the potential exists. The issue of potential versus actual risk is complicated by several factors which prevent a definite conclusion about the health hazards.

- ◆ There are many common diseases with similar symptoms and many of these are not reported.
- ◆ Enteric disease organisms are not unique to wastewater. Wastewater aerosols are not the only transmission route or source.
- ◆ These are variations in the types and densities of microorganisms; and these reflect the diseases in the population served by the wastewater treatment plant.

- ◆ Workers may have developed immunity due to their routine exposure to low aerosol levels of disease organisms.
- ◆ Studies are difficult to conduct since the population size of the entire community is large in comparison to the small numbers who actually live near the treatment plant.
- ◆ Sensitivity of the monitoring methods for disease resistance is lacking, especially at low levels of antibodies.
- ◆ The dose-response relationship is unknown as to what minimum numbers of disease organisms are needed to initiate infection (especially via inhalation) for most pathogens.

Workers who are exposed occasionally may be more affected than those who stay in the most exposed areas continuously. This may be of importance to workers who rarely rotate to different exposure areas in the plant. For example, a worker who normally has very little exposure to wastewater aerosols, upon being moved to another area, may be more likely to develop an infection than a worker who typically works around an aeration basin or dewatering equipment.

15. Do wastewater workers develop allergies, other than reactions to endotoxins or mycotoxins?

Yes, there have been documented cases of allergic asthma caused by exposure to sewer flies (a Dipteran fly of the Psychodidae family). One worker experienced seasonal runny nose, eye irritation, and severe wheezing, but controls his symptoms by avoiding heavy sewer fly debris exposures such as floor sweepings, as well as using medication.

16. How can exposure to chemicals and diseases be reduced?

Administrative controls can be used for rotating personnel among the various treatment plant operations. This would reduce inhalation of air-stripped chemicals and aerosols, and may help development of immunity to diseases by keeping exposure low, perhaps too low for a disease-causing dose to be inhaled.

Engineering controls involve the use of ventilation for processes located within building, as well as splash guards where appropriate for dewatering equipment, and a variety of design or operational features to reduce air-stripping and aerosols of disease potential. Air sampling for chemicals and airborne levels of organisms such as Gram-negative rods can be determined to see if controls are needed. Some of the controls as reported in the technical literature are described below.

- A. Enforce pre-treatment regulations to reduce air-strippable chemicals at the source.
- B. Plant trees around the aeration basin to capture the droplets and particles.
- C. Reduce the amount of air-stripping and aerosol formation by using finer bubbles for aeration.
- D. Reduce air-stripping and aerosols by using diffused aeration rather than mechanical aeration.
- E. Some researchers have theorized that it should be possible theoretically to reduce the size of the bubble for aeration so that eventually the resulting droplets and particles would be too small to carry any microorganisms, but unfortunately this does not appear to be a very practical solution.
- F. Reduction of aeration rate, if possible. Certainly your process control strategy may not allow this.
- G. Consider floating covers on the mixed liquor of the aeration basin. Some plants have had success with:
 - ◆ Biodegradable oils
 - ◆ Collapsing foam - detergents
 - ◆ Permanent foam - Polyurethane sheets
 - ◆ Ping-pong balls - floating on the surface

H. Consider suppressing the droplets just above the surface by using these methods:

- ◆ Single layer screen - 100-200 mesh
- ◆ Multiple layer or knitted mesh screen
- ◆ Fiber beds
- ◆ Foam or granular bed
- ◆ Flat plate or slats over the tank
- ◆ Water spray to beat down the wastewater droplets
- ◆ Rotating brush

I. Consider collecting the droplets by:

- ◆ Sedimentation
- ◆ Multiple cyclone
- ◆ Scrubber
- ◆ Electrostatic precipitator
- ◆ Fabric Filtration

J. Consider disinfecting the airborne particles by using:

- ◆ Ultraviolet lights

K. Cover the primary clarifier weir area. Shield the weir area from wind. Use submerged effluent collector (such as pipes with orifices) rather than weirs.

L. Avoid handling screenings by hand to prevent needlestick injuries.

M. Label piping so that potable and nonpotable water are clearly distinguished.

N. Processes to significantly reduce pathogens are treatment processes such as aerobic and anaerobic digestion, air drying, low temperature

composting, lime stabilization or other techniques giving equivalent pathogen reduction. If sludge treated in one of these ways is applied to land: food crops which could contact sludge cannot be grown for 18 months, animals whose products are for human consumption cannot graze for 1 month, and public access is restricted for at least 12 months.

Anaerobic digestion: Greater reductions of pathogens depend upon longer detention times and higher temperatures: e.g., 24 hours at 35 C, virus kill 74.9 - 97.1%. Almost complete destruction of schistosome and hookworm ova possible, but *Ascaris ova* survival is high. Parasitic protozoans tend to be reduced to nondetectable levels. Greater reductions are accomplished in smaller-scale reactors than in full-scale plants due to elimination of short-circuiting. If sludge is dewatered and dried, *Salmonella* are reduced by 90% when the sludge is dried to 95% solids at room temperature. *Taeni* (tapeworm) ova survive about 14 days in the absence of surface moisture.

Lime stabilization: elevating the pH to 11.5 to 12.0 for 30 minutes can reduce pathogenic bacteria and poliovirus. Lime stabilized sludge at pH = 12 were reduced by a factor of 10 to 1000 times less than anaerobically digested sludge. After the pH is elevated, it later drops and this subsequent drop in pH can result in regrowth. For example, fecal streptococcus bacteria was able to regrow to its original density in 24 hours.

Q. Process to further reduce pathogens: These are sludge treatment methods such as gamma or beta irradiation to an absorbed dose of 1 Mrad, pasteurization or other methods of equivalent pathogen reduction. PSRP is required first to reduce volatile solids. If PFRP is used, the restrictions applied to PSRP are no longer required.

Irradiation: Ionizing radiation kills salmonella and coliforms. *Aspergillus niger* spores and polio virus; the larger microorganisms are the more susceptible.

Worker exposure can also be reduced by the use and proper care of protective clothing and equipment.

- Heavy duty rubber gloves and boots can be used to prevent skin contact with wastewater and sludges. Especially cover any skin trauma such as cuts and abrasions to prevent infection. Use protective clothing and goggles to prevent contact with spray and splashes.
- Remove contaminated clothing after completion of a job. Avoid laundering work clothes at home. If they are cleaned at home, place them in a bag and leave them bagged until they are actually to be placed in the washing machine. Wash them separately from other clothing with the hot water cycle. Consider using chlorine bleach if appropriate for the fabric; if not, use a nonchlorine.
- Shower at work and change into clean clothes and shoes.
- Wash hands with soap and water before eating or smoking and whenever hands come in contact with wastewater and sludge. Care for cuts and abrasions promptly.

17. Should a wastewater worker be immunized to reduce the risk of infection?

The following are recommended by the U.S. Public Health Service and the Centers for Disease Control. Currently no additional immunizations have been recommended for wastewater workers.

The Centers for Disease Control has so far concluded that there have been no work-related cases of hepatitis. A transmission among workers exposed to sewage; principally because the data from the serologic (antibody) studies on workers exposed to sewage were not controlled for other risk factors. If prophylaxis is desired for hepatitis A, there are two approaches available: immune globulin and vaccination. Two vaccines are currently licensed in the United States for hepatitis A and both consist of inactivated hepatitis A virus. HAVRIX (manufactured by SmithKline Beecham Biologicals) and VAQTA (manufactured by Merck & Company, Inc.). For adults (over the age of 18 years), HAVRIX is administered in 2 doses with the second dose

given 6-12 months after the first. For adults (over the age of 17 years), VAQTA is administered in 2 doses with the second dose given 6 months after the first.

18. Are there adverse health effects from chronic exposure to low levels of hydrogen sulfide gas?

There appear to be 2 types of effects depending upon the level of exposure:

- A. central nervous system effects, and
- B. local irritation

Systemic effects are the result of action on the nervous system; for hydrogen sulfide this generally means respiratory failure followed by asphyxiation. The respiratory center is stimulated causing rapid breathing which rids the lungs of excessive carbon dioxide and breathing stops. Mortality from hydrogen sulfide exposure can be reduced by increased attention to cardiopulmonary resuscitation at the exposure site and during transportation to the hospital. These effects may be accompanied by short-term electrocardiographic changes, elevation of blood nonprotein nitrogen levels, and the appearance in the urine of red blood cells and hyaline casts. Cardiac effects have included both hypertension and hypotension, tachycardia (abnormally rapid heartbeat of over 100 beats per minute) and bradycardia (very slow heartbeat of under 60 beats per minute).

Several mechanisms have been proposed as to why this happens: the most popular theory says that the enzyme cytochrome oxidase, which is involved in the body's use of oxygen, is poisoned in a similar manner to cyanide poisoning. A different theory suggests that hydrogen sulfide may cause an initial increase in nervous tissue activity which brings about neuropsychological and other effects such as a slow heartbeat. It has also been suggested that autooxidation of hydrogen sulfide in the tissues results in the formation of hydrogen peroxide which is actually responsible for some of the toxic effects of sulfide on the central nervous system.

DISEASE	WHO NEEDS IMMUNIZATION	IMMUNIZATION
Hepatitis A	Individuals with close personal contact with persons with hepatitis A; travelers; persons with chronic liver disease; children living in communities with high rates of hepatitis A; children and young adults in communities that have intermediate rates of hepatitis A.	immune globulin; hepatitis A vaccine
Hepatitis B	Homosexual males and household and sexual contacts with carriers. For direct exposure to blood of a person known or suspected to be a carrier. For occupational transmission, as per USDOL.OSHA CFR 1910.1030.	Immune globulin; hepatitis B vaccine
Influenza	Adults 65 years or older	Annual influenza vaccine
Measles	Adults born in 1957 or later, unless they have evidence of vaccination on or after their first birthday, documentation of physician diagnosed disease, or laboratory evidence of disease	Combined measles, mumps, rubella (MMR)
Pneumococcal disease	Adults 65 years or older	Pneumococcal polysaccharide vaccine
Mumps	Adults, especially males, who have not been previously infected	Mumps vaccine
Rubella	Women of childbearing age, unless proof of vaccination or lab evidence of immunity is available	Rubella vaccine
TD (Tetanus and Diphtheria Routine	Adults every 10 years after initial doses, after wounds unless less than 5 years since last dose	TD vaccine

When taken into the body, some of the hydrogen sulfide dissociates to form the hydrosulfide anion (HS^-). Methemoglobin (an early stage of hemoglobin manufacture) competes with cytochrome oxidase to react with and bind the toxic hydrosulfide anion. When methemoglobin binds to hydrosulfide to form sulfmethemoglobin, this causes a condition called methemoglobinemia which actually helps provide protection against death from acute sulfide poisoning. This enables the reactivation and protection of cytochrome oxidase and aids the patient's recovery by enhancing aerobic metabolism. The treatment of hydrogen sulfide poisoning has involved giving a patient injections of sodium nitrate to purposely cause methemoglobinemia: this was used successfully to resuscitate one human severely poisoned by hydrogen sulfide.

The local irritant effects of hydrogen sulfide involve a direct action on the tissues and local inflammation of the moist membranes of eyes and respiratory tract because of hydrogen sulfide's high solubility in water. This ability to dissolve rapidly in the mucous of the nasal passages is what leads to an overloading of the olfactory nerve endings causing their paralysis and thus the loss of the ability to detect this gas at higher concentrations. The hydrogen sulfide gas dissolves in the moisture and dissociates to form an acid which then irritates the conjunctival and respiratory mucosa. There are infrequent incidences of this irritation being accompanied by pulmonary edema (fluid in the lungs). When inhaled, the irritant action is more or less uniform throughout the respiratory tract, although the deeper pulmonary structures suffer the greatest damage. Studies in animals suggest that the macrophages in the alveoli of the lungs are impaired in their ability to combat bacteria by the action of hydrogen sulfide; this may cause increased susceptibility to infections such as pneumonia.

The eye irritation and inflammation of the conjunctival and corneal tissues is called "gas eye." It is often accompanied by pain, watery eyes, and sensitivity to light ("photophobia"). In severe form, it may progress to acute keratoconjunctivitis with associated blistering of the corneal epithelium. Rupture of these blisters may, in some cases be followed by corneal ulceration which could heal with scar formation and permanent impairment of vision.

The dry surfaces of the skin are seldom affected by gaseous hydrogen sulfide. The most important route of absorption is through the lungs; proper respiratory protection covering only the face and head will permit work to be carried on in atmospheres containing concentrations which would be immediately fatal to the unprotected worker. It does not appear that skin absorption of the gas is a major route of entry; industrial experience suggests that cutaneous absorption must be many times less efficient than pulmonary absorption. Hydrogen sulfide has been noticed to retard the healing of minor skin wounds.

Overall, if the victim survives, there appear to be no long-term adverse effects from acute exposure (aside from the possible eye damage mentioned above). It appears that if the exposure is not immediately lethal and adequate support is provided, there is a reasonable expectation for complete recovery. The site of exposure is an important factor; most exposures necessitating hospitalization occur in enclosed or confined spaces; exposures in the open air to hydrogen sulfide has produced problems of lesser magnitude. Substantial decreases in worker mortality have been attributed to improved first-aid training, increased awareness of the dangers of hydrogen sulfide, conducting continuous personal and environmental monitoring, and the effective use of personal protective equipment.

Hydrogen sulfide has not so far been linked with carcinogenesis, mutagenesis, or teratogenesis. One study concluded that there was a “weak teratogenic effect” in rats following low-level exposures to a combination of hydrogen sulfide and carbon disulfide, but no strong evidence supporting teratogenic effects from hydrogen sulfide exposure only.

What happens with chronic exposure to hydrogen sulfide gas? Hydrogen sulfide is principally eliminated by the body through the kidneys in the form of sulfate or sulfide. There is some elimination of the gas through the lungs although there appear to be no reliable estimates on the quantitative importance of this method of excretion. Hydrogen sulfide generated by intestinal microflora in flatulence is, at least in part, systematically absorbed and detoxified.

The characteristic signs and symptoms of systematic hydrogen sulfide poisoning occur only when free, unoxidized gas is present in the circulating blood stream. To cause systematic intoxication, the gas must be absorbed at a rate faster than it can be eliminated or detoxified. Because detoxification proceeds at a very rapid rate, H₂S can be considered as a noncumulative poison; this is why it tends not to be considered as having chronic

The effects of hydrogen sulfide gas on the body may be divided into three categories: acute, subacute, and chronic. "Acute" has been used to describe episodes of systematic poisoning that have occurred rapidly and in which the central nervous system effects (such as respiratory paralysis) have predominated. "Subacute" has been used to describe cases in which the local irritant effects have predominated. As for "chronic" there is no unanimity of opinion that this condition actually exists; it has been suggested that it is probably a series of low-grade "acute" episodes. As with any toxic substance, the effects of exposure depend so much upon both the duration and the intensity of exposure. Because the body has an inherently large capacity for detoxifying sulfide, the toxicity of the gas is more closely related to concentration than to length of exposure. It should be understood that in any case of exposure to this gas, both local and systematic injury may result.

Can adverse health effects be caused by low levels of hydrogen sulfide gas (e.g., under 5 parts per million)? There is some evidence that hydrogen sulfide alone at low concentrations or in combination with other chemical substances (e.g., hydrocarbons or carbon disulfide) has caused nervous system, cardiovascular, and gastrointestinal disorders such as diarrhea, headache, fatigue, irritability, insomnia, and effects of the eyes. The American Congress of Governmental Industrial Hygienists (ACGIH) lowered the threshold limit value (TLV) of hydrogen sulfide from 20 parts per million (ppm) to 10 ppm with short-term exposure limits at 15 ppm because, at low concentrations, eye effects are predominant with conjunctivitis the most common effect, while keratitis frequently occurs. Some eye effects have been reported as low as 4 or 5 ppm. Low concentrations have been observed to interfere with the healing of small wounds.

Are there adverse health effects from chronic exposure to low levels of hydrogen sulfide gas (under 5ppm)? Conclusive evidence of adverse health effects from reported, long-term exposure to hydrogen sulfide at low concentrations does not appear to have been found. Although persistent effects on humans after long-term exposure have not been conclusively demonstrated, numerous studies suggest that there may be a series of low-grade subacute effects.

Cases of eye irritation similar to those more recently attributed to chronic exposure were reported in the 18th century by the Italian physician Ramazzini, considered the father of occupational medicine. His treatise on occupational health reports on the physical condition of sewer cleaners who experienced eye irritations.

One historical case of what may have been chronic hydrogen sulfide poisoning has been reported in a workman who was exposed to hydrogen sulfide for 2 years in the early 1900's at a sulfur-black establishment. In 1905 he experienced lack of muscular coordination, pains, paresthesias (sensations of numbness, prickling, or tingling), muscular atrophy, and a narrowing of the visual field. By 1906, he was totally blind with pain and persistent paresthesia; he died of bronchopneumonia in 1910.

Microscopic examination of sections of the spinal cord revealed no inflammation, but extensive degeneration.

One study in rats showed abnormal changes in the cerebral cortex of the brain from chronic exposure to 7 ppm (10 milligrams per cubic meter) for 12 hours a day for 3 months.

Chronic exposure to low concentrations may result in conjunctivitis (gas eye) or occasionally pulmonary edema. Habituation to exposure does not appear to exist; on the contrary, hypersusceptibility may result.

19. *What medical surveillance is recommended for the wastewater worker?*

Table 1 Waste Water Treatment Plants: Medical Surveillance

<i>Process</i>	<i>Hazard</i>	<i>Health effect</i>	<i>Screening Devices</i>	<i>Special Precautions</i>
<i>Primary</i>	<i>Domestic waste</i>		<i>Medical History</i>	
<i>Secondary</i>	<i>Feces with:</i>	<i>GI infections</i>	<i>Medical History</i>	
	<i>Viruses</i>	<i>Hepatitis</i>	<i>Hepatitis antigen</i>	
	<i>Bacteria</i>		<i>Baseline liver functions</i>	
	<i>Fungus</i>			
	<i>Worms</i>			
	<i>Protozoa</i>			
	<i>Industrial waste/ heavy metals</i>	<i>Kidney disease</i>	<i>Medical History</i>	
			<i>Baseline renal function (U/A, creatinine, BUN)</i>	
<i>Chlorination</i>	<i>Chlorine gas</i>	<i>Anemia</i>	<i>CBC</i>	
		<i>Pulmonary and mucous membrane irritation</i>	<i>Medical History</i>	<i>Outline emergency procedures</i>
				<i>Self-contained breathing Apparatus (SCBA)</i>
<i>Sludge treatment</i>	<i>Methane</i>	<i>Asphyxiation</i>	<i>Medical History</i>	<i>Emergency procedures</i>
	<i>Hydrogen sulfide</i>	<i>Respiratory arrest</i>		<i>SCBA</i>
	<i>Pathogens present in domestic waste</i>	<i>GI infection</i>		<i>Non-sparking tools</i>
	<i>Oxides of metal</i>			

TABLE II. Recommended Pre-assignment and Periodic Health Assessments

<i>Pre-placement examination</i>
<i>Comprehensive physical examination</i>
<i>Liver and kidney function (urinalysis/multiphasic)</i>
<i>Hematologic function (CBC)</i>
<i>Yearly Periodic Health Assessment</i>
<i>Medical history</i>
<i>Includes a review of systems for symptoms suggestive of water-borne diseases (e.g. hepatitis intestinal infections)</i>
<i>Attempts to elicit history of reactions to exposure to toxic gases (e.g. chlorine, lime)</i>
<i>Immunization update : influenza, tetanus, diphtheria, polio (as indicated)</i>
<i>On-going health care</i>
<i>Prompt reporting of illnesses lasting greater than 2 days to safety director to monitoring</i>
<i>Evaluation of suspected work-related illnesses as needed</i>

REFERENCES

- Babish, J.G. et al. 1983. "Mutagenicity of municipal sewage sludges of American cities." ES&T: 272.
- Baker, E.L. et al. 1980. "Metabolic consequences of exposure to polychlorinated biphenyls (PCB) in sewage sludge." Am. J. Epidemiol.: 112.
- Bergh, A.K. et al. 1977. "Distribution of PCB's in a municipal wastewater treatment plant and environments." Sci. Total Environ. 8: 197.
- Bishop, K. et al. 1985. "Identification of asbestos and glass fibers in municipal sewage sludges." Bulletin Environment Contam. Toxicol. 34: 301.
- Blanchard, D. et al. 1970. "Mechanism for water-to-air transfer and concentration of bacteria." Science 170: 626.
- Brauning, S. et al. 1986. "Radioactivity levels in municipal sludge." Biocycle 27(5): 48.
- Burrell, R. et al. 1990. "Toxic risks from inhalation of bacterial endotoxin." Br. J. Ind. Med. 47: 688.
- Callahan, M. et al. 1979. "Water-related environmental fate of 129 priority pollutants." EPA 440/4-79-029a,b. Versar, Inc. Springfield, VA. Dec. 1979.
- Camann, D.E. et al. 1988. Microorganism levels in air near spray irrigation of municipal wastewater: the Lubbock infection surveillance study. JWPCF 60 (11): 1960.
- Cannon, R.E. 1983. "Aerosol release of cyanophages and coliforms from activated sludge basins." JWPCF 55(8): 1070.
- Celli, T.R. et al. 1981. "Respiratory risks in sewer workers. A cross sectional survey." (Abstract) Am. Review of Respiratory Diseases 123: 129.
- Centers for Disease Control. 1979. "PCB exposure - Indiana." Morb. & Mort. Weekly Report 27: 99.
- Christman, R. et al. 1981. "Chlorination of aquatic humic substances." EPA-600/S2-81-016. SUDOC EP1.89/2:C45/2.
- Clark, C.S. et al. 1989. Correspondence to AFSCME, USEPA, and NIOSH. "An update of hepatitis B study of WW workers in Cincinnati, Chicago, and Memphis."
1976. "Disease risks of occupational exposure to sewage." J. Env. Eng. Div., Am. Soc. Civil Eng. 102: 375.
1986. "Use of serum antibody as a means to determine infections from exposure to waste waters and refuse." CRC Critical Reviews in Environmental Control, Vol. 16(4): 305.
1985. "Serologic survey of Rotavirus, Norwalk Agent, and Prototheca wickerhamii in wastewater workers." Amer. J. Public Health 75(1): 83.
1984. "Enteric parasites in workers occupationally exposed to sewage." J. Occup. Med. 26(4): 273.
1981. "Health risks of human exposure to wastewater." EPA-600/S1-81-001. SUDOC EP1.89/2:H34.
1980. "Health effects of occupational exposure to wastewater." in: Wastewater aerosols and disease. EPA 600/9-80-028.
- Clevenger, T. et al. 1983. "Chemical composition and possible mutagenicity of municipal sludges." JWPCF 55(12): 1470.
- Clover, D.O. 1980. "Infection with minimal quantities of pathogens from wastewater aerosols." in: EPA-600/9-80-028. Wastewater aerosols and disease. U.S. Environmental Protection Agency. EP 1.23/6:600/9-80-028.
- Dean, R.B. et al. 1980. "Disease rates among Copenhagen sewer workers." in Wastewater aerosols and disease. EPA 600/9-80-028.
- Dixon, F.R. et al. 1963. "Health aspects of wastewater treatment." JWPCF 36(8): 984.

REFERENCES

- Doby, J.M. et al. 1980. "Amoebiasis, an occupational disease of sewer workers?" Nouv. Presse Med. 9: 532.
- Dunovant, V. et al. 1986. "Volatile organics in the wastewater and air spaces of three wastewater treatment plants." JWPCF 58(9): 886.
- Elia, V.J. et al. 1983. "Hazardous chemical exposure at a municipal wastewater treatment plant." Environ. Res. 32: 360.
1980. "Worker exposure to organic chemicals at an activated sludge wastewater treatment plant." in: Wastewater aerosols and disease. EPA 600/9-80-028.
- "Encountering hazardous gases in wastewater treatment facilities." 1981. WPCF Highlights 18(10): 12.
- Forsman, J.P. 1987. "Designing an effective safety program." Oper. Forum 4(2): 18.
- Furr, A. et al. 1976. Multielement and chlorinated hydrocarbon analysis of municipal sewage sludges of American cities. ES&T 10: 683.
- Gartside, P.S. et al. "Interim report on a mortality study of former employees of the Metropolitan Sanitary District of Greater Chicago." in: Wastewater aerosols and disease. EPA 600/9-80-028.
- Gaudy, A.F. et al. 1963. "Biological treatment of volatile waste components." JWPCF 35(1): 75.
- Glaser, J.R. et al. 1967. "Sizes and numbers of aerosols generated by activated sludge aeration." Water and Sewage Workers 114: 219.
- Goff, G.D. et al. 1973. "Emission of microbial aerosols from sewage treatment plants that use trickling filters." Health Services Reports 88(7): 640.
- Gold, B.J. et al. 1985. "Occupational asthma caused by severe flus." Am. Rev. Resp. Dis. 131: 949.
- Goldsmith, J. et al. 1985. in: Two comments on fetal loss and ww workers) I. Maternal age a factor. Letter to the Editor. Am. J. Public Health 75: 98.
- Golueke, C.G. 1983. "Epidemiological aspects of sludge handling and management, I. Biocycle 24(3): 52; II. Biocycle 24(4): 50.
- Gupta, P. 1991. "Personal communication (09/06/91) on HIV inactivation by wastewater."
- Hadeed, S.J. 1988. "AIDS risk potential at municipal wastewater treatment and collection facilities." Operations Forum (AUG.): 4.
1986. "Inexperienced workers injury prone." Operations Forum 3: 8.
- Hertzberg, V. et al. 1991. "Statistical issues in risk assessment of reproductive outcomes with chemical mixture." Env. Health Persp. 90: 171.
- Hopke, P.K. et al. 1982. "Multitechnique screening of Chicago municipal sewage sludge for mutagenic activity." ES&T 16(3), 140.
- Johnson, D. et al. 1980. "Health effects from wastewater aerosols at a new activated sludge plant: John Egan Plant, Schaumburg, Illinois." in: Wastewater aerosols and disease. EPA 600/9-80-028.
- Jordan, D. 1977. "The town dilemma -- discharging a duty." Environment 19: 6.
- Kominsky, Jr. 1980. "Nonviable contaminants from wastewater: Hexachlorocyclopentadiene contamination of a municipal wastewater treatment plant." in: Wastewater aerosols and disease. EPA 600/9-80-028.
1980. "Hexachlorocyclopentadiene contamination of a municipal wastewater treatment plant." Am. Industrial Hygiene Association Journal 41(8): 552.
- Kraut, A. et al. 1988. "Neurotoxic effects of solvent exposure on sewage treatment workers." Archives of Env. Health 43(4): 263.
- LaFleur, J. et al. 1991. "Retrospective cohort mortality study of cancer among sewage plant workers." Amer. J. of Ind. Medicine 19: 75.
- Lee, K.M. et al. "Destruction of enteric bacteria viruses during two-phase digestion." JWPCF 61(8): 1421.

REFERENCES

- Lucas, J.B. 1980. "Health effects of nonmicrobiologic contaminants." in: Wastewater aerosols and disease. EPA 600/9-80-028.
- Lue-Hing, C. et al. 1980. "Suppression of aerosols at a wastewater treatment plant." in: Wastewater aerosols and disease. EPA-600/9-80-028.
- Lundholm, M. et al. 1983. "Work related symptoms amongst sewage workers." Brit. J. Ind. Med. 40: 325.
- Lurker, P.A. et al. 1984. "Aerial organic chemical release from activated sludge." Water Resources. (G.B.) 18(4): 489.
1982. "Atmospheric release of chlorinated organic compounds from the activated sludge process." JWPCF 54(12): 1566.
- Majeti, V.A. et al. 1981. "Potential health effects from viable emissions and toxins associated with wastewater treatment plants and land application sites." EPA-600/S1-81-006. SUDOC EP1.89/2:H34/2.
- Mattsby, I. et al. 1978. "Clinical and immunological findings in workers exposed to sewage dust." JOM 20: 690.
- Matter-Muller, C. 1981. "Transfer of volatile substances from water to atmosphere." Water Res.(G.B.) 15, 1271.
- McCunney, R.J. 1986. "Health effects of work at wastewater treatment plants:" A review of the literature with guidelines for medical surveillance. Amer. J. of Ind. Medicine 9: 271.
- Nethercott, J. et al. 1988. "Health status of a group sewage treatment workers in Toronto, Canada." AIHAJ 49(7): 346.
1981. "Airborne irritant contact dermatitis due to sewage sludge." JOM 23(11): 771.
- Northrup, R. et al. 1981. "Health effects of sewage aerosols: Additional serological surveys and search for Legionella pneumophila in sewage." EPA-600/S1-81-032. SUDOC EP1.89/2:A1 8/7. EPA-600/9-82-003. Estimating microorganism densities in aerosols from spray irritation of wastewater.
1980. "Health effects of aerosols emitted from an activated sludge plant." in: Wastewater aerosols and disease. EPA 600/9-80-028.
- Norwood, D. et al. 1980. "Reactions of chlorine with selected aromatic models of aquatic humic material." ES&T 14(2): 187.
- "OSHA hears about AIDS/hepatitis risk from New York workers, 1990." UPCF Safety and Health Bulletin (summer): 9.
- Petrasek, A. et al. 1983. "Metals removal and partitioning in conventional wastewater treatment plants." JWPCF 55(9): 1183.
1983. "Fate of toxic organic compounds in wastewater treatment plants." WPCF 55(10): 1286.
- Pitt, R. 1988. "Asbestos as an urban area pollutant." JWPCF 60(11): 1993.
- Princine, A.B. 1991. "Transfer of oxygen and emissions of volatile organic compounds at clarifier weirs." JWPCF 63(2): 114.
- Rappaport, S. et al. 1979. "Mutagenic activity in organic wastewater concentrates." ES&T 13(8): 957.
- Roberts, P.V. et al. 1984. "Modeling volatile organic solute removal by surface and bubble aeration." JWPCF 56(2): 223.
- Rubin, A.J. et al. 1983. "Disinfection of amoebic cysts in water with free chlorine." JWPCF 55(9): 1174
- Savage, D.C. 1980. "Colonization by and survival of pathogenic bacteria on intestinal mucosal surfaces." in: Bitton, G. et al. (Eds.). Adsorption of microorganisms to surfaces. John Wiley and Sons. NY.

REFERENCES

- Scarlett-Kranz, J.M. 1986. "Urinary mutagens in municipal sewage workers and water treatment workers." Amer. J. of Epidem. 124(6): 884.
- Scheff, P.A. et al. 1981. "Characterization of air pollutants from an activated sludge process." JWPCF 53(2): 223.
- "Sewer collapse and toxic illness in sewer repairmen - Ohio." Morbidity and Mortality Wkly Rprt 30(8):89. (03/06/81).
- Skinhoj, P. et al. 1980. "Infectious liver diseases in 3 groups of Copenhagen Workers: Correlation of Hepatitis A infection sewage exposure." Arch of Environmental Health 36(3): 139.
- Soldano, B.A. et al. 1975. "Airborne organo-mercury and elemental mercury emissions with emphasis on central sewage facilities. Atmos. Environ. 9: 941.
- Sorber, C.A. et al. 1980. "Indicators and pathogens in wastewater aerosols and factors affecting survivability" in: EPA-600/9-80-028. Wastewater aerosols and disease. U.S. Environmental Protection Agency. EP 1.23/6:600/9-80-028.
- Spendlove, J. et al 1980. "Effectiveness of aerosol suppression by vegetative barriers." in: Wastewater aerosols and disease. EPA 600/9-80-028.
- "Study finds no apparent threat to plant workers from HIV waste water." 1991. BNA Occupational Safety Report (02/06/91): 1363.
- Viraraghavan, J. 1973. "Occupationally related health hazards in waste water treatment systems. WPCF Deeds and Data (Nov.): 2
- WPCF. 1983. "Safety and Health in WW systems. M.O.P. #1. WPCF 2626 Penn. Ave., NW., Washington DC 20037.
1990. "Wastewater biology: the microlife." Water Pollution Control Federation. Alexandria, VA.
- White, D.O. et al. 1986. Medical virology. 3rd ed. Academic Press, Inc. NY.
- Woodcock, A.H. 1985. Bursting bubbles and air pollution. Sew. Ind. Waste 27: 1189.
- Yeager, J.G. et al. 1983. "Irradiation as a means to minimize public health risks from sludge-borne pathogens." JWPCF 55(7): 977.