United States
Environmental Protection
Agency

Office of Research and
Development
Washington, DC 20460

EPA/600/R-92/064
June 1992

EPA

Preliminary Risk Assessment for Viruses in Municipal Sewage Sludge Applied to Land

EPA/600/R-92/064
June 1992

Preliminary Risk Assessment for Viruses in Municipal Sewage Sludge Applied to Land

Environmental Criteria and Assessment Office
Office of Health and Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

DISCLAIMER

This document has been reviewed in accordance with U.S. Environmental Protection Agency policy and approved for publication. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

PREFACE

Section 405 of the Clean Water Act requires the U.S. Environmental Protection Agency to develop and issue regulations that identify: (1) uses for sludge including disposal; (2) specific factors (including costs) to be taken into account in determining the measures and practices applicable for each use or disposal; and (3) concentrations of pollutants that interfere with each use or disposal. To comply with this mandate, the U.S. EPA has embarked on a program to develop four major technical regulations: land application, including distribution and marketing; landfilling; incineration and surface disposal. The development of these technical regulations requires a consideration of pathogens as well as chemical constituents of sludge. Public concern related to the reuse and disposal of municipal sludge often focuses on the issue of pathogenic organisms.

This report is one of a series whose purpose is to use the methodology described in *Pathogen Risk Assessment for Land Application of Municipal Sludge* to develop preliminary assessments of risk to human health posed by parasites, bacteria and viruses in municipal sewage sludge applied to land as fertilizer or soil conditioner. The preliminary risk assessment includes a description of the most critical data gaps that must be filled before development of a definitive risk assessment can be accomplished and recommends research priorities.

DOCUMENT DEVELOPMENT

Cynthia Sonich-Mullin, Project Officer
Norman E. Kowal, Technical Project Manager
Environmental Criteria and Assessment Office
U.S. Environmental Protection Agency
Cincinnati, OH

Authors

Marialice Wilson, Project Manager
Charles T. Hadden
Mary C. Gibson
Science Applications International Corporation
Oak Ridge, TN

Reviewers

Joseph B. Farrell
Risk Reduction Engineering Laboratory
Andrew W. Breidenbach Environmental Research Center
U.S. Environmental Protection Agency
Cincinnati, OH

Mary-Ellen Morris
Virology Branch, Microbiol. Res. Div.
Environmental Monitoring Systems Laboratory
U.S. Environmental Protection Agency
Cincinnati, OH

Babasaheb Sonawane
Reproductive and Developmental Toxicology Branch
Human Health Assessment Group
U.S. Environmental Protection Agency
Washington, D.C.

Richard Walentowicz
Exposure Assessment Group
U.S. Environmental Protection Agency
Washington, D.C.

Judith Olsen, Editorial Review
Environmental Criteria and Assessment Office
U.S. Environmental Protection Agency
Cincinnati, OH

TABLE OF CONTENTS

TABLE OF CONTENTS (continued)

TABLE OF CONTENTS (continued)

LIST OF TABLES

LIST OF FIGURES

ABBREVIATIONS AND SYMBOLS

AGI	Acute gastrointestinal illness
CEC	Cation exchange capacity
CFU	Colony-forming units
D&M	Distribution and marketing
dia	Diameter
ffu	Focus-forming units
g	Gram
ha	Hectare
HAV	Hepatitis A Virus
HCV	Human coronavirus
HID	Human infective dose
hr	Hour
HVCS	High volume cyclone scrubber
ID	Infective dose
MID	Minimum infective dose
min	Minute
MPN	Most probable number
NOAA	National Oceanic and Atmospheric Administration
PFRP	Processes to Further Reduce Pathogens
PFU	Plaque-forming Units
PSRP	Processes to Significantly Reduce Pathogens
RH	Relative humidity
sec	Second
SRV	Small round viruses
TCID	Tissue Culture Infective Dose
TDS	Total dissolved solids
TPB	Tryptose phosphate broth
USDA	U.S. Department of Agriculture
wt	Weight

1. EXECUTIVE SUMMARY

This preliminary risk assessment study focuses on the probability of human infection from enteric viral pathogens in municipal sludge applied to land. It is based on the Pathogen Risk Assessment computer model and methodology described in *Pathogen Risk Assessment for Land Application of Municipal Sludge* (U.S. EPA, 1989a).

This document reports (1) the results of a literature review designed to find the data on pathogenic viruses required by the pathogens methodology, and (2) the results of numerous site-specific computer simulations, running the Pathogen Risk Assessment Model with a wide range of values for the parameters required. The parameters required for viruses are (1) density of infective viruses in treated sludge destined for land application; (2) minimum infective dose; (3) inactivation rates in soil, dry particulates, liquid aerosols and water; and (4) dispersion in the environment, i.e., transport in water, soil and air.

Literature values for virus density in treated sludge were so variable, both by treatment methodology and by virus type, that no single number could be selected as typical. However, 2000 virus particles/kg was chosen as representative of viral density in composted sludge and 100,000 particles/kg in digested sludge. Infective doses, while varying by detection method and by virus type, have been reported to be as low as 1 infective particle. As a conservative assumption, this minimum value was used for the model runs. Reported inactivation rates range from 7.1×10^{-5} to 1.6×10^{-1} logs/hour in soil, 1.6×10^{-4} to 1.4×10^{-1} logs/hour in water, and 4.9×10^{-5} to 8×10^{-7} logs/second in aerosols. Like the density values, these rates are quite variable. Information on dispersion of viruses in the environment is limited in its applicability to generating a rate of transport in environmental media. Development of a variety of transport models has been an attempt to quantify the movement of viruses, especially in the subsurface and in groundwater.

Six sites were chosen to provide diversity in geographic location, topography, soil type, rainfall pattern and temperature. Locations selected for site-specific application of the model include Anderson County, TN; Chaves County, NM; Clinton County, IA; Highlands County, FL; Kern County, CA; and Yakima County, WA.

An initial sensitivity analysis was performed using site-specific parameters for Site 1, Anderson County, TN. Main program variables used in the model run were varied over a range of values to determine the sensitivity of the model to variations in conditions. In general, the default value of a given parameter was compared with a reasonable higher and a reasonable lower value, where the high and low values were taken from available literature or estimated when literature values were not available.

In this analysis, it is assumed that viruses are transported into subsurface soil and subsequently into groundwater and are included in any droplet aerosols formed by spray application, as well as in any particulate aerosols formed by disturbance of the soil by wind or by cultivation. It is also assumed that the viruses are inactivated at a characteristic rate that depends on the ambient temperature and the medium in which they are found.

Using baseline parameters at Site 1, the maximum probabilities of infection in each practice were evaluated. Infection ONSITE was similar for all practices, between 1% and 7%. The probability of infection to the OFFSITE receptor was calculated as zero in every case. Risk of infection via contaminated food products (EATER) was shown only in Practice IV, and risk via offsite wellwater (DRINKER) was shown in Practice III. In contrast, infection by contact with onsite surface water (SWIMMER) was significant in all three practices (Practices I-III) that include the pond, the risk level being dependent on site-specific as well as practice-specific variables.

The effects of site-specific and practice-specific differences in parameters and assumptions are illustrated by comparing the outcome of baseline model runs. The second set of model runs, in which inactivation rates were decreased in soil, water, and droplet aerosols, showed higher probabilities of infection at all sites and for most exposure compartments. The results of these model runs, using baseline parameters except for the more conservative inactivation rates, showed the maximum calculated probabilities of infection ONSITE were similar for each site, and again no OFFSITE infection was predicted. Infection via contaminated food products was calculated to be significant only in Practices I and IV, whereas infection via contaminated wellwater was indicated in Practices I-III at all sites. Infection to the SWIMMER was predicted at significantly higher levels than with the default inactivation parameters.

Results show that the inactivation rate of virus particles is extremely important in determining whether a groundwater well is likely to become contaminated and in determining how long surface soils or surface water are likely to remain infectious. The results also demonstrate the importance of accurate characterization of inactivation rate for viruses of different kinds in the various transport and exposure media.

Using reference values including the conservative inactivation rates, the baseline maximum probability of infection was 0.270 for Practice I, 0.046 for Practices II and III, 0.0055 for Practice IV, and 0.0028 for Practice V. Generally, site-specific variables did not have a significant effect on the probability of ONSITE infection, because the site-specific variables alter temperature-dependent inactivation rates and rainfall-dependent runoff and sediment transport, none of which exerts major effects on the ONSITE exposure compartment before the time of maximum infection. Significant impacts on the probability of infection were observed in all application practices with changes in pathogen density in the applied sludge [ASCRS, P(1)] and sludge application rate [APRATE, P(2)], both of which determine the number of viral particles applied. Because of the exponential nature of the probability algorithm, the changes in probability were not directly proportional to the change in parameter values, but varied as would be expected for a proportional change in exposure.

In all model runs the probability of infection OFFSITE was calculated as zero, indicating that although the inactivation of viruses in aerosols may be less than initially expected, the calculated quantities of liquid and dry particulate aerosols and concentrations of viruses in the aerosols were too low to provide an infective dose to the modeled receptor.

Consumption of contaminated vegetable crops was shown by model calculations to be a potential source of human infection, provided that inactivation rates were sufficiently low or harvesting times were sufficiently close to application of the sludge. Infection via food crops was sensitive not only to infectious dose, inactivation rates, and the parameters that directly affect the number of pathogens applied to the soil, but also to the relative fractions of pathogens transferred among surface soil, subsurface soil, and crop surface and to the type of crop or fraction of the total crop grown aboveground, below-ground, or on the ground.

Contamination of meat or milk by viruses from sewage sludge did not appear to pose a significant risk to human health.

Transport of viruses via groundwater to an offsite well was not shown by this model to be a major risk, but exposure by contaminated groundwater was shown to be likely if the rate of inactivation of viruses in water was less than the default values. The probability of infection was related to the periodic introduction of pathogens to groundwater by the infiltration of rainwater. The most important parameter related to subsurface transport of viruses appeared to be the inactivation rate of viruses in water. The results also showed an increase in probability of infection at the offsite well whenever the time required for the viruses to reach the well was decreased.

Contaminated surface water, represented by the SWIMMER in an onsite pond, was the most significant source of exposure. A peak in probability of infection occurred after each rainfall, when additional contaminated surface water and soil were washed into the pond.

The following information is needed to improve the usefulness of the Pathogen Risk Assessment Model and to allow for a more reliable risk assessment of the land application of sewage sludge:

- Simple and accurate standardized methods for detecting and quantifying, by type, pathogenic viruses in treated sludge destined for land application, in final D&M sludge products, and in environmental media;
- Improved understanding of minimum infective doses, particularly low-dose effects and MIDs for sensitive subjects;
- More accurate persistence and transport data on all pathogenic viruses of major concern in sludge;
- Development of an index of soil types that would correlate capacity for solute transport and suitability for sludge application (also valuable for onsite waste disposal or solid waste disposal);
- Research on subsurface injection of sludge and the relative probability of virus transport in groundwater; and
- Epidemiologic studies evaluating enteric viral transmission.

The following revisions would improve the accuracy of the model:

- Revision of default parameter values, especially for inactivation rates in aerosols and temperature-dependent inactivation rates in soil and water;
- Revision of temperature-dependent inactivation algorithms;
- Incorporation of factors for humidity and temperature in inactivation equations for aerosols;
- Incorporation of subroutines for subsurface transport under conditions of transient flow; and
- Incorporation of factors to allow for subsurface transport through solution channels, cracks, etc.

In addition, field validation of the model's predictions is necessary before the Pathogen Risk Assessment Model can be considered an accurate predictor of health risk.

2. INTRODUCTION

This preliminary risk assessment study focuses on the probability of human infection from enteric viral pathogens in municipal sludge applied to land. Sludge, a byproduct of sewage treatment, is the mixture of solids and liquids remaining after treatment processes remove solids from municipal or domestic wastewater. Secondary and tertiary sludges contain biomass resulting from microbial digestion of the sewage. Derived from human sanitary wastes, sludge contains microorganisms that colonize humans and can cause infection and disease.

This risk assessment is based on the Pathogen Risk Assessment computer model and methodology described in the U.S. Environmental Protection Agency's (U.S. EPA's) *Pathogen Risk Assessment for Land Application of Municipal Sludge* (U.S. EPA, 1989a). Appendix A provides an overview of the model. The purpose of the model is to determine the probability of infection of a human receptor from pathogens in land-applied sludge. The model consists of a series of compartments (Table 2-1) representing discrete points in the application pathway. The compartments are the various locations, states or activities in which sludge or sludge-associated pathogens exist; they vary to some extent among practices. Compartments representing sources of human exposure are designated with an asterisk in Table 2-1. In each compartment, pathogens increase, decrease or remain the same in number with time, as specified by "process functions" (growth, die-off or no population changes) and "transfer functions" (movement between compartments). Infection rather than disease is used to measure risk in the methodology, since exposures to pathogenic viruses may lead to no infection, human infection that is asymptomatic or subclinical (no illness), or human infection with illness (Kowal, 1985). The outputs produced by running the model are numerical values for the probability of a human receptor receiving an exposure exceeding the minimum infective dose (MID) in a 24-hr period. The MID for humans is considered to be as low as 1 virus particle, although infective doses can vary depending on the virus and susceptibility of the human receptor (Kowal, 1985). The model will run until the day specified or until the number of pathogens in each compartment decreases to <1, at which point the number is rounded to zero.

TABLE 2-1

Compartments Included in the Sludge Management Practices

Compartment Name and Number	Liquid Sludge Management Practices			Dried/Composted Sludge Management Practices	
	I	II	III	IV	V
Application	1	1	1	1	1
Incorporation	2	2	2		
Application/Tilling Emissions	3*[b]	3*	3*	3*	3*
Soil Surface	4	4	4	4	4
Particulates	5*	5*	5*	5*	5*
Surface Runoff	6*	6*	6*		
Direct Contact	7*	7*	7*	7*	7*
Subsurface Soil	8	8	8	8	8
Groundwater	9	9	9		
Irrigation Water	10	10	10		
Soil Surface Water	11	11	11	11	11
Offsite Well	12*	12*	12*		
Aerosols	13*	13*	13*		
Crop Surface	14	14	14	14	14
Harvesting	15		15	15	
(Commercial) Crop	16*			16*	
Animal Consumption		17	17		
Meat		18*	18*		
Manure		19	19		
Milk		20*	20*		
Hide		21	21		
Udder		22	22		

[a]Source: U.S. EPA, 1989a
[b]Asterisk indicates exposure compartments.

Two categories of land application are employed in the methodology: (1) agricultural utilization and (2) distribution and marketing (D&M). The source of pathogenic viruses is either liquid or dried/composted municipal sewage sludge. The five municipal sewage-sludge management practices (Table 2-2) included in the model are application of liquid treated sludge to (I) commercial crops for human consumption, (II) grazed pastures, and (III) crops to be processed before being consumed by animals; and application of dried or composted sludge to (IV) residential vegetable gardens and (V) residential lawns. Practices III and V, while ostensibly limited to hay fields and residential lawns, respectively, can be modified by selection of appropriate parameters to represent sludge application to golf courses, reclaimed strip mines or logged sites, parks, roadsides, etc. Although Practice V does not include an onsite pond, the risk to a human swimming in a pond (SWIMMER) can be modeled by using appropriate parameters in Practice III.

Risk assessment for pathogens in land-applied municipal sludges requires the following input data:

- Types of pathogens and their concentrations in the sludge, their survivabilities, and their infective doses;
- The sludge reuse/disposal option used and the conditions of sludge application (quantities, frequencies, application method);
- The fate of the pathogens in the environment, i.e., the inactivation rate under different conditions including moist soil, dry particulates, droplet aerosols and water; and
- The level of exposure of human receptors to the applied sludge.

In general, these data are sparse, and in many cases parameter values must be selected by using best scientific judgement. In addition, no field experimental work has been done to validate the predictions of the model. The purpose of this study is not to provide a definitive health risk assessment for viruses in municipal sewage sludge applied to land, but rather to identify parameters most in need of further research and validation. Therefore, this document serves as an evaluation of data needs for use of the model. The recommendations for further research are intended to provide those data, but field validation of the model will still be necessary for application of the model.

TABLE 2-2

Sludge Management Practices and Descriptions in Pathogen Risk Assessment Model

PRACTICE	DESCRIPTION[b]
I	Application of Liquid Treated Sludge for Production of Commercial Crops for Human Consumption
II	Application of Liquid Treated Sludge to Grazed Pastures
III	Application of Liquid Treated Sludge for Production of Crops Processed before Animal Consumption
IV	Application of Dried or Composted Sludge to Residential Vegetable Gardens
V	Application of Dried or Composted Sludge to Residential Lawns

[a]Source: U.S. EPA, 1989a
[b]Two types of sludge are used in this model - liquid and dried/composted. The extent of treatment or conditioning prior to application is variable and must be determined for each case.

This document reports the results of a literature review designed to find the viral data required by the pathogens methodology, and the results of numerous computer simulations, i.e., running the Pathogen Risk Assessment Model with a wide range of values for the parameters required. Six sites, chosen to provide diversity in geographic location, topography, soil type, rainfall pattern and temperature, were selected for site-specific applications of the model: Anderson County, TN; Chaves County, NM; Clinton County, IA; Highlands County, FL; Kern County, CA; and Yakima County, WA. Since the number of possible sites was essentially unlimited, the final selections, although somewhat arbitrary, were based on an attempt to represent different geographic regions and to ensure a variety of weather patterns.

Exposure pathways, i.e., migration routes of viruses from or within the application site to a receptor, for sludge applied to land include the following:

- Inhalation and ingestion of aerosols from the application of liquid sludge or wastewater;
- Inhalation and ingestion of windblown or mechanically generated particulates;
- Swimming in a pond fed by surface water runoff;
- Direct contact with sludge-contaminated soil or crops (including grass, vegetables, or forage crops);
- Drinking water from an offsite well;
- Consumption of vegetables grown in sludge-amended soil; and
- Consumption of meat or milk from cattle grazing on or consuming forage from sludge-amended fields.

Because the focus of the model is enteric pathogens, this methodology assumes that exposure to viruses will not result in infection unless the virus particles are actually swallowed. Risks due to inhalation of enteric pathogens will be considered only because the infectious agents can be subsequently swallowed. However, disease can result through routes of exposure other than the alimentary tract; risks from such exposures can be modeled by choice of the appropriate virus-specific parameter values.

The model calculates the probability of infection by viruses for the following human receptors:

- Onsite person (ONSITE) who is exposed by ingestion (includes pica in children) of soil, vegetables or forage, or by inhalation and subsequent ingestion of aerosols (particulates or liquid);
- Offsite person (OFFSITE) who is exposed to particulate or liquid aerosols carried by wind;
- Food consumer (EATER) who eats vegetable crops, meat or milk produced on sludge-amended soil;
- Groundwater drinker (DRINKER) who consumes water from a well near but not on the sludge application site;
- Pond swimmer (SWIMMER) who ingests a small amount of water while swimming in the pond that receives the surface runoff from the application site.

The model conceptualization (U.S. EPA, 1989a) specifies that workers engaged in the transportation, handling and application of liquid sludge are not included as exposed individuals because such activity is an occupational exposure.

The U.S. EPA (1986, 1988) has provided extensive information relevant to the conceptual risk assessment framework for land application of sludge. These key studies address the pathogens associated with sewage sludge, as well as exposure pathways and the potential risks to humans from each of the pathways. Most of that information will not be repeated here. Additional information about the computer model and methodology, including basic assumptions, model limitations and sources of uncertainty, is available in Volumes I and II of *Pathogen Risk Assessment for Land Application of Municipal Sludge* (U.S. EPA, 1989a) and in Wilson et al. (1989); a brief overview of the model is included as Appendix A.

3. LITERATURE REVIEW OF VIRUSES

A literature search was performed to find the most current information available for the parameters required by the model for simulating land application of sludge. This literature was reviewed for data on which to base the ranges of values for each of those parameters. The parameters required for viruses are (1) minimum infective dose; (2) density of infective viruses in treated sludge destined for land application; (3) inactivation rates in soil, dry particulates, liquid aerosols and water; and (4) dispersion in the environment, i.e., transport in air, soil and water.

Appropriate codes and keyword truncation were used to produce the most effective search strategy for query of each data base. Table 3-1 lists the computerized data bases queried and the keywords used. The three columns of keywords were "anded" together to produce a set in which at least one keyword in each column was a descriptor or was contained in a retrieved record.

References in reviews and in relevant articles retrieved by the computer search were also evaluated, and names of pertinent authors were searched to find recent papers. Because the scope of this literature review is limited to information satisfying the parameters required by the model, the reader is directed to cited references for more comprehensive background information.

3.1. SIGNIFICANCE OF PATHOGENIC VIRUSES

The presence of pathogenic viruses in sewage sludge has been well-researched and documented. Several reviews include information on specific types of viruses present in municipal sewage, their persistence and density in sludge, their pathogenicity, and potential health risks associated with land application (WHO, 1981; Kowal, 1985; Carnow et al., 1979; Loehr et al., 1979; IAWPRC, 1983; U.S. EPA, 1986, 1988; Lund, 1978; Burge and Marsh, 1978; Pedersen, 1981; Elliot and Ellis, 1977; Feachem et al., 1983).

From the Latin word meaning poison, the word *virus* was originally used to mean any poison or noxious agent. Organisms that could pass through bacteriologic filters and be transferred serially from one animal to another were termed "filterable viruses" and later,

TABLE 3-1

Computer Search Strategy

Data Bases	Keyword Groups		
AGRICOLA	VIRUS	SURVIVAL	SEWAGE
AGRIS	ENTEROVIRUS	DISPOSAL	SOIL
BIOSIS	ENTERIC VIRUS	TRANSPORT	AIR
CAB ABSTRACTS	POLIOVIRUS	FATE	AEROSOL
CRIS/USDA	COXSACKIE(VIRUS)	VIABILITY/	WATER
ENVIROLINE	ECHOVIRUS	VIABLE	SLUDGE
FSTA	HEPATITIS A	DIE-OFF	GROUND-WATER
NTIS	ADENOVIRUS	MOVEMENT	
POLLUTION ABSTRACTS	REOVIRUS		
TOXLINE	ROTAVIRUS		
WATER RESOUR. ABS	PARVOVIRUS		
ZOOLOGICAL RECORD	CORONAVIRUS		
	NORWALK/ NORWALK-LIKE		
	PAPOVAVIRUS		
	ASTROVIRUS		
	CALICIVIRUS		

viruses. The complete infectious virus particle is called the virion, and it consists of nucleic acid or nucleoid (DNA or RNA), a protein shell (capsid), and, for some viruses, an envelope derived from the host cell membrane. As obligate intracellular parasites, viruses lack independent metabolism and can replicate only within living host cells (Dorland's Illustrated Medical Dictionary, 1981; Kucera, 1983). As a group, the viruses are the smallest agents of infectious diseases, ranging in size from 20 nm (e.g., poliovirus) to 200-300 nm (Berk, 1983; Yates and Yates, 1988).

Although viruses cannot multiply outside a host cell (unlike bacteria, which can grow extracellularly in a suitable nutrient-rich medium if conditions are favorable) and will always decrease in numbers following excretion, the enteric (relating to the intestines or, more generally, the alimentary tract) viruses can persist in the environment for many weeks, particularly if temperatures are cool (<15°C) (Feachem et al., 1983).

Table 3-2 lists the major viruses found in wastewater and therefore those most likely to be of concern from the land application of municipal sewage sludge.

3.1.1. Transmission/Exposure Routes. Many viruses infect the intestinal tract, are excreted in the feces, and can infect new human hosts, following either their direct ingestion or their mucociliary translocation and subsequent ingestion after being inhaled by the host (Slote, 1976). While the concentration of viruses in the feces of an uninfected person is normally zero (Kowal et al. 1981), concentrations of $>10^6$ to 10^9 infectious virus particles may be shed in 1 g of human feces from an infected individual, even if that person does not exhibit frank disease (Feachem et al., 1983).

Transmission of enteric viruses is by the fecal-oral route, with water- and food-borne outbreaks being of major importance; by direct personal contact or contact with contaminated surfaces or fomites; by contact with recreational water; and possibly by the airborne route. Although the fecal-oral route of transmission of enterovirus infections is well-established, it is not fully understood. Ingestion of contaminated food or water or swimming in contaminated water are important routes of viral infections (Cliver, 1987). Human exposure to viruses in surface water is possible through deliberate or accidental ingestion as well as through contamination of facial mucosal surfaces during recreational

TABLE 3-2

Human Viruses in Sludge and Wastewater

Virus	Disease or Symptoms
Enteroviruses	
Poliovirus	poliomyelitis, meningitis, fever
Coxsackievirus A	herpangina, respiratory disease, meningitis, fever
Coxsackievirus B	myocarditis, congenital heart anomalies, meningitis, respiratory disease, pleurodynia, rash, fever
Echovirus	meningitis, respiratory disease, rash, diarrhea, fever
New Enteroviruses	acute hemorrhagic conjunctivitis, meningitis, encephalitis, respiratory disease, fever
Hepatitis A Virus	infectious hepatitis
Rotavirus	acute gastroenteritis with severe diarrhea, vomiting
Norwalk-like Agents (or Small Round Viruses, SRVs)	epidemic gastroenteritis with diarrhea, vomiting, abdominal pain, headache, myalgia
Adenovirus	respiratory and eye infection
Reovirus	possibly fever, diarrhea and upper respiratory disease, but relationship to clinical disease in humans is not clear
Papovavirus	may be associated with progressive multifocal leukoencephalopathy
Astrovirus	may be associated with gastroenteritis, diarrhea
Calicivirus	gastroenteritis
Coronavirus-like Particles	respiratory tract infections
Parvovirus and Parvovirus-like Agents	gastroenteritis, aplastic anemia, fever, rash, fetal death or damage including hydrops fetalis
Non-A non-B Hepatitis	hepatitis
Snow Mountain Agent	gastroenteritis
Pararotavirus	gastroenteritis
Sources: Kowal, 1985; Feachem et al., 1983; Kucera, 1983; Akin et al., 1978; U.S. EPA, 1986; Rao et al., 1986; Levy and Read, 1990	

water use (Hurst et al., 1989). Following sludge application, these viruses can find their way into surface waters either by overland runoff or infiltration into the subsurface and subsequent transport to an intersection with the surface.

Although it is unknown whether animals serve as reservoirs for viruses likely to be transmitted from land-applied sludge via soil or water (Kowal, 1985), it has been proven that consumption of virus-contaminated shellfish, especially raw shellfish, from seawater or estuarine habitats is a persistent problem. Shellfish can serve as a virus reservoir in nature because of the possible bioaccumulation of viruses associated with particulates and the persistence of those viruses for up to months if shellfish are dormant (Metcalf, 1987).

Viral contamination of water supplies may lead to localized epidemics of viral gastroenteritis. More than 5000 persons suffered from gastroenteritis caused by Norwalk-like virus in a multistate outbreak traced to commercially-produced ice made from contaminated well water. That well was flooded by a creek during heavy rains. Nearby residents with private wells flooded by the same creek were believed to have been infected (Levine and Craun, 1990).

Acute gastrointestinal illness (AGI) of unknown etiology comprised 48% of the outbreaks of waterborne disease reported by the Waterborne Outbreak Surveillance System in 1986-1988. Characteristics of these outbreaks suggest that many were caused by Norwalk-like viruses. These reported outbreaks were associated only with water intended for drinking and probably represent only a small percentage of all illnesses associated with waterborne-disease agents. Additional reports of illness associated with recreational water use during 1986-1988 included 41 cases of gastroenteritis caused by a Norwalk-like agent in a lake. Hundreds of other cases of AGI were attributed to recreational water use, and at least some of these were undoubtedly caused by viruses. The authors indicate that improving the availability of testing for viral serology and detection of viral antigen in stool would aid in determining the etiology of these outbreaks of AGI (Levine and Craun, 1990).

Transmission of viruses by the airborne route has been well established (Sattar and Ijaz, 1987). A number of environmental factors affect the survival of viruses in aerosols, particularly temperature and relative humidity, as discussed in Section 3.3.3 (Spendlove and Fannin, 1982; Sattar and Ijaz, 1987). Foster et al. (1980), reporting on a rotavirus epidemic

on Truk Island, suggested that in addition to the fecal-oral route, human rotavirus infection may have been spread by the respiratory route. Prince et al. (1986) indicate that the airborne route could be a pathway for the spread of gastrointestinal tract infections caused by rotavirus. Sattar and Ijaz (1987) emphasize that the potential exists for airborne transmission of all viruses that can survive the aerosolization process, even if the typical or dominant route of transmission is direct person-to-person contact or some other vehicle such as food, water or fomites.

3.1.2. Occurrence of Viruses in Sludge. Although viruses do not normally inhabit the gastrointestinal tract of uninfected persons, the incidence of viral infection is high enough that viruses are ubiquitous in sewage. Many of the viruses in wastewater adsorb to suspended particles and are removed in sewage treatment processes, becoming concentrated in the sludge product. Excreted viruses are described by Feachem et al. (1983), and their occurrence and persistence in the environment, their inactivation in treatment processes, and their transmission routes and epidemiology are discussed. U.S. EPA (1988), Pedersen (1981), Hurst (1989), Englande (1983) and Ward et al. (1984) review viral concentrations in sludge and the effectiveness of sludge treatment processes in inactivating viruses. Information on the densities of viruses in treated sludge is summarized in Section 3.2.2.

3.1.3. Infective Dose. The ability of pathogenic viruses to cause infection and disease in the human exposed to them depends on the number and virulence (infectivity or pathogenic potential) of the virions and the susceptibility of the host. Host factors include the route of entry, the mode of virus spread, and the resistance and immunity factors of the human receptor (Menna and Soderberg, 1983). Consequently, infection is often considered a dose-response relationship in which the dose is the number of virions to which the human is exposed and the response is the level of infection, i.e., no infection, subclinical infection with no disease, or infection with disease (Kowal, 1985).

Estimating infective dose can involve exposing volunteers to known doses of the virus, inferring from epidemiologic data the probable levels of exposure associated with observed frequencies of infection or disease, or measuring cytopathic effect by infecting tissue cultures. When virus concentrations are determined by infecting an indicator cell line, the information gained is a ratio of infective activities. The most quantitative determination,

the plaque assay, yields a concentration of infective virus units, but the dose response of plaque formation must also be known because, in some instances, more than one infectious virus particle may be required to initiate plaque formation. When infection can only be measured by cytopathic effect, it may be necessary to calculate a tissue culture infective dose (usually $TCID_{50}$ or TCD_{50}, i.e., dose required to infect 50% of the cultures). In this case, also, the dose response of the indicator cell line to infection must be known for an accurate calculation of dose response in humans (U.S. EPA, 1989a).

The MID, or minimum infective dose, is generally considered to be the dose that will infect 50% of the population (U.S. EPA, 1988). It is now thought that a few virus particles can produce an infection if conditions are favorable, and data suggest that the infective dose of enteroviruses to humans is possibly 10 or fewer virus particles. Reported infective doses may vary widely, however. For example, the oral infective dose to humans for poliovirus ranges from 1 to $1x10^{7.6}$ $TCID_{50}$, and the range of reported plaque-forming units (PFU) is 0.2 to $5.5x10^{6}$ (Kowal, 1985). In a review of the issue of minimum human infectious dose (IAWPRC, 1983), the evidence suggests that while the minimum dose of enteric viruses required to produce infection in healthy adults may be much larger than 1 PFU, susceptible individuals are much more likely to be infected by a single PFU. Ward and Akin (1983), in their review of minimum infective doses of animal viruses, urge more studies using larger numbers of subjects and employing the most current techniques to answer some of the questions regarding viral infective doses.

Ward et al. (1986) exposed volunteers to human rotavirus in doses ranging from $9x10^{-3}$ to $9x10^{4}$ focus-forming units (ffu). The human 50% infectious dose was ~10 ffu, and it was estimated that ~25% of susceptible adults would be infected by 1 ffu. Since the occurrence of illness was not dose-related, it appeared that the same dose that could cause infection could also cause illness.

Schiff et al. (1984) found that the oral 50% human infective dose (HID_{50}) of echovirus-12 in volunteers was 919 PFU, and by statistical analysis they predicted a 1% infective dose (HID_{1}) of 17 PFU. Their results indicated that previous infection did not provide lasting protection of volunteers against reinfection.

3.1.4. Epidemiology. According to Yates (1990), "viruses may be responsible for one-third of all the waterborne disease outbreaks that occur in this country." Wellings (1987) suggests that as many as 60% of waterborne disease outbreaks may be caused by viruses. Historically, viruses have been difficult to detect and isolate from environmental media and from clinical samples. As detection methods have improved, there has been an increase in the percentage of waterborne diseases characterized as virus-caused (Gerba, 1984c).

Several of the viruses associated with sewage sludge and possibly with human gastroenteritis are poorly understood, such as papovaviruses, astroviruses, caliciviruses and coronavirus-like particles (Kowal et al., 1981). Some of these have not been well-characterized because they cannot be grown *in vitro* in cell cultures (Righthand, 1983). Reliable, comparable information on others is limited by inadequate detection methods. In fact, Rao et al. (1986) characterize the extraction, concentration and enumeration techniques for viruses in soils as "marginally efficient" for some enteroviruses and "totally inadequate" for detecting HAV, Norwalk virus, and human rotaviruses. They conclude that detecting any enteric virus in soil implies that many other types may be present.

Epidemiologic evidence of adverse health effects associated with exposure to viruses from land application of sludge or wastewater has been mixed. Katzenelson et al. (1976) found sewage-associated disease incidence 2-4 times higher in Israeli kibbutzim using wastewater for spray irrigation than in those not using wastewater. However, the researchers did note the possibility that pathogens could expose the affected population by other routes, such as on the clothes of irrigation workers returning from the fields. The microbiologic quality of the wastewater was similar to that of raw wastewater and the likelihood of disease transmission is probably increased by the closed nature of the kibbutzim communities.

Fattal et al. (1986a), in a subsequent retrospective study, noted a 2-fold excess risk only in the 0-4 year old age group during summer irrigation months but no significant risk on a year-round basis. In a prospective epidemiologic morbidity and serology study (Fattal et al., 1986b), no excess rate of enteric disease was seen in kibbutzim using wastewater aerosols compared with those using no wastewater or using wastewater but not exposed to aerosols. There was a significant excess of ECHO 4 virus antibodies found in the 0-5 and

6-17 year old groups, compared with all other categories of those exposed to wastewater aerosols from nearby towns, but there was no morbidity excess. The authors conclude that little or no wastewater-associated health risks were detected in spite of the poor water quality of the effluent used, but they caution that the possibility of aerosol transmission of viruses "under extraordinary circumstances is supported by the circumstantial evidence provided by the serological study." Ward et al. (1989), as part of the Lubbock Infection Surveillance Study, found that wastewater spray irrigation had no detectable effect on the incidence of rotavirus infection in a community surrounding a spray irrigation site.

Johnson et al. (1980b) found no significant health hazards to residents near a new sewage treatment plant in Schaumburg, IL, and they were unable to distinguish levels of microorganisms in the air of residential areas from background levels.

Recent and current epidemiologic studies are providing important additional information on the survival and transport of viruses in aerosol. Currently, however, the preponderance of evidence suggests that, even though it is thought aerosolized pathogens may have a lower infective dose than when ingested (Loehr et al., 1979), public health risks from aerosolized enteric viruses do not appear to be of major concern.

Clark et al. (1980) did not demonstrate an increased risk of infection from viruses in wastewater workers, although there was evidence of increased minor gastrointestinal illnesses in inexperienced sewage-exposed workers compared with experienced workers. However, the illnesses, occurring during the second quarter of the year, did not correspond to enteroviral infections.

Loehr et al. (1979) list normal precautions that should be observed at land application sites to protect workers, but they also point out that continual low-level exposure to pathogens can be beneficial to workers by building their immunity to infectious diseases.

Several viruses, including Norwalk agent in the United States (Dolin et al., 1971), the W agent in Britain (Clarke et al., 1972), and rotaviruses in many countries (Davidson et al., 1975), have been associated with gastroenteritis. Astroviruses were isolated from feces of 17 of 27 symptomatic children suffering from gastroenteritis in a pediatric ward in England (Kurtz et al., 1977). Human calicivirus (HCV), an important cause of gastroenteritis in day care centers (Matson et al., 1989), was detected in 32% of symptomatic

cases of gastroenteritis in a day care center in Australia, but the mode of transmission could not be identified (Grohmann et al., 1991). The authors suggest that HCV may be a common cause of gastroenteritis that is underrecognized because of insensitive detection methods.

Lew et al. (1990) reviewed 6 years of viral gastroenteritis data in which 16% of specimens were positive for a virus. The most commonly observed agent was rotavirus (26-83%), followed by adenoviruses (8-27%, including respiratory and enteric) and small round viruses (SRVs) (0-40%). Rotavirus and astrovirus detections were most common in winter. Lew et al. (1990) attribute insensitive screening methods to the underestimation of disease prevalence cause by astroviruses, caliciviruses and SRVs.

Payne et al. (1986) studied viral agents associated with gastroenteritis over an 8-year period. Of the stool specimens submitted, 41% were positive for viruses or virus-like particles belonging to 7 groups: coronavirus-like particles were present in 69.8% of positive specimens, rotavirus in 17%, adenovirus in 4.5%, picornavirus/parvovirus agents in 2.9%, Norwalk-like agents in 2.9%, astrovirus in 1.9%, and calicivirus in 0.5%, with 0.5% unclassified SRVs. Excretion of all viruses except coronavirus-like particles exhibited a seasonal distribution, with the majority of viruses being identified in the cooler, drier months.

The Norwalk agents cause ~36% of the outbreaks of infectious, nonbacterial gastroenteritis; they have been transmitted by ingestion of contaminated food and water (Righthand, 1983). Keswick et al. (1985) suggest that the Norwalk agent is responsible for ~23% of all reported waterborne outbreaks.

The total reported cases of hepatitis A was 28,507 in 1988, the highest number reported since 1980. Total poliomyelitis cases were 9 in 1988 with a range of 6-34 cases annually over the past 20 years (CDC, 1989).

In children between 6 and 24 months, rotavirus is the most frequent cause of nonbacterial gastroenteritis, but frequently neonatal infections are asymptomatic. Rotavirus infections occur less frequently among children 5 years old or older because by age 5 they have acquired circulating antibody to rotavirus (Estes et al., 1983). The spread of rotaviral infections is poorly understood, but it is thought that air may be a vehicle for these viral

outbreaks (Ijaz et al., 1985c). Viral survival was greatest at 50% relative humidity, and reducing the temperature from 20°C to 6°C enhanced that survival. Even after 24 hours, there was only a 30% reduction in infectivity of the virus. In addition, rotavirus suspended in fecal matter from an infant with rotaviral diarrhea survived longer as an aerosol than rotavirus suspended in tryptose phosphate broth (TPB). After 24 hours, the fecal suspension rotavirus lost <20% infectivity compared with a 50% reduction in infectivity under the same conditions for the TPB suspension of rotavirus (Ijaz et al., 1985c). These results support the suggestion of Brandt et al. (1982) that a low outdoor temperature, low indoor relative humidity and indoor crowding may contribute to rotavirus survival and spread of rotavirus infections. Likewise, epidemiologic observations seem to correlate rotaviral gastroenteritis outbreaks with climate, i.e., outbreaks in cool, dry weather of tropical climates and during winter in temperate zones (Ijaz et al., 1985c; Brandt et al., 1982).

Ward et al. (1986), in their study of infectious dose of rotavirus in adults, suggest several problems with epidemiologic understanding of adult rotavirus infections. They point out that many cases of adult gastroenteritis may be caused by rotavirus because the dose required to cause infection and illness is very small (1-10 ffu). The lack of evidence of high incidence of adult rotavirus illness could be because infected adults may manifest the disease differently than children or because the amount of virus shed by infected adults is below detection limits of current assay methods.

Most of the well-known viruses that are dangerous to fetuses of women infected with systemic viral illness during pregnancy, such as rubella, cytomegalovirus, and herpesvirus (Gold and Nankervis, 1989; Nahmias et al., 1989), are not typically found in sewage sludge. It should be recognized, however, that some viruses that may be present in sludge have been found to be associated with risk of adverse effects on the fetus or neonate. These include human parvovirus B19, which can result in fetal hydrops and death (Levy and Read, 1990) and enteroviruses, such as echovirus and coxsackievirus B, associated with fetal heart effects and neonatal disease ranging from asymptomatic infection to death (Modlin, 1988; Rosenberg, 1987).

It appears that the difficulties inherent in epidemiologic studies are particularly significant in determining the incidence and distribution of viral infections and diseases.

More research is needed in this area to characterize the potential public health risks associated with transmission of viruses by application of sludge and wastewater.

3.2. ENTERIC VIRUSES IN TREATED SLUDGE

The frequency of isolation and the quantity of virus recovered from sewage depend upon water use in a community, the day and season of sample collection, the level of infection in the population and the efficiency of recovery procedures (Rao et al., 1986). Concentrations of viruses in wastewater in the United States vary greatly, reflecting the infection and carrier status of the population. The larger the contributing population, the more uniform the viral concentration becomes. Concentrations tend to peak in late summer and early fall when enteric viral infections increase (Kowal, 1985).

3.2.1. Effects of Treatment Processes. Wastewater treatment processes are less effective in removing viruses than in removing bacteria (Sobsey et al., 1980). Viruses in wastewater tend to become adsorbed to suspended particles; consequently, during primary and secondary sewage treatment processes, viruses will be removed from the wastewater and concentrated in the sludge. Chlorination is not as effective in destroying viruses in effluent wastewater as it is in killing bacteria (Feachem et al., 1983). Viruses in wastewater may be protected by particulates so that longer exposure times or higher chlorine concentrations may be required to destroy them (Sproul, 1978). According to Hurst (1989), wastewater sludges cannot be readily disinfected by chlorine because of their solid nature and high organic content.

The most important factors affecting the stability of viruses in wastewater sludges are temperature, loss of moisture, and the presence of aerobic microorganisms. Other factors affecting viruses in sludges are pH levels; presence of detergents, ammonia and certain salts; and the type of virus (Hurst, 1989).

In his review of density levels of pathogenic organisms in municipal wastewater sludges, Pedersen (1981) concludes that levels of enteroviruses in primary and secondary sludges are similar; few data were available on levels of enteric viruses in mixed sludge. Primary sludge has received primary treatment such as screening and settling; secondary

sludge is produced by biologic waste treatment, or secondary treatment; primary and secondary sludge are combined to produce mixed sludge (U.S. EPA, 1986).

Conventional sludge treatment processes designed to stabilize sludge and reduce volatile solids also lower densities of pathogens in sludge to varying degrees. These processes are: (1) anaerobic digestion, the microbiologic degradation of the organic matter in sludge in the absence of oxygen; (2) aerobic digestion, the biochemical oxidation of organic matter; (3) composting, the natural, aerobic microbiologic process of decomposing organic matter to produce humus; (4) lime stabilization, the application of lime to sludge to raise the pH; and (5) air drying, exposure of a layer of sludge to air to drain or dry. These sludge treatment processes reduce but do not eliminate viruses (Melnick, 1987). U.S. EPA has established regulations in 40 CFR 257 for these processes to qualify as Processes to Significantly Reduce Pathogens (PSRP). Additional processes, either singly or in combination with PSRPs, have been defined as Processes to Further Reduce Pathogens (PFRPs).

Anaerobic Digestion. Following high-rate anaerobic digestion at 35°C, Jewell et al. (1980) and Berg and Berman (1980) report log reductions for enteroviruses of 1.36 and 1.05, respectively. Eisenhardt et al. (1977) and Sanders et al. (1979) found that by raising the temperature 3°C during mesophilic anaerobic digestion, two- to four-fold increases in viral inactivation rates were achieved. In full-scale anaerobic digesters, Ohara and Colbaugh (1975) observed that mesophilic digestion achieved a 1 log reduction in virus content while thermophilic digestion achieved a 3 log reduction. Although virus reductions of 1 log (90%)/day at 30°C have been demonstrated in studies of anaerobic digestion with seeded enterovirus, Moore et al. (1978) reported a reduction of 1 log/week at 30°C for poliovirus when it was bound to the sludge solids.

During sludge treatment processes, temperature is not always the most important factor responsible for virus inactivation; the relative effects of heat are dependent on the temperature range (Traub et al., 1986). When the effects of mesophilic and thermophilic anaerobic digestion and aerobic thermophilic fermentation were examined, heat accounted for 19% of bacteriophage f2 inactivation at 34.5°C (mesophilic anaerobic digestion), for 32% at 54.5°C (thermophilic anaerobic digestion) and, in combination with pressure, for

100% of inactivation at 60°C (aerobic thermophilic fermentation). The authors of this study suggest that enteric viruses would be inactivated by 1 day of thermophilic anaerobic digestion (54-55°C) and that virus recovery after thermophilic digestion, as reported by Lund (1971) and Berg and Berman (1980), probably resulted from short circuiting in the sludge digesters.

Inactivation rates will vary with the type of virus. Bertucci et al. (1977) report significant differences in inactivation rates among viruses during mesophilic anaerobic digestion. Poliovirus I (Sabin) was 98.8% inactivated after 2 days of digestion at 35°C.

According to Spillmann et al. (1987), mesophilic anaerobic digestion achieved only minor inactivation of a human rotavirus, coxsackievirus B5, and bovine parvovirus. With thermophilic digestion, the rotavirus and coxsackievirus were rapidly inactivated; the parvovirus was heat stable. Temperature was the predominant contributor to total inactivation only for processes above 54°C. The authors classify these viruses as: thermolabile/comparatively chemoresistant (rotaviruses and enteroviruses), and thermostable/chemolabile (parvoviruses). "Chemoresistant" is defined as relatively insensitive to ammonia, detergents and microbial factors in the sludge.

The presence of aqueous ammonia enhanced the inactivation of poliovirus types 1 and 2, coxsackievirus A13 and B1 and echovirus 11 during anaerobic digestion, according to Ward (1977). However, reovirus was insensitive to the concentration of ammonia. Cationic detergents present in wastewaters increase susceptibility of reoviridae to heat inactivation; these same detergents protect enteroviruses from heat (Goddard et al., 1981; Ward et al., 1976).

Although sludges with undetectable viral levels were not produced by any of the treatments examined, anaerobic mesophilic digestion with subsequent thickening and aerobic thermophilic digestion were both found to be efficient in reducing infectious virus concentrations (Goddard et al., 1981). Anaerobic mesophilic digestion alone was not reliable in reducing virus levels in sludge.

Large quantities of sludge that had been anaerobically digested and lagooned were applied to a 15,000-acre site in central Illinois by the Metropolitan Sanitary District of Greater Chicago during 1971-1978 (MSDGC and IIT Research Institute, 1979). Water, soil,

and sludge sampling detected no viruses in the sludge product applied or sprayed and no effects on the surface or groundwater at the site.

Aerobic Digestion. In laboratory bench studies of mesophilic aerobic digestion, Scheuerman (1984) observed log reductions of 0.219-0.779 for poliovirus type 1, 0.189-0.59 for echovirus, 0.439-0.449 for rotavirus SA-11, and 0.469 for coxsackievirus. Ward et al. (1984) conclude that standard aerobic digestion probably achieves pathogen reductions equal to or greater than those achieved in mesophilic anaerobic digestion.

Composting. Although hepatitis A has been shown to withstand temperatures of 80°C, most enteric viruses succumb to composting temperatures (U.S. EPA, 1988; Pedersen, 1981). Inactivation depends on equal distribution of heat throughout the compost pile (Englande, 1983). In seeding experiments with bacteriophage f2, which is more heat resistant than most enteric viruses, Burge et al. (1978) studied the effects of windrow and static pile composting methods on viral inactivation. Virus concentrations in windrows decreased 90% every 4-7 days in dry weather, and 50% of this rate in wet weather. Reductions were greater in aerated piles. According to Ward et al. (1984), enteric viruses would be inactivated at greater rates; but some viruses, such as hepatitis A and parvovirus (Spillman et al., 1987), may be more heat resistant.

Lime Treatment and Air Drying. According to Ward et al. (1984), it has not been shown that viruses in sludge are inactivated by lime treatment, but viruses have been shown to be destroyed rapidly at high pH values. Pedersen (1981) reports a single laboratory-scale study (Sattar et al., 1976) in which a >4-log reduction of poliovirus type 1 was achieved in the sludge product resulting from injection of the virus into raw sewage that was limed to pH 11.5, settled and centrifuged.

Ward and Ashley (1977) observed that the air drying process inactivates enteric viruses in sludge. Inactivation of enteric viruses seeded into raw sludge was proportional to water loss until the sludge reached ~70% solids; viral concentrations decreased 3 orders of magnitude between 70 and 90% solids. The drying process itself apparently causes the inactivation since storage of viruses in sludge at low moisture levels has little effect on persistence. Similar results were observed for indigenous viruses in air-dried sludge by Brashear and Ward (1983).

Conclusions. Ward et al. (1984), U.S. EPA (1988), Hurst (1989), Rao et al. (1986), Englande (1983) and Yanko (1988) discuss the potential for reduction of enteric viruses by conventional sludge treatment processes. Ward et al. (1984) summarize viral reductions in sludge treatment processes as follows:

anaerobic digestion (mesophilic)	0.5-2 log reductions
aerobic digestion	0.5-2
composting	2->4
air drying	0.5->4
lime stabilization	>4

They conclude that composting is the best of conventional sewage treatment methods since the high temperatures destroy most sludge pathogens. The results of Yanko (1988) support the conclusions of Ward et al. (1984).

3.2.2. Density of Viruses in Treated Sludge. Viral densities in raw sewage will vary with season, region, and the nature and size of the population. Akin and Hoff (1978) report that the concentration of viruses in raw wastewater is most often < 1000 units/L; however, Sorber and Guter (1975) estimate 7000 PFU/L. Gerba (1983a) reports a range of 2-215 enteric virus units/g of raw sludge in the United States. According to Booz-Allen and Hamilton, Inc. (1983), the density of viruses in raw sludge is several hundred PFU/L, and Melnick (1987) reports virus concentrations of 5000-28,000 PFU/L in raw sludge. Ward et al. (1984) report densities of 10^2-10^4 enteric viruses/g dry weight in primary sludges and $3x10^2$ organisms/g dry weight in secondary sludges. U.S. EPA (1988) and Pedersen (1981) report average geometric mean values for enteric viruses of $3.9x10^2$ PFU/g dry weight in primary sludge, $3.2x10^2$ PFU/g dry weight in secondary sludge, and $3.6x10^2$ $TCID_{50}$ in mixed sludge. Due to the limitations of recovery procedures, the actual numbers of viruses in sludges and wastewater could be 1-2 logs higher than those reported (Akin et al., 1978). Table 3-3 lists virus densities in treated sludge.

Anaerobic Digestion. Limited data are available on the effects of sludge treatment processes on indigenous sewage viruses. Mesophilic anaerobic digestion results in significant decreases (90%) in viral concentrations, and thermophilic digestion reduces concentrations by at least 99% (Berg, 1978). The results of Jewell et al. (1980), Berg and Berman (1980)

TABLE 3-3

Virus Densities in Sludge

Virus	Sludge Treatment Process	Mean Density no/100 mL	Detention Time (days)	Conditions	Reference
Enteroviruses	Anaerobic Digestion	5.9 PFU/100 mL, range 1.3-17 PFU/100 mL	12, 8	30°C, 7-10°C	Cliver, 1975
Enteroviruses	High Rate Anaerobic Digestion	19 PFU/100 mL, range 4-100	21	35°C	Jewell et al., 1980
Enteroviruses	High Rate Anaerobic Digestion	138 PFU/100 mL, range 30-410	20	35°C	Berg and Berman, 1980
Enteroviruses	High Rate Anaerobic Digestion	<3.3 PFU/100 mL, range <1.4-16.7	20	49°C	Berg and Berman, 1980
Enteroviruses	Aerobic Digestion	0.3-1.2 $TCID_{50}$/g dry wt	90	mesophilic	Bitton et al., 1980
Enteroviruses	Aerobic Digestion	ND	180	mesophilic	Bitton et al., 1980
Enteroviruses	Aerobic Digestion	53 $TCID_{50}$/g dry wt, range 14-260 $TCID_{50}$/g dry wt	30	mesophilic	Bitton et al., 1980
Enteroviruses	Aerobic Digestion	3.35 PFU/g dry wt		mesophilic	Hurst et al., 1978
Enteric Viruses	None	2-215 units/g			Gerba, 1983a
Enteric Viruses	Anaerobic Digestion	0.04-17 units/g			Gerba, 1983a
Enteric Viruses	Anaerobic Digestion	0.007 PFU/mg TSS	3		Moore et al., 1978

TABLE 3-3 (continued)

Virus	Sludge Treatment Process	Mean Density no/100 mL	Detention Time (days)	Conditions	Reference
Enteric Viruses	3 stage Anaerobic digestion	1.2 MPNCU/100 mL, range 0-19 MPNCU/100 mL	40	33°C (for 10 days)	Palfi, 1972
Enteric Viruses	Anaerobic Digestion	0.007 PFU/mg TSS	5		Moore et al., 1978
Enteric Viruses	Anaerobic Digestion	0.007-0.04 PFU/mg TSS			Moore et al., 1978
Enteric Viruses	Aerobic Digestion	0-260 units/g			Gerba, 1983a
Picornavirus (ECHO)	Windrow Composting	<2.3 PFU/g	40-90		Yanko, 1988
Picornavirus (ECHO)	Static Pile Composting	<2.3 PFU/g	21 composting, 30 curing		Yanko, 1988
Reoviruses	Anaerobic Digestion	8 PFU/100 mL, range 6-17 PFU/100 mL	12, 8	30°C, 7-10°C	Cliver, 1975
Echovirus type 7	Sand Drying	0.1 PFU/g	13		Wellings et al., 1976
Viruses	Mesophilic Digestion	50-360 PFU/100 mL	20	~35°C	Berg, 1978
Viruses	Thermophilic Digestion	1.7-16.7 PFU/100 mL	20	49°C	Berg, 1978
Viruses	Anaerobic Digestion	2.1 PFU/g 0.03 PFU/g		mesophilic thermophilic	Ohara and Colbaugh, 1975

$TCID_{50}$ = Tissue culture infectious dose for 50% response
ND = Not detected
MPNCU = Most probable number of cytopathogenic units
TSS = Total suspended solids
Sources: Pedersen, 1981; U.S. EPA, 1988; Kowal, 1985.

and Cliver (1975) suggest a range of mean densities for enteroviruses of 5.9-138 PFU/100 mL following mesophilic digestion, and Berg and Berman (1980) report a mean density of <3.3 PFU/100 mL after anaerobic digestion under thermophilic conditions.

Aerobic Digestion. Little research has been done on the effects of mesophilic aerobic digestion on viral pathogens in sludge (U.S. EPA, 1988). Bitton et al. (1980) tested sludges for enteroviruses at 30, 90, and 180 days following mesophilic aerobic digestion. No viruses were detected after 180 days, and mean concentrations were greatest (53 $TCID_{50}$/g dry weight) with the shortest digester time (30 days). Kabrick et al. (1979) report that virus concentrations (in total PFUs) are below detection levels when the digestion temperature is ≥40°C and pH is >7.

Composting. Most studies of viral inactivation during composting have involved seeding of viruses prior to mixing the piles. A recent U.S. EPA study looked at indigenous pathogens in sludge following composting. An aerated static pile composting facility and a windrow composting facility designed to meet PFRP criteria were sampled weekly for a year by Yanko (1988). Elution/concentration techniques proven effective for high-solid samples were used in viral testing, and, in addition to several procedures on two cell lines in the project laboratory, samples were tested by different procedures in two other laboratories. However, during the testing period, indigenous viruses (untypable picornavirus) were found in only two samples, one from each facility. Isolates from "blind seeds" were found in six other samples. The picornavirus was found at a level below the quantitative limit for the plaque assay, <2.3 PFU/g. During the bimonthly sampling for a year of 24 treatment facilities (including anaerobic and aerobic digestion, heat drying, and composting facilities), viruses were detected in only one sample and were probably laboratory contaminants, according to the author. Yanko (1988) concludes that none of the treated sludges in this study produced viral health hazards since only two low-level isolations were made from many samples.

Lime Stabilization and Air Drying. Little research has been done on the effectiveness of the lime stabilization process in destroying indigenous sludge viruses, although viruses are known to be destroyed by high pH (Ward et al., 1984). Koch and

Strauch (1981) report inactivation of poliovirus in raw and digested sludges at pH levels resulting from lime treatment.

Several studies have reported the detection of indigenous enteric viruses in lime sludges at pH 10 or 10.5 (Pancorbo et al., 1988). Pancorbo et al. (1988) suggest that the pH achieved and maintained determines the effectiveness of this treatment. In their experiments, almost complete inactivation of seeded poliovirus type 1 was attained when alum sludge was treated with lime at pH 11.5. Similarly, when Sattar et al. (1976) treated raw sewage with lime at pH 11.5, only 0.005% of the poliovirus type 1 input was recovered from the lime sludge. In the United Kingdom, Goddard et al. (1982) did not detect indigenous enteroviruses in sludge conditioned with lime at pH 11.

Laboratory studies and seeding experiments suggest that air drying is an effective method of inactivating enteric viruses (Ward et al., 1984). Wellings et al. (1976) reported a 0.1 PFU/g concentration of echovirus type 7 from a full-scale sand drying bed facility for sludge treatment. Dewatering of sludge by evaporation inactivated human poliovirus, coxsackieviruses and reoviruses (Ward and Ashley, 1977).

Conclusions. According to Feachem et al. (1983), "any sludge treatment process that involves temperatures of 50°C or above should yield a virus-free product if the process is well controlled and carried out for sufficiently long to ensure that all parts of the mass are heated." However, results reported in a more recent review (Rao et al., 1986) suggest that higher temperatures may be necessary to ensure virus inactivation. They report that HAV was not inactivated after heating to 60°C for 12 hours and was infective at 80°C in the presence of high concentrations of some salts.

Other factors affect virus persistence in relation to temperature. Ward et al. (1976) report that an ionic detergent protected poliovirus from heat in raw sludge. Detergents from wastewater become associated with solids and are concentrated in sludges. Composting causes degradation of the detergents; poliovirus is more readily inactivated during composting, but the survival of reovirus is enhanced (Ward and Ashley, 1978).

Aqueous ammonia, which is formed during anaerobic digestion, added to raw sludge speeds inactivation, allowing moderate heat treatment to inactivate enteroviruses.

Adsorption of virus particles to sludge particles may protect them from heat inactivation and from inactivation by ammonia (Ward and Ashley, 1978).

After monitoring a human rotavirus, a coxsackievirus B5, and a bovine parvovirus during sludge treatment processes, Spillman et al. (1987) conclude that the best treatment to eliminate viruses from sludge would be thermal (60°C) treatment to inactivate thermolabile viruses, followed by anaerobic mesophilic digestion to eliminate thermostable viruses that are sensitive to chemicals and microbes.

3.3. OCCURRENCE OF VIRUSES IN NATURAL MEDIA

3.3.1. Persistence in Soil. Persistence of viruses in the subsurface is dependent upon the type of virus, the nature of the soil, and the climate of the environment (Yates and Yates, 1988). Although specific factors that control the fate of viruses in soil have been identified, interactions between factors make consideration of separate effects difficult. Factors known to affect the fate of viruses in the environment include: temperature, microbial activity, moisture content, pH, salt species and concentration, soil properties, virus association with soil, virus aggregation, virus type and organic matter. In many cases, the mechanisms by which these factors influence viral inactivation or protection are not clear. Table 3-4 summarizes data on virus persistence in soils.

Temperature. Temperature is probably the most important factor influencing persistence and inactivation of viruses in the environment (Yates and Yates, 1988; Gerba and Bitton, 1984; Bitton, 1978). Most enteric viruses are inactivated at temperatures of 60°C or above (Morris and Darlow, 1971); however, some types, such as hepatitis A, have been shown to withstand higher temperatures (U.S. EPA, 1988). Hurst et al. (1980a) observed that in vials of soil the inactivation rate for poliovirus increased as temperature increased, and Lefler and Kott (1974) made a similar observation with poliovirus in a sandy soil in Israel. This relationship of viruses to soil temperatures has been confirmed by other studies (Gerba and Bitton, 1984). Viruses persisted up to 170 days in waste-treated soil at 3-10°C, according to Bagdasar'yan (1964). After irrigation of crops with sewage effluent, the time for 99% inactivation of viruses in soil was 2 months during winter but 2-3 days in summer (Larkin et al., 1976a; Tierney et al., 1977).

TABLE 3-4

Virus Inactivation in Soil

Virus	Inactivation Rate in Days T_{90} T_{99}	Inactivation Rate Constant (log_{10} day^{-1})	Sludge Treatment/ Application	Conditions	Reference
Poliovirus type 1			Inoculated viruses applied to vials of soil in:	Sandy loam soil, pH 7.8	Hurst, 1988b
	455	0.0022 (0.000092/hr)	Aerobic: Sterile media	1°C	
	152	0.0066 (0.00027/hr)	Nonsterile media		
	588	0.0017 (0.000071/hr)	Anaerobic: Sterile media	1°C	
	323	0.0031 (0.00013/hr)	Nonsterile media		
	31	0.0323 (0.00135/hr)	Aerobic: Sterile media	23°C	
	10	0.1035 (0.0043/hr)	Nonsterile media		
	30	0.0338 (0.00141/hr)	Anaerobic: Sterile media	23°C	
	33	0.0304 (0.00126/hr)	Nonsterile media		
	3.0	0.3331 (0.0139/hr)	Aerobic: Sterile media	37°C	
	1.4	0.7077 (0.0294/hr)	Nonsterile media		
	1.7	0.5809 (0.0242/hr)	Anaerobic: Sterile media	37°C	
	3.5	0.2884 (0.0120/hr)	Nonsterile media		

TABLE 3-4 (continued)

Virus	Inactivation Rate in Days T_{90} T_{99}	Inactivation Rate Constant (log_{10} day^{-1})	Sludge Treatment/ Application	Conditions	Reference
Poliovirus	Not given	0.10 (0.0042/hr) 0.09 (0.0038/hr)	Soil flooded with inoculated secondary effluent Soil treated with sewage sludge	Not given	Larkin et al., 1976a
Poliovirus	Not given	0.04 (0.0017/hr) 0.16 (0.0068/hr)	In forest soil	4°C 20°C	Duboise et al., 1974
Viruses	Not given	1.45 avg. (0.060/hr) {0.04 min. (0.0017/hr), 3.69 max. (0.154/hr)}	Soil/water/plant system	Not given	Reddy et al., 1981
Viruses	Not given	0.26 (0.0108/hr) 0.2171 (0.0091/hr) 0.057 (0.0024/hr) 0.2796 (0.0116/hr) 0.328 (0.0137/hr)	Aerobically digested sludge, indigenous viruses	Late summer (20-31°C daily temp) No rain Moderate rain	Hurst et al., 1978

Inactivation at higher temperatures may result from protein denaturation of the viral capsid (Yates and Yates, 1988). Dimmock (1967) suggests that at temperatures >44°C, viral inactivation is associated with structural changes in the viral capsid; but at temperatures <44°C, inactivation depends on the inactivation rate of viral nucleic acid. Temperature also has an indirect effect on virus persistence due to its effect on the growth of aerobic bacteria (Lance and Gerba, 1982).

Soil Moisture Content. According to Bitton et al. (1987) and Gerba and Bitton (1984), temperature and soil desiccation synergistically influence the fate of viruses in the soil environment. In their review of virus survival in nature, Bitton et al. (1987) report that temperature and moisture are the primary controls of viral persistence in sludge-amended soil, and Bitton et al. (1981) report that soil drying is a major detrimental factor in viral persistence in sludge-soil mixtures. During development of methods for the detection of enteroviruses in sludges, Hurst et al. (1978) applied aerobically digested sludges to land and subsequently found reductions of naturally occurring enteroviruses at a rate of 2 $\log_{10}$/week. After 3 months in the field, no viruses were detected in sludge solids. The authors suggest that virus inactivation in the sludge solids was directly related to desiccation.

When columns of sand treated with sludge were exposed to warm, dry fall weather (<0.13 cm cumulative rainfall), infectivity of seeded echovirus and poliovirus declined more rapidly than when the columns were exposed to wet summer weather (>13 cm cumulative rainfall) (Bitton et al., 1981, 1984). Bagdasar'yan (1964) reports that enteroviruses, including poliovirus 1, coxsackievirus B3, and echoviruses 7 and 9 persisted 2-3 months in soil with 10% moisture compared with 15-25 days in air-dried soils. Poliovirus type 1 inactivation was much more rapid in drying soil (1 week for 99% inactivation as moisture decreased from 13% to 0.6%) than in soils maintained at higher moisture levels (7-8 and 10-11 weeks for 99% inactivation at levels of 25 and 15%, respectively) (Sagik et al., 1978). During rapid infiltration of wastewater in a field study, Hurst et al. (1980b) found that viral inactivation rates were higher in rapidly drying soil. Periodic drying followed by aeration was found to enhance viral inactivation.

Comparing the inactivation rate of poliovirus in eight soils saturated with river water, groundwater, or septic wastewater with the same soils allowed to dry out, Yeager and

O'Brien (1979a) noted a sharp increase in inactivation rate at 1.2% soil moisture compared with the rate at 2.9%. Hurst et al. (1980a) found that viral survival did not correlate linearly with soil moisture, but decreased with increasing moisture up to the saturation point, then increased with moisture beyond that point. Possible explanations for this enhanced survival at both high and low soil moisture levels include differences in extent of viral adsorption to soil, in mechanisms of adsorption, and in microbial growth rates at various soil moisture levels.

Yeager and O'Brien (1979a) used radiolabeled viruses to show that viruses did not become irreversibly bound to soil particles but were inactivated during the drying process. Yeager and O'Brien (1979b) suggest that the mechanisms for viral inactivation in moist soils are different from the mechanisms in drying soils.

Microbial Activity. Microbial activity may play a role in the inactivation of viruses in soil. Although some studies (Gerba and Bitton, 1984) have not observed any difference in viral decline in sterile and nonsterile soils, others noted greater inactivation in nonsterile soil. The inactivation rates of poliovirus and reovirus were found to be greater in nonsterile soil suspensions than in sterile soil suspensions (Sobsey et al., 1980). Comparing four different combinations of aerobic/anaerobic and sterile/nonsterile conditions on poliovirus type 1, Hurst (1988b) observed that aerobic microorganisms exerted a statistically significant effect on the rate of viral inactivation in sandy loam soil at three incubation temperatures (1, 23, and 37°C). He concludes that microbial antagonism appears to be a major determinant of viral stability in soil and suggests that the antagonism results from metabolic products released from bacteria or from interference with adsorption onto soil particles. Mechanisms of virus inactivation by microbes in soil have not been clarified at this point.

Sobsey et al. (1986) report a temperature effect on the antiviral activity of microbes. Sterile and nonsterile soil samples gave similar survival rates for HAV, poliovirus type 1, and echovirus type 1 at 5°C, but at 25°C the time for inactivation of all three viruses was shorter in the nonsterile samples.

pH. Bitton (1978) asserts that, in general, enteric viruses will not be affected by the pH values of the natural environment. The direct effects of pH on viral persistence in soil have not been studied extensively. Reported results indicate that pH can influence virus

inactivation, but the extent of this influence is not clear (Sobsey and Shields, 1987). Hurst et al. (1980a) observed that poliovirus 1 and bacteriophages MS-2 and T2 persisted longer at lower soil saturation pH values. The results of Salo and Cliver (1976) with aqueous solutions indicate that inactivation by pH varies with the type of virus. Various mechanisms have been suggested for the direct effects of pH on virus persistence, including alterations in the viral capsid and increase in sensitivity of the nucleic acids to DNase or ribonuclease (Yates and Yates, 1988).

In addition, pH may have indirect effects on viral persistence by affecting adsorption. Above pH 7, the net charge on the virus particle is negative; and sand, clay minerals, and organic matter are also negatively charged at pH>7 (Gerba and Bitton, 1984). Although adsorption would appear to be at a minimum at alkaline pH values, conflicting reports in the literature indicate that this relationship is not clear-cut (Gerba and Bitton, 1984).

Salt Species and Concentration. Inactivation of enteroviruses in the environment can be influenced by salt species and their concentrations. Several investigators have reported that poliovirus, echoviruses, and bacteriophages were less susceptible to thermal inactivation in the presence of certain cations (e.g., Ca and Mg) in the media (Yates and Yates, 1988). According to Gerba and Bitton (1984), this phenomenon may be significant to viral persistence in soil. Several studies have indicated that the type and concentration of salts in the soil affect virus adsorption to soil, adsorption increasing with increasing ionic strength (Yates and Yates, 1988).

Organic Matter. Organic matter may have a protective effect on the persistence of viruses in soil. Although Hurst et al. (1980a) did not find that virus persistence was significantly related to the amount of soil organic matter, Lefler and Kott (1974) found that poliovirus persisted longer in sand watered with waste pond effluent than with distilled water.

Organic matter has been found to decrease virus adsorption by competing for sites on soil particles (Yates and Yates, 1988). Moore et al. (1981) and Bitton et al. (1976) report that organic matter interfered with viral adsorption in soil, indirectly affecting viral persistence; and other investigators report that organic material acts as an eluting agent, desorbing viruses from the soil (Gerba, 1984a). Humic and fulvic acids have also been

reported to prevent adsorption and to cause loss of virus infectivity (Yates and Yates, 1988). The effects of organic matter on soil properties such as pH, moisture content, and ion exchange capacity may indirectly influence the persistence of enteric viruses in soils (Sobsey and Shields, 1987).

Adsorption. Depending upon the sorbent, the survival of viruses may be enhanced or reduced by adsorption to soils or other materials (Yates and Yates, 1988). Significant, rapid inactivation of poliovirus type 1 occurred in soil after its adsorption to manganese, aluminum, and copper oxide particles but not after adsorption to silica and iron oxide, according to Murray and Laband (1979). Moore et al. (1982) report that reovirus adsorption to organic muck, montmorillonite, dolomite, and Ottawa sand resulted in considerable inactivation. Sobsey et al. (1980) found that survival was not prolonged in every case when poliovirus type 1 and reovirus type 3 adsorbed to eight different soil materials.

Recent studies indicate that adsorption of virus particles by the soil is a major factor in virus persistence (Gerba, 1985). In their experiments, Hurst et al. (1980a) found that soil adsorption was one of the most important of the factors that significantly affected viral survival, with survival increasing with greater adsorption. The enhanced survival of viruses in soil with a high adsorption capacity presents a dilemma for land application of wastes. Although adsorption of viruses prevents their movement to the groundwater, soils with this capacity are likely to enhance virus survival (Hurst et al., 1980a).

Gerba and Schaiberger (1975) suggest several mechanisms by which adsorption of virus particles to various solids may enhance or reduce their survival. Among these are interference with the action of virucides, increased stability of the protein capsid, prevention of aggregate formation, and adsorption of enzymes and other inactivating substances.

Formation of Aggregates. Although there are no studies on the relationship of aggregate formation to viral persistence in soil, the fact that aggregates affect virus persistence in water suggests that aggregates might protect viral particles from environmental factors in the soil (Yates and Yates, 1988; Sobsey and Shields, 1987).

Soil Characteristics. Soil properties probably influence viral persistence by affecting the degree of adsorption of virions to soil particles (Yates and Yates, 1988). According to

Bitton et al. (1987), the relationship of soil type to virus persistence is difficult to determine because of the influence of other environmental factors. Mineral and organic content of soil, moisture level, pH, and cation exchange capacity (CEC) affect viral persistence in soil and are related to soil type.

Hurst et al. (1980a) found that virus survival was significantly correlated to adsorption and to soil saturation pH but not to other soil characteristics. However, stepwise multiple regression analysis of virus survival and 19 soil characteristics revealed that extractable phosphorus and exchangeable aluminum were the next highest ranking of soil characteristics affecting survival. Hurst et al. (1980a) suggest that the relationship between survival and aluminum is due to the increase in virus adsorption at high aluminum levels, and the increase in virus survival with decrease in level of resin-extractable phosphorus is due to increased adsorption at lower levels of resin-extractable phosphorus.

Fine-textured soils that contain clay remove more viruses by adsorption than coarse-textured soils (Yates and Yates, 1988; Gerba and Bitton, 1984). Gerba and Bitton (1984) observe that humic materials and clay minerals are the two most active components of the soil. Clays increase viral adsorption to soil as well as influencing survival of microbial populations in the soil; survival of microbes could be influenced by water that may be tightly bound to clay. Also, clay can be protective to the viral genome.

The soil CEC may indirectly affect persistence of viruses by influencing adsorption. The work of Sobsey (1983) and others indicates that virus adsorption increases with increasing CEC, as well as with clay content, exchangeable aluminum and low flow rate.

In examining the retention of poliovirus by 34 soils and minerals, Moore et al. (1981) found that adsorption was negatively correlated with soil organic matter and with available negative surface charge. Adsorption was not significantly correlated with soil pH, surface area or elemental composition. Their results indicate that soils are potentially efficient at binding viruses since 10^6 viruses adsorbed to a gram of Ottawa sand with only 1% of the surface covered. Sobsey and Shields (1987) report that minerals were better adsorbents than soils in some studies. According to Gerba and Bitton (1984), soil iron oxides have been shown to increase the retention of viruses in soil. Magnetite sand adsorbed 99.99% of virus particles while muck soil retained 16-79%.

Van der Waals forces, double-layer interactions, and hydrophobic interaction are discussed by Gerba (1984a) as mechanisms for viral adsorption to soil particles.

Type of Virus. Susceptibility to inactivation in soil may vary with the type of virus and the particular strain. According to Lefler and Kott (1974), bacteriophage f2 survived longer than poliovirus in saturated and in dry sand. Hurst et al. (1980a) found different inactivation rates for seven viruses under the same conditions. The inactivation rates of poliovirus, reovirus, echovirus and HAV in several types of soil material at 25°C were quite different, although all three survived well in soil suspensions at 5°C (Sobsey et al., 1986). Goyal and Gerba (1979), studying a number of viruses including human enteroviruses, found strain and type differences in adsorption to soil.

3.3.2. Persistence in Water. Viruses persist longer in groundwater than in surface waters; Keswick et al. (1982) attributes this to lower temperatures, protection from sunlight, and lack of microbial antagonism in groundwater. For poliovirus type 1, the decay rate reported by Bitton et al. (1983a) was 0.0019 hr^{-1} in groundwater, and O'Brien and Newman (1977) report a decay rate of 0.031 hr^{-1} in river water. A field study by Wellings et al. (1975) suggests that viruses persist for up to 28 days in groundwater, and Sattar (1981) reports persistence of 560 days in surface water in the laboratory. In natural waters, several interacting factors, including temperature, chemicals, pH, light, biologic factors and suspended particulate matter, affect virus persistence (Melnick and Gerba, 1980).

Table 3-5 summarizes data on virus inactivation in water. Virus inactivation follows apparent first-order kinetics (O'Brien and Newman, 1977). Virus inactivation curves in aquatic systems range from linear to S-shaped, with variations being a result of clumping of the viruses (Berg et al., 1967). O'Brien and Newman (1977) attributed S-shaped curves to higher initial virus titers ($> 10^6$ PFU/mL) and noted that linear inactivation curves (straight lines on semilog plots) were associated with initial virus concentrations of 10^5 PFU/mL or less.

Temperature. The length of time that viruses persist and remain infective in surface waters depends to a great extent upon temperature, with lower temperatures enhancing survival and infectivity (Yates and Yates, 1988). In river water, enteroviruses persisted 5-20 days at 20°C in the laboratory (Clarke et al., 1964), and in farm pond water, enteroviruses

TABLE 3-5

Virus Inactivation in Aquatic Systems

System Description	Organism	pH	Season or Temperature	Length of Study	k (hr^{-1})	Inactivation Rate (log_{10} $days^{-1}$)
Organisms inoculated in lab into groundwater from 475-ft deep well (Bitton et al., 1983b)	Poliovirus 1	7.6	22°C		0.0019	0.046
Organisms inoculated in lab into McFeters' type survival chambers with groundwater from 275-ft deep well (Keswick et al., 1982)	Poliovirus 1	7.8	3-15°C		0.0088	0.21
McFeters type survival chambers *in situ* in river (O'Brien and Newman, 1977)	Poliovirus 1		23-27°C 12-20°C 7-17°C 4-8°C	1 year	0.031 0.040 0.032 0.028 0.022	0.77 T_{90} 25 hr T_{90} 31 hr T_{90} 36 hr T_{90} 46 hr
	Poliovirus 3		23-27°C 12-20°C		0.053 0.042	T_{90} 19 hr T_{90} 24 hr
	Coxsackievirus A-13		23-27°C 12-20°C		0.14 0.083	T_{90} 7 hr T_{90} 12 hr
	Coxsackievirus B1		12-20°C 7-17°C 4-8°C		0.034 0.023 0.017	T_{90} 29 hr T_{90} 44 hr T_{90} 58 hr

TABLE 3-5 (continued)

System Description	Organism	pH	Season or Temperature	Length of Study	k (hr^{-1})	Inactivation Rate (log_{10} $days^{-1}$)
Organisms inoculated into groundwater in lab (Yates et al., 1985)	Poliovirus 1					
	Wisconsin		12°		0.0025	0.060
	Arizona		23°C		0.015	0.357
	North Carolina 1		12°C		0.0057	0.138
	North Carolina 2		12°C		0.0047	0.114
	Univ. of Arizona		23°C		0.028	0.676
	New York 1		12°C		0.0015	0.035
	New York 2		12°C		0.0021	0.051
	Texas 1		13°C		0.0015	0.036
	Texas 2		13°C		0.0057	0.137
	California 1		18°C		0.0077	0.185
	California 2		17°C		0.0034	0.081
Organisms inoculated into groundwater in lab (Yates et al., 1985)	Echovirus 1					
	Wisconsin		12°C		0.0028	0.066
	Arizona		23°C		0.0078	0.188
	North Carolina 1		12°C		0.0077	0.186
	North Carolina 2		12°C		0.0072	0.174
	Univ. of Arizona		23°C		0.0026	0.628
	New York 1		12°C		0.0022	0.054
	New York 2		12°C		0.0021	0.051
	Texas 1		13°C		0.0057	0.138
	Texas 2		13°C		0.0033	0.079
	California 1		18°C		0.0063	0.151
	California 2		17°C		0.0038	0.091
Organisms inoculated in lab into McFeters type survival chambers with groundwater from 275-ft deep well (Keswick et al., 1982)	Coxsackievirus	7.8	3-15°C		0.0079	0.19

TABLE 3-5 (continued)

System Description	Organism	pH	Season or Temperature	Length of Study	k (hr^{-1})	Inactivation Rate (log_{10} $days^{-1}$)
Organisms inoculated into McFeters type survival chambers with groundwater from 275-ft deep well (Keswick et al., 1982)	Rotavirus SA11	7.8	3-15°C		0.015	0.36
Summary data from review sources (Kutz and Gerba, 1988)	Enteric viruses and coliphage					
polluted river			5-28°C		0.013	0.325
unpolluted river			4-37°C		0.010	0.250
impounded			5-22.5°C		0.016	0.374
groundwater			4-30.5°C		0.007	0.174
Surface freshwater from 5 sites (Hurst et al., 1989)	Coxsackie-virus B3		-20°C	12 wk	0.00016	0.0039
			1°C	12 wk	0.0023	0.0475
			22°C	8 wk	0.0102	0.2455
	Echovirus 7		-20°C	12 wk	0.00016	0.0039
			1°C	12 wk	0.0023	0.0544
			22°C	8 wk	0.0062	0.1498
	Poliovirus 1		-20°C	12 wk	0.00031	0.0075
			1°C	12 wk	0.0021	0.0498
			22°C	8 wk	0.0093	0.2232
Natural surface waters (Haas, 1986)	Viruses		3-5°C		0.0032-0.0071	0.077-0.17
			22-25°C		0.032-0.12	0.76-2.8
			37°C		0.058	1.4

TABLE 3-5 (continued)

System Description	Organism	pH	Season or Temperature	Length of Study	k (hr^{-1})	Inactivation Rate (log_{10} $days^{-1}$)
(McDaniels et al., 1983)	Calf Rotavirus					
Distilled Water	CPE assay		8°C		0.00057	T_{90} 73 days
	IFA assay				0.00022	T_{90} 185 days
	CPE assay		26°C		0.0055	T_{90} 7.6 days
	IFA assay				0.0050	T_{90} 8.4 days
Secondary Wastewater	CPE assay		8°C		0.00050	T_{90} 84 days
	IFA assay				0.00018	T_{90} 236 days
	CPE assay		26°C		0.0060	T_{90} 7.0 days
	IFA assay				0.0053	T_{90} 7.8 days
(McDaniels et al., 1983)	Calf Reovirus					
Distilled Water	CPE assay		8°C		0.00032	T_{90} 130 days
	IFA assay				0.00027	T_{90} 154 days
	CPE assay		26°C		0.0061	T_{90} 6.8 days
	IFA assay				0.0063	T_{90} 6.6 days
Secondary Wastewater	CPE assay		8°C		0.00016	T_{90} 262 days
	IFA assay				0.000066	T_{90} 630 days
	CPE assay		26°C		0.0031	T_{90} 13.3 days
	IFA assay				0.0049	T_{90} 8.5 days
River (Haas, 1986)	Viruses		Moderate	20-70 hr	0.014-0.050	1-3
River water, in the presence or absence of sunlight with low or high turbidity (Hurst, 1988a based on data in Cubbage et al., 1979)	Poliovirus					
	sunlight					
	low turbid.				0.099	2.383
	hi turbid.				0.056	1.334
	no sunlight					
	low turbid.				0.034	0.808
	hi turbid.				0.030	0.710

persisted 84 days at 20°C and 91 days at 4°C (Joyce and Weiser, 1967). O'Brien and Newman (1977) found that the rates of inactivation of polioviruses 1 and 3 and coxsackieviruses A-13 and B-1 in membrane dialysis chambers in the Rio Grande River were affected principally by the water temperature.

In laboratory experiments, human rotavirus persisted longer in river water at 4°C than at 20°C (Raphael et al., 1985). Since virus concentrations in filtered river water were essentially the same irrespective of temperature, the effect of the higher temperature may be indirect, with higher temperature promoting growth of bacteria and other microorganisms with antiviral activity.

Groundwater samples from 11 locations were analyzed for various chemical and physical factors; inoculated with poliovirus 1, echovirus 1, and MS-2 coliphage; held at the *in situ* temperature; and examined at intervals for virus persistence (Yates et al., 1985). Temperature was significantly correlated with the inactivation rates of the three viruses.

In their study of HAV in soils, groundwater and wastewater, Sobsey et al. (1986) report that at 5°C, HAV, poliovirus type 1 and echovirus type 1 persisted (<90% inactivation) for at least 12 weeks in groundwater. However, in 12 weeks at 25°C, HAV was somewhat less affected than the other two viruses, with 90-99% inactivation compared with 99.9% inactivation of poliovirus and echovirus.

Hurst et al. (1989), analyzing viral inactivation rates, found that the statistically significant factors affecting persistence of three human enterovirus serotypes (coxsackievirus B3, echovirus 7 and poliovirus 1) were incubation temperature, viral serotype and water source. T_{90}, the number of days required for 90% inactivation or a 1-log reduction, varied with temperature for the three viruses studied (Hurst et al., 1989):

	-20°C	1°C	22°C
Coxsackievirus B3	255 days	21 days	4 days
Echovirus 7	196 days	18 days	6.7 days
Poliovirus 1	133 days	20 days	4.5 days

Through its effect on the chemical and biologic reactions in natural waters, temperature may have an indirect effect on virus persistence that is reflected in seasonable

variations (Niemi, 1976). Kapuscinski and Mitchell (1980) also suggest that temperature may not directly affect virus inactivation but may control other inactivation mechanisms.

The inactivation rates for viruses in aquatic systems (Table 3-5) were examined to determine whether they could be used to generate a general temperature-dependence equation like those used for die-off of bacteria and parasites in soil and water and for inactivation of viruses in soil. These data are presented in Figure 3-1, which demonstrates a relationship between temperature and inactivation rate for all three viruses examined, as well as a difference between the viruses in inactivation rates as a function of temperature. The figure also shows the extensive scatter observed among the different studies and different viruses. This scatter makes it difficult to predict viral inactivation rates with a high degree of confidence.

Water Characteristics. Hurst et al. (1989) analyzed viral inactivation rates in relation to surface water characteristics. The average viral inactivation in five surface water samples from different sites (expressed in $\log_{10}$ units of viral loss/day of incubation) was 6.5-7.0 logs over 8 weeks at 22°C, 4-5 logs over 12 weeks at 1°C, and 0.4-0.8 log over 12 weeks at -20°C. Hardness and conductivity, strongly correlated with each other; turbidity and suspended solids content, strongly correlated with each other; and the number of generations of bacterial growth supported in the sample, also correlated with hardness and conductivity, were the apparent water characteristics affecting viral persistence (Hurst et al., 1989).

In their study of groundwater samples collected in the United States, however, Yates et al. (1985) found that water characteristics (pH, nitrate, ammonia, sulfate, iron, total dissolved solids (TDS), hardness, and turbidity) were not significantly correlated with inactivation of three viruses held at *in situ* temperatures. However, the decay rate of one virus, MS-2 coliphage, was significantly correlated to calcium concentration.

Jansons et al. (1989) examined the inactivation rates of enteroviruses (echoviruses 6, 11, and 24; coxsackievirus type B5; and poliovirus type 1) in dialysis bags lowered into the groundwater. Survival was variable and was influenced by temperature and dissolved oxygen concentrations, with the inactivation rate increasing as dissolved oxygen increased. They suggest that dissolved oxygen may indirectly affect enteroviruses by influencing the activity

INACTIVATION RATES OF VIRUSES IN WATER

AS A FUNCTION OF TEMPERATURE

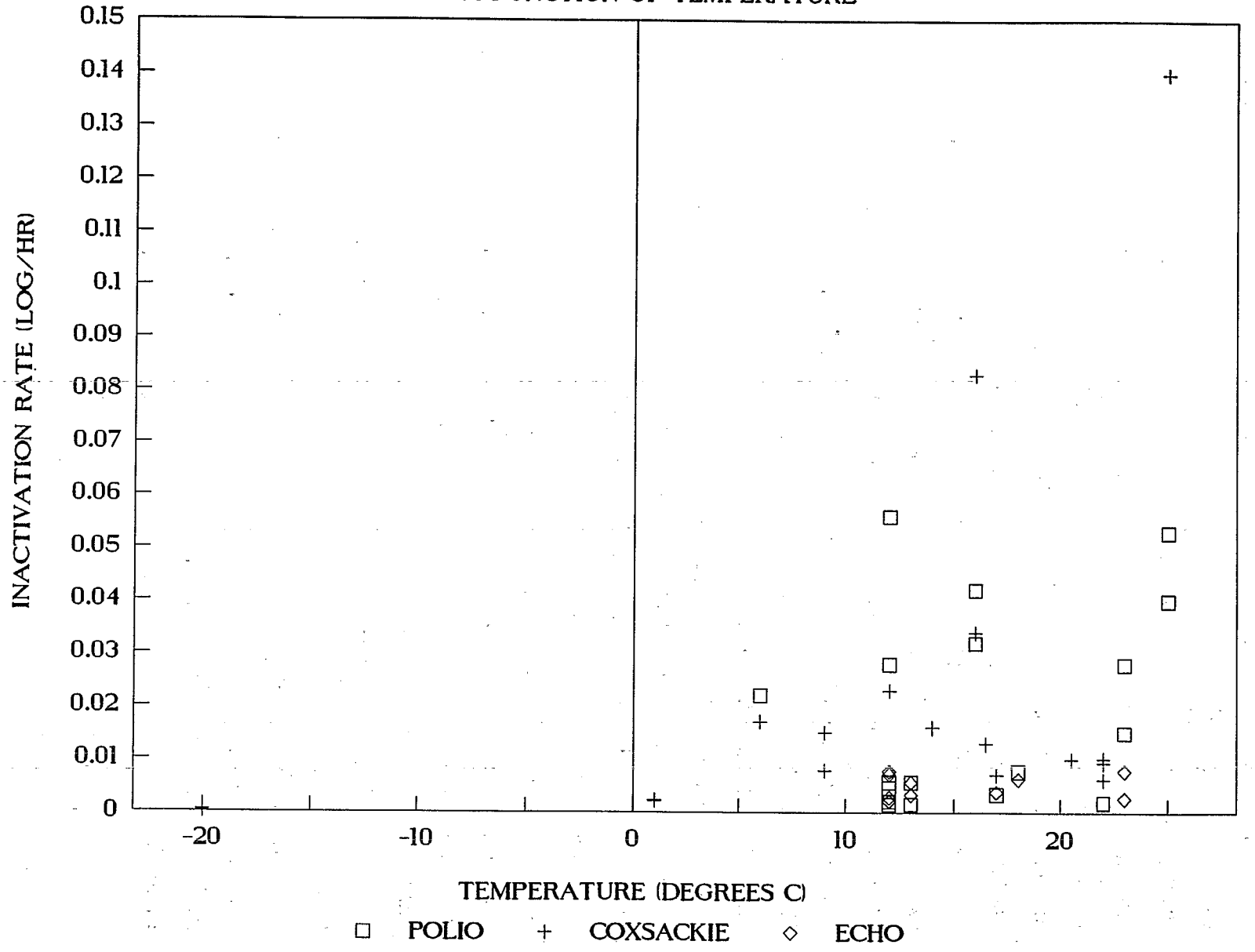

FIGURE 3-1

of antagonistic organisms or may have a direct effect by oxidation of virus capsid components.

Light. At wavelengths <370 nm, light has been shown to be detrimental to viruses in water (Bitton et al., 1987). Ultraviolet light inactivated virus particles, and visible sunlight inactivated poliovirus type 1 suspended in clean water; inactivation decreased with depth and addition of clay particles (Bitton et al., 1979a). Algae that may shade viruses from inactivation by light are controlled by light, indirectly affecting virus persistence (Bitton et al., 1979a); and naturally occurring chemicals such as humic and fulvic acids may photosensitize viruses in aquatic environments (Kapuscinski and Mitchell, 1980).

Hurst (1988a) reviewed six studies examining enteric virus persistence in systems using natural surface fresh waters. Rotaviruses appeared to be more stable in fresh surface water than enteroviruses. High turbidity levels decreased the inactivation rate of poliovirus in river water, possibly by reducing the inactivating effects of UV radiation from sunlight (Cubbage et al., 1979). Inactivation rates associated with low turbidity in the presence of sunlight were 2.383 $\log_{10}$ units/day compared with 1.334 $\log_{10}$ units/day for high turbidity. When sunlight was absent, the inactivation rate was 0.808 logs/day with high turbidity (36 nephelometric turbidity units [NTU]) and 0.710 logs/day with low turbidity (<2.5 NTU).

Microbial Activity. Investigations on the antiviral activity of microorganisms in natural waters have yielded inconsistent results. According to the results of O'Brien and Newman (1977), raw river water and filter-sterilized river water had comparable virucidal activity, but virus inactivation rates were lower in autoclaved river water. They suggest that the water may contain a heat-labile or volatile inactivating factor. With lake water, Herrmann et al. (1974) observed more rapid enterovirus inactivation in untreated water in dialysis bags *in situ* than in filtered water under similar laboratory conditions, suggesting that a biologic factor was involved in inactivation.

In a study of >30 groundwater samples, Yates (1984) found that indigenous bacteria had an inconsistent effect on virus inactivation. In some cases, viruses survived longer in sterile samples, but with other samples, survival was greater in the nonsterile portion. Filtered and unfiltered groundwater samples from several states were analyzed by Yates et al. (1990). Temperature was the only water characteristic that was consistently, significantly

correlated to decay rates of coliphage MS-2 and poliovirus type 1, which had been inoculated into the samples. Although there was no consistent trend associated with the presence or absence of bacteria, differences in inactivation rates were observed among some water samples incubated at the same temperature. In another experiment in which bacteria were enumerated throughout the experiment, the coliphage MS-2 inactivation rate in unfiltered samples was significantly correlated to an increase in bacterial numbers in the first 24 hours, suggesting that the bacteria produced some substance that inactivated viruses. Yates et al. (1990) summarize suggested mechanisms by which organisms may affect inactivation: production of enzymes that destroy the virus protein coat, production of substances that increase virus susceptibility to photodynamic inactivation, and production of oxidizing and reducing agents.

Jansons et al. (1989), examining the inactivation rates of enteroviruses (echoviruses 6, 11, and 24; coxsackievirus type B5; and poliovirus type 1) in groundwater, found no association between rate of virus inactivation and bacterial numbers. However, they suggest that the presence of large numbers of specific microorganisms in some bores may have contributed to the more rapid inactivation in those bores.

pH. In general, viruses survive well at the pH level of natural waters (pH 5-9) (Bitton et al., 1987). The pH of the water affects virus adsorption to surfaces, virus sensitivity to enzymes, and their heat stability (Bitton et al., 1987). According to Salo and Cliver (1976), virus persistence relative to pH in the aqueous environment varies with the type of virus. Poliovirus 1 survived best at near-neutral values (5 and 7), but coxsackievirus A9 was inactivated more rapidly at pH 5 than at higher and lower values. Pancorbo et al. (1987) found that for human rotavirus type 2 (strain Wa), inactivation was significantly correlated with the water pH; inactivation increased with increasing pH.

Salt Species and Concentration. Various salts affect the inactivation rates of viruses in aqueous solutions. With increasing concentrations of NaCl in solutions, inactivation rates of poliovirus type 1 increased (Salo and Cliver, 1976). Specific ion effects are indicated since different inorganic salts cause different inactivation rates under conditions of equal ionic strength. However, Cords et al. (1975) found that low ionic strength solutions

inactivated type A coxsackieviruses more rapidly than high ionic strength solutions. This did not hold true for group B coxsackieviruses or poliovirus type 1.

Studies have shown an enhanced thermal stabilization of enteroviruses in the presence of high concentrations of some salts (Yates and Yates, 1988). Burnet and McKie (1930) observed that bacteriophage inactivation at 60°C was partially prevented with low concentrations of $CaCl_2$ and $BaCl_2$, but thermal inactivation was increased with a higher concentration.

In their analysis of groundwater samples from locations within the United States, Yates et al. (1985) found that the decay rate for MS-2 coliphage was significantly correlated with the concentration of calcium in the 11 samples examined; decay rate increased with increasing calcium concentrations. This was not true for poliovirus 1 and echovirus 1. However, Yates (1984) found that calcium concentration was not significantly correlated with MS-2 inactivation rate when concentrations of calcium in the same water sample were varied. Yates et al. (1985) suggest that, in their experiment, some property of the water was correlated with calcium concentration and was involved in the observed inactivation rate.

Formation of Aggregates. According to Bitton et al. (1980), virus persistence in natural water is affected by the formation of aggregates. The formation of aggregates in water has been found by Young and Sharp (1977) to influence the inactivation of viruses by chemicals such as bromine. Bitton et al. (1980) suggest that virus particles within the aggregate are more protected from environmental factors.

In a discussion of experiments on the inactivation rates of human rotavirus type 2 (strain Wa) and poliovirus type 1, Pancorbo et al. (1987) observed that inactivation rates measured with single virus particles may not reflect the inactivation rates of indigenous viruses, which are often aggregated in water. They review several studies in which aggregated viruses (human and simian rotaviruses and polioviruses) were more resistant to chlorine inactivation than those in single-particle suspensions.

Adsorption. The association of viruses with organic and inorganic particles in water may enhance survival; survival is enhanced when these solids-associated viruses settle into the sediments (Bitton et al., 1987). Gerba and Bitton (1984) report that in aquatic

environments, clays have been shown to protect viruses from light, heat, and biologic degradation.

By monitoring pollution of two rivers in Germany, Walter et al. (1989) determined that, in general, virus levels in surface water depend upon virus input from domestic sewage, dilution by dispersion and typical metabolic interactions that have not been fully elucidated. The authors suggest that the industrial wastes in one river may have had a toxic effect on the virus capsid and that the oxygen content may have stimulated growth of a virus inactivating microflora. They suggest that virus adsorption to solid particles was promoted in the second river and that adsorption protected viruses against other inactivating mechanisms.

Virus Type. Yates et al. (1985) found no overall significant differences in survival of poliovirus, echovirus and coliphage MS-2 in different groundwater samples, but there were differences in individual samples. HAV has been shown to persist longer than polio and echovirus at 25°C in groundwater, wastewater, and soil suspensions, although all three did well at 5°C (Sobsey et al., 1986). Human enteric virus in survival chambers exposed to a flow of groundwater from a 275-foot deep well persisted for >24 days (Keswick et al., 1982); coxsackievirus B3 and poliovirus type 1 persisted longer than rotavirus SA-11.

Pancorbo et al. (1987) found that inactivation rates for human rotavirus type 2 (strain Wa) were significantly different from that of poliovirus type 1 (strain CHAT) when seeded into vials of mountain lake water, groundwater, wastewater effluent, or polluted creek water. Both viruses persisted longest in the lake water. Human rotavirus persisted longer than poliovirus in the polluted samples (creek water and wastewater effluent), but poliovirus persisted longer in the unpolluted waters (groundwater and lake water).

Hydrostatic Pressure. Although elevated hydrostatic pressures affect microbial persistence in seawater, Bitton et al. (1983a) found that pressures ranging from 500-4000 psi did not inactivate poliovirus in groundwater. According to Bitton et al. (1987), hyperbaric pressure is not expected to have a significant impact on virus persistence in groundwater.

Sediments. In their summary of data on the isolation of viruses from estuarine and freshwater sediments, Rao and Bitton (1987) observed that there are few studies on virus detection in freshwater sediments. Representatives of most groups of enteric viruses were

isolated from the sediments examined. Viruses adsorb readily to solids in water; in the laboratory, virus persistence has been shown to be prolonged by association with solids (Rao et al., 1986).

3.3.3. Persistence in Aerosols. The particle size range for aerosols is ~0.01-50 μm (10-50,000 nm), and infection by inhalation of these biologic particles depends on the depth of respiratory penetration, which is greatest for particles in the 1-2 μm range or for those below 0.25 μm (Sorber et al., 1979).

The potential for human exposure and associated health risk from viruses in aerosols is derived from the concentration of pathogens in the wastewater or liquid sludge and the aerosolization efficiency of the spray irrigation; the effect of the impact factor or aerosol shock on the viruses within the first fraction of a second as they become aerosolized; and the inactivation rate or decay rate, which, like aerosol shock, is influenced by meteorologic factors and whether the viruses are released by day or night (Camann et al., 1978; Sorber et al., 1979). The potential exists for adverse human health effects from wastewater or sludge aerosols because viruses are at the low end of the size range for aerosol particles and can be inhaled and subsequently ingested and because the infective dose can be as small as one virus particle or PFU.

Sorber et al. (1984) studied microbiologic aerosols sampled near liquid sludge spray application sites. Liquid sludge was applied by tank trucks or by high-volume spray guns. No enteric viruses were detected in pooled samples representing 1470 m^3 of air sampled. Converted to <0.0016 PFU/m^3 of air, this value implies that virus aerosolization from land application of liquid sludge was not a significant problem.

Spray irrigation of wastewater, however, did produce levels of enteroviruses in ambient air at 50 m downwind of the spray site (Johnson et al., 1978). A geometric mean concentration of 0.076 of poliovirus/ml wastewater resulted in a geometric mean aerosol concentration of 0.002/m^3 of air. For other enteroviruses, the geometric mean concentration of 0.12/ml wastewater resulted in a geometric mean aerosol concentration of 0.014/m^3 of air. The data indicate that the viruses detected were very hardy. The authors suggest that because of the extraordinary methods required to monitor viruses in air near the spray site, a more feasible approach would be to measure the concentration of viruses in the

wastewater and use a predictive model to estimate virus concentrations in air. Such a model was developed by Camann et al. (1978) to predict pathogen concentrations in sprayed wastewater aerosols downwind from the spray site. Use of the model predicted a nighttime enterovirus concentration of 0.01 PFU/m^3 ~650 m from the spray site, a value similar to the measured enterovirus aerosol concentrations of 0.011 PFU/m^3 and 0.017 PFU/m^3 at 50 m downwind from the same spray site described in Johnson et al. (1978). There were too few aerosol runs and virus concentrations were measured only at 50 m downwind, so it was not possible to generate age decay rate estimates for enteroviruses. The authors noted, however, that the enteroviruses appeared to have impact factor values in the median range even higher than the hardy bacteria, i.e., the enteroviruses survive better during entry into the aerosolized state and during the initial travel in the aerosol (Camann et al., 1978). This feature is particularly significant since the indicator organisms that are often used to measure aerosols had much lower impact factors than the enteroviruses, implying that failure to detect bacteria or indicator organisms does not mean a human health risk from exposure to viruses in aerosols is diminished or absent.

Camann et al. (1980) monitored school attendance and its relationship to generation of wastewater aerosols from an adjacent wastewater treatment plant. No enteroviruses were recovered in the total air volume (1980 m^3) sampled 30 m downwind of the aeration basin, giving a calculated enterovirus aerosol concentration of <0.002 PFU/m^3. There was no adverse effect on communicable disease incidence in the school at this exposure level.

A more recent study by some of the same authors (Camann et al., 1988) investigated the possibility of adverse human health effects from spray irrigation of municipal wastewater in Lubbock, TX. Enteroviruses were recovered at a geometric mean level of 0.05 PFU/m^3 and a maximum level of 16 PFU/m^3 at 44-60 m downwind. These levels were higher than those observed at other wastewater aerosol sites in the United States (Fannin et al., 1985; Johnson et al., 1980a,b; Camann et al., 1980) and Israel (Katzenelson et al., 1976), characterizing the irrigation site as a source of infectious microbial aerosols.

Moore et al. (1988) evaluated the virus levels in irrigation wastewater in the Lubbock study. Prior to reservoir storage, the wastewater levels of viruses ranged from 100-1000 PFU/L. Following impoundment, viral levels were lowered to <10 PFU/L. Because of

variable sample concentration processes and virus enumerating systems, there were daily and seasonal variations in human enterovirus levels and serotypes.

Shuval et al. (1989) completed field investigations on the spread of enteric viruses by wastewater sprinkler irrigation at kibbutzim in Israel. Of 152 air samples taken at 30-730 m downwind of irrigation sites, 15 (~10%) were presumptively positive for enteric viruses. Even at the distant 730-m station, 3 were positive out of 24 samples taken. Samples were taken using High-Volume Cyclone Scrubbers (HVCS) for viable microbial aerosols. The fact that 31% of positive virus samples were negative for bacterial indicators suggests that aerosolized enteric viruses are better able to withstand hostile environmental conditions than are the indicators. In this study, the authors did not determine whether the very low virus concentrations (0.03-2 PFU/m^3) measured could lead to infection and disease in exposed human receptors.

Sorber et al. (1979) conclude that, although microorganisms can be aerosolized and transported at spray irrigation sites, the risk of public health impacts from use of treated and disinfected wastewater is probably minimal.

Ijaz et al. (1985a,b,c) have investigated survival characteristics of several viruses in aerosols. Table 3-6 summarizes the effects of temperature and relative humidity on persistence and illustrates the complexity of the factors determining persistence of viruses in aerosols. For example, when aerosolized viruses were tested at 20-24°C, human coronavirus persisted significantly longer at medium (50%) relative humidities (RH) than at high or low RHs (Ijaz et al., 1985a); but at the mid-range of 45-55% RH, there was a pronounced decline in infectivity of reovirus particles compared with high levels of infectivity at high and low RHs (Adams et al., 1982).

Generally speaking, lipid-containing viruses are more stable in aerosols and persist better at low humidity than lipid-free viruses. They are typically more contagious as aerosols and are more likely to be infective in winter when indoor relative humidity is <50%, unlike lipid-free viruses that survive better in moist air and infect more frequently in summer (de Jong et al., 1973). The authors caution, however, that these epidemiologic generalizations are based on insufficient data and, in fact, contradict the higher frequency of winter infections with rhinoviruses and adenoviruses.

TABLE 3-6

Virus Inactivation in Aerosols

Organism	Temperature (°C)	Relative Humidity (± 5%)	% Virus Survival	$t_{1/2}$ (hr)	Inactivation Rate[a] ($\times 10^{-6}$) (sec^{-1})	Reference
Human coronavirus 229E	20±1	30%		26.76±6.21	3.1	Ijaz et al., 1985a
		50%		67.33±8.24	1.2	
		80%		3.34±0.16	25.0	
	6±1[b]	30%		34.46±3.21	2.4	
		50%		102.53±9.3	0.8	
		80%		8	0.97	
				86.01±5.28		
Poliovirus type 1 (Sabin)	20±1	30%		NR[c]		Ijaz et al., 1985a
		50%		NR		
		80%		9.07±1.82	9.2	
Human rotavirus (subgroup 2, strain Wa)	20±1	30%		24.5±3.5	3.4	Ijaz et al., 1985c
		50%		44.2±6.3	1.9	
		80%		3.8±1.0	22	
	6±1	30%		21.6±4.0	3.9	
		50%		57.4±7.2	1.5	
		80%		1.71±0.7	48.9	
Calf rotavirus	20±1	30%	36.5±5.2			Ijaz et al., 1985b
		50%	70.0±1.5			
		80%	27.3±1.8			
Poliovirus type 1 (Sabin)	20±1	30%	0			Ijaz et al., 1985b
		50%	0			
		80%	100.7±9.0			
Reovirus particles (infectious, IV, and potentially infectious, PIV)	21-24	25-35%		avg. decay rate: 3.2%/min	6.67	Adams et al., 1982
		45-55%		2.85%/min		
		65-75%		3.25%/min		
		85-95%		2.0%/min		

[a]Calculated; standard deviation not included in values.
[b]Half-life values were predicted by regression analysis of the 24-hour survival results.
[c]NR, no virus recovered.

Adams et al. (1982) found reovirus particles to be relatively stable as aerosols. At high humidities (90-100%), reoviruses had <10-fold loss after 12 hours, but at lower relative humidities the aerosolized virus decayed more quickly. Table 3-6 illustrates the effect of RH on reovirus decay during equilibration; average decay rates range from a high of 3.2-3.25%/min at both 25-35% and 65-75% RH to 2.85% at 45-55% RH, but decay rates at 85-95% RH were much lower, averaging 2%/min. Overall decay rates averaged ~0.1%/min over a 12-hour period, with rates of 0.6%/min for infectious particles held at 45-55% relative humidity and 0.3%/min for potentially infectious particles held at 85-95% humidity over a 2.5-hour period.

Brandt et al. (1982) emphasize that one possible result of low humidity is that rotavirus-laden dust would be more likely to form from fecally contaminated clothes and bedding, tending to stay suspended in air and thus reaching susceptible individuals.

3.3.4. Persistence in Agricultural Products. Because of the concentration of viruses in sludges during treatment processes, land application of sludges may present a severe problem in contamination of fruits and vegetables. Adsorbed onto the sludge solids, viruses may be protected from thermal inactivation. Enteroviruses have persisted for 10-15 days on vegetables at refrigerator temperatures and up to 36 days on vegetables after spray irrigation (Hurst, 1989).

Parsons et al. (1975) have indicated that enteroviruses persist for 4-6 days on vegetables. Grigor'eva et al. (1965) reported enterovirus survival times of 4, 12 and 18 days on artificially contaminated cabbage, pepper and tomato plants, respectively. Survival times taken from Kowal (1985) suggest ranges of 4-23 days for aboveground crops such as tomatoes and lettuce and >60 days for below-ground crops like radishes. Konowalchuk and Speirs (1975a,b) studied virus persistence on vegetables and fruits stored at 4°C, determining that most were undetectable after 4-6 days, although viruses inoculated in feces persisted longer than those inoculated in water. The same authors (Konowalchuk and Speirs, 1977) reported a 99% reduction in poliovirus 1 and coxsackievirus B5 after 5 days on bunches of grapes hung indoors at 22°C. Feachem et al. (1983), summarizing a number of survival studies, conclude that practically complete elimination of viruses will occur in <5

days, with negligible survival of enteroviruses, on crops exposed to >2 weeks of temperatures above 25°C.

Larkin et al. (1976b) spray-irrigated lettuce and radishes with sewage sludge and effluent and found that although poliovirus persisted on the vegetables for 36 days, there was a 99% loss in detectable viruses within the first 5-6 days. On a second crop planted the following year, the virus persisted only 14 days. When the same authors (Tierney et al., 1977) investigated poliovirus survival under natural field conditions in plots flooded 1 inch deep with inoculated sewage, they determined that the longest survival (96 days) occurred in winter compared with 11-day survival in summer. Poliovirus was recovered from the mature vegetables 23 days after cessation of flooding, supporting the idea of a minimum 1-month waiting period following final sewage sludge or wastewater application before harvest (Kowal, 1985). Bagdasar'yan (1964) showed that enterovirus levels were reduced 90% in 10 days at 3-8°C and 99% in 10 days at 18-21°C.

Lasowski and Kott (1990) introduced poliovirus LSC 1, coxsackievirus A9, coxsackievirus B5 and echovirus 6 into secondary wastewater, some chlorinated and some not, then sprayed on parsley, kohlrabi, lettuce, onion leaves, tomatoes and grapevine leaves. With or without chlorination, poliovirus persisted longest on parsley leaves and consistently persisted longer than coxsackie strains. When applied at a concentration of 163,000 PFU/ml wastewater, >500 PFU poliovirus/cm^2 of surface adhered to the tomato. Natural (no chlorination) inactivation occurred in 3-7 days. Chlorination enhanced the inactivation process resulting in no viable enteric viruses within 1 day.

Ward and Irving (1987) spray-irrigated field-grown vegetables with stored wastewater, which was seeded with poliovirus or adenovirus at concentrations typically found in secondary effluent ($5.1x10^2$ - $2.6x10^5$ infectious units/L). Poliovirus was inactivated within 48 hours on field crops, but there was low-level persistence of the virus for ~13 days. Adenovirus was inactivated even more quickly, as early as 24 hours after irrigation. Poliovirus persisted for 76 days on celery and for 55 days on spinach that had been irrigated with wastewater and then refrigerated at 4°C.

A few studies have investigated the uptake of viruses by the root systems of plants (Kowal, 1985), but Katzenelson and Mills (1984) find little evidence that viruses penetrate the roots or stem.

Wallis et al. (1984) applied sludge to hayfields and pasture, but no enteric viruses were detected throughout the study. They caution that techniques for detection of viruses in sludge may be limiting.

The variability in environmental conditions, crops and methods of virus introduction (spraying, flooding, etc.) makes any reliable comparison of inactivation rates on crops difficult. Generally, the exposure of viruses on aboveground crops to desiccation, sunlight, high temperatures, or rainfall limits their persistence. Viruses on below-ground or on-ground crops will have inactivation rates more like those in soil, suggesting a safe waiting period might be close to 100 days (Kowal, 1985).

3.4. TRANSPORT

Transport of viruses in soil, water and air is influenced by many of the same factors that affect their persistence, discussed in Section 3.3 and reviewed by Yates (1990), Sattar and Ijaz (1987), Rao et al. (1986), Duboise et al. (1979), Keswick and Gerba (1980), Gerba and Bitton (1984), Goyal and Gerba (1979), Yates and Yates (1988) and others.

3.4.1. Transport in Soil. According to Sobsey and Shields (1987), small particles can be removed or retained in soils by straining or filtering action, by sedimentation, or by adsorption to soil surfaces. Adsorbed virions or those associated with each other in aggregates may be removed by sedimentation or by straining as they move through the soil. However, for free virions, adsorption is the primary mode of removal in soil. Adsorption to soil particles retards virus movement through soil; desorption allows further transport through the soil (Yates and Yates, 1988).

In addition to adsorption, soil moisture, hydraulic conditions, pH, salt species and concentrations, virus aggregation, virus type, soil type and properties, and organic matter affect and interact to influence the transport or movement of viruses through the soil (Yates and Yates, 1988; Gerba and Bitton, 1984). Many of these factors influence virus adsorption by soil (Rao et al., 1986). Low pH and high-ionic-strength water, for instance, contribute

to the retention of viruses by soils (Keswick and Gerba, 1980). Unsaturated soil, which also restricts movement of viruses, may enhance adsorption of viruses by holding them in close proximity to soil surfaces (Lance and Gerba, 1984).

Although enteric viruses bound to sludge particles are not easily released, Bitton et al. (1978) note that unbound virions in liquid sludge may penetrate the soil. Most studies have not found viruses in groundwater beneath land application sites, but a few have reported isolating viruses. Jorgensen and Lund (1985) found enteroviruses in water samples 3 m below the surface of a forest 11 weeks after municipal sludge application. Gerba (1987) summarizes information on virus isolation from groundwater near sites of land application of sewage and concludes that "...this information clearly demonstrates that if enteric viruses are present in sewage being applied to the land, at least some of the viruses can be expected to penetrate the subsurface and gain entrance to the underlying groundwater." Sobsey and Shields (1987) caution that migration of enteric viruses through soil to groundwater at application sites is a significant public health concern.

Adsorption. Initially, most viruses applied to soil are retained in the upper soil layers (Rao et al., 1986). When soils containing 7.6-81% sand were studied for their ability to adsorb coliphage f2 from septic tank effluent, most viruses were found in the first 15 cm, although several isolations were found below 85 cm. Similar results were found by Hurst et al. (1980b) and Landry et al. (1980) with seeded poliovirus in soil. Lance et al. (1976) found that flooding a soil column with a sewage virus mixture for 27 days did not saturate the surface layer of soil with viruses. Increasing the concentration of viruses in the sewage water increased the numbers adsorbed at various soil levels but did not change the maximum depth of penetration of the virus (Lance and Gerba, 1980).

Vaughn et al. (1981) suggest that formation of a surface mat of sewage solids explains the greater removal of poliovirus found at low application rates through a coarse sand-fine gravel soil. However, Lance and Gerba (1980) found that the concentration of viruses was greatest near the soil surface when soil columns that had not previously been exposed to sewage water were flooded with a virus-enriched sewage, suggesting that build-up of organic matter near the surface was not responsible for the high concentrations of virus particles detected there.

Lance and Gerba (1982) suggest that viruses applied in sludge will be less mobile that those in sewage water because they will be adsorbed to sludge solids. Bitton et al. (1979b) found no viruses in groundwater after spread of sludge on agricultural land in Florida.

pH. The influence of pH on virus transport is related to its effects on virus adsorption to soil. Studying poliovirus adsorption in 34 minerals and soils, Moore et al. (1981) found that adsorption was strong by most of the neutral and acidic materials. However, because of the great variation in viral adsorption in alkaline materials, substrate pH was not significantly correlated to viral adsorption in this study. Sobsey and Shields (1987) report on a number of studies that indicate that virus retention by soils increases with lower pH levels. With poliovirus, Taylor et al. (1981) found a characteristic pH region of transition from strong to weak adsorption for each adsorbent studied (three soils, a sand, and a clay mineral).

In soil suspension experiments on the effects of 7 soil properties on the adsorption of 15 viruses to 9 soils, Goyal and Gerba (1979) observed that a soil saturation pH<5 provided good viral adsorption. Gerba et al. (1981) divided these 15 viruses into two groups based on their adsorption behavior in soils. For the poorly adsorbed viruses, (including coxsackie B4 viruses, echo 1 viruses, and phages ϕx174 and MS-2), viral adsorption to soil was greatly affected by pH, as well as by CEC and organic matter; but for the highly adsorbed viruses (including polio 1, echo 7, coxsackie B3, and phages T4 and T2), pH and other soil characteristics were not correlated with soil adsorption. Adsorption varies not only with virus type but also with isolates within the same type; this explains the conflicting results from various virus studies (Gerba and Bitton, 1984).

Because of the strong repulsive forces that will result between viruses and soil particles at higher pH values, virus desorption and therefore virus migration will occur if high pH values are induced in the soil environment. However, except under unusual circumstances, these pH values are not expected to be found in the normal environment (Rao et al., 1986).

Mechanisms for pH influence on adsorption of viruses to soils have not been thoroughly studied. Murray and Parks (1980) suggest that van der Waals forces are

responsible for adsorption and that electrostatic repulsion inhibits adsorption. Sobsey and Shields (1987) report that recent studies indicate hydrophobic interactions may also be important in adsorption of viruses to soils.

Ionic Strength. Cations reduce the repulsive forces on both virus and soil particles, allowing adsorption to occur (Gerba and Bitton, 1984). Several studies using soil suspensions or soil columns have shown enhanced adsorption of viruses to a variety of materials with increasing ionic concentrations (Sobsey and Shields, 1987). Taylor et al. (1981) observed that both type and concentration of electrolyte affected the adsorption of poliovirus 2 to soils, and Sobsey et al. (1980) report that addition of divalent cations such as Mg^{2+} caused some viruses to adsorb to poor sorbents.

Since increasing the concentration of ionic salts increases virus adsorption to soil particles, virus transport in the soil is retarded by increasing concentrations of these salts (Sobsey, 1983). During rainfall, the salt concentration decreases, and thus the ionic strength of the soil water; desorption and redistribution of viruses within the soil may occur with the potential for groundwater contamination (Gerba, 1983b). This remobilization of soil-bound viruses has been shown to be more pronounced in sandy than in clay soils and depends on virus type and strain (Gerba and Bitton, 1984). After a heavy rain at a land application site, Wellings et al. (1975) detected viruses in wells that had been virus-free.

Virus Type. Overall virion electronegativity, which is type-dependent, affects adsorption of viruses to soils and hence desorption and migration in the soil (Rao et al., 1986). According to Sobsey and Shields (1987), virus-specific differences in soil adsorption are probably due to physicochemical differences in virus capsid surfaces.

Studying adsorption and subsequent elution with rainwater from columns of intact cores of sandy soil dosed with wastewater, Landry et al. (1979) found differences in extent of adsorption and elution among enterovirus types and among strains of poliovirus type 1. This variation in ability to adsorb to soils has been confirmed in several studies (Sobsey and Shields, 1987). Goyal and Gerba (1979) report differences in adsorption efficiencies by enterovirus types and strains. Comparing adsorptive capacities of various strains of poliovirus type 1, echoviruses 1,7 and 29, and coxsackieviruses B4 and B3 in the laboratory, Gerba et al. (1980) found that adsorption was both type- and strain-dependent.

On the other hand, when comparing movement of echo 1 and echo 29 viruses with polio 1 in soil columns, Lance et al. (1982) observed similar movement of the viruses in soils and suggest poliovirus as a model for virus movement in soil. Although echo 1 did not adsorb well near the surface, leaching patterns were similar for echo 1, echo 29, and polio 1 below the 40-cm depth.

Sobsey et al. (1986) studied virus adsorption and persistence in soil suspensions, and transport in soil columns of poliovirus type 1, echovirus type 1, and HAV. Poliovirus adsorbed more extensively to soils than HAV, which adsorbed better than echovirus. In soil columns, echovirus was transported through the column to the effluent in greatest concentrations, and poliovirus concentrations were lowest in the effluent. The authors suggest that neither poliovirus type 1 nor echovirus type 1 is suitable as a model for adsorption, persistence, and transport of HAV in soil.

Soil Moisture and Flow Rate. Movement of viruses through soil is affected by soil moisture. In laboratory studies with deionized water, Lance et al. (1976) report that viruses near the surface desorb and migrate through soil columns; they suggest that the viruses will continue to travel vertically through the soil with periodic rainfall. Periods of drying between application of water reduced desorption, suggesting that virus movement in soil would not be great unless rainfall occurred within the first few hours after application of sewage. In a study of a land application site, Wellings et al. (1974) report that rainfall influences penetration into the soil depths. No viruses were detected in wells 3 and 6 m below the soil surface until after periods of heavy rainfall.

Lance and Gerba (1984) found that with unsaturated flow conditions, poliovirus did not move below 40 cm in a column of loamy sand; but with saturated conditions, the virus penetrated at least 160 cm. Gerba and Bitton (1984) suggest that with unsaturated-flow conditions, water fills only the smaller soil pores or remains as a film around soil particles, allowing viruses to get closer to particle surfaces. Under saturated soil conditions, coliphage ϕX174 was found to move laterally ~350 m/day (Noonan and McNabb, 1979a).

When soil columns of Red Bay sandy loam were treated with chemical sludge or with anaerobically-treated, polyelectrolyte-conditioned, dewatered sludge and leached with natural rainwater, none of the seeded poliovirus type 1 were found in the leachates during

saturated flow conditions for a sustained period (Pancorbo et al., 1988). According to Pancorbo et al. (1988), association with the solids in the sludges may have immobilized the viruses in the top portion of the soil columns. The high level of cations in these sludges probably enhanced the adsorption of viruses to sludge and soil particles. However, they caution that poliovirus adsorbs more readily to soils and does not migrate through soil as easily as other enteric viruses.

Virus removal by the soil depends on the rate of application of water or effluent (Yates and Yates, 1988). Thus, the application rate of wastewater and sludges will affect the number of viruses passing through the soil and entering the groundwater (Rao et al., 1986). Increasing the application rate from 0.6 m/day to 1.2 m/day increased the number of virus particles moving through the soil column in the effluent, but increasing the flow rates up to 12 m/day gave no further increase in movement (Lance and Gerba, 1980). Lance and Gerba (1980) suggest that virus adsorption is not affected by increases in flow rate up to a breakthrough rate point that corresponds to the rate at which water begins to move only through the large soil pores with little or no contact between viruses and soil particles. Viruses have not generally been detected in well samples or lysimeter samples with infiltration rates below 1 m/day (Lance and Gerba, 1982). Lance and Gerba (1982) state that both column and field studies suggest that water flow velocity is possibly the most important soil characteristic affecting virus movement.

Soil Type. Soil properties have an important effect on virus transport; migration is promoted by coarse-textured soils that do not adsorb well (Yates and Yates, 1988). Virus aggregates or particulate-associated viruses may be strained or filtered out by the smaller soil pores (Yates and Yates, 1988). Wang et al. (1980) report that adsorption is inversely proportional to the permeability of soil. In field studies, movement of viruses to groundwater was a problem primarily when the soil contained coarse sands or gravels (Lance and Gerba, 1982). According to Gerba (1987), viruses can travel long distances in sandy and gravel soils, but studies have not been done in the field to determine just how far viruses move from application sites.

Clay is most active in virus adsorption because of its high CEC and large surface area (Yates and Yates, 1988). Using soil columns flushed with virus-laden settled sewage, Sobsey

et al. (1980) report that four soil types varied in retention of poliovirus type 1, sandy clay loam retaining more than sandy or organic soils. Funderburg et al. (1981) flooded soil columns with simulated rain after application of poliovirus 1 and reovirus 3 in wastewater and found that of the eight different soils tested, those with a high CEC demonstrated stronger retention. However, Goyal and Gerba (1979) did not find a significant correlation between CEC and enterovirus adsorption to soil.

Studying five different soils, Burge and Enkiri (1978) found that bacteriophage ϕX174 adsorption differed among soils and, in most cases, was correlated with soil CEC, specific surface area, and organic matter content. Organic compounds reduce virus adsorption since they compete with viruses for adsorption sites (Gerba and Lance, 1978). In their review of virus persistence and transport in soils, Sobsey and Shields (1987) report evidence that virus association with organic substances may protect them from inactivation by preventing their adsorption to soil, thereby enhancing persistence and mobility in soils. Because of their influence on virus adsorption, humic and fulvic acids increase virus transport through the soil (Bixby and O'Brien, 1979).

In their study of the adsorption of enteroviruses to soils, Goyal and Gerba (1979) found that the nine soils examined differed in their abilities to adsorb several enteric viruses. Although the most important soil property influencing retention was pH, exchangeable aluminum correlated with high adsorption for some viruses. Minerals such as iron oxides have also been shown to increase the retention of viruses in the soil (Sobsey and Shields, 1987).

3.4.2. Transport in Surface Runoff. Virus particles adsorbed to solids in natural water may settle in the bottom sediments. According to Rao et al. (1986), when associated with large particles (>6 μm), viruses settle into sediments; but when adsorbed onto smaller particles (<3 μm), they may remain suspended for a longer time. Accumulated solids-associated viruses settle into a loose, fluffy layer that is easily resuspended by mild turbulence and transported to distant locations. Increased stream velocity or seasonal turnover may also resuspend the sediments, releasing the viruses into the water (Bitton, 1978).

There have been few studies on the transport of microorganisms in runoff from sludge-amended fields. MSDGC and IIT (1979) analyzed water samples from streams, reservoirs, wells and runoff for viruses for 15 months during a 7-year reclamation project in which an aerobically digested and lagooned sludge was applied in large quantities to 15,000 acres. Of the 68 water samples, only 3 contained viruses (an echovirus and 2 unidentified virus isolates). These isolates were found in surface waters. No viruses were found in runoff water from fields, in groundwater or in sludge and soil samples.

Both human and nonhuman enteric viruses were found in large numbers in waters from sites along the Assomption River and its tributaries (Payment, 1989). Untreated wastewaters from the two major cities in the area are discharged into surface water, and runoff occurs from heavy land disposal of untreated farm animal wastes. Concentrations of all viruses varied with season, increasing in early fall.

3.4.3. Transport in the Subsurface and in Groundwater. In sandy gravel aquifers, groundwater flows largely through pores at rates of <1 m/day to a few m/day; in hard rock aquifers, transport is through fissures at 0.3-8000 m/day, or $\leq$26,000 m/day in karstic aquifers (Matthess and Pekdeger, 1985). The larger diameter flow paths in the hard rock and karstic aquifers permit rapid passage of suspended microorganisms. Due to the small diameter of viruses, the filtering action of the porous aquifers is not very effective. Since viruses are subject to adsorption on underground particles, passage through loamy aquifers with high cation concentrations can effectively remove viruses, especially those that adsorb well. The sorptive small particles and microbial slime at the boundary of water and sediment is very effective in reducing virus transport. Desorption may occur with decrease in cation concentration, as in heavy rainfall, with further virus transport. The continuous adsorption/desorption reactions retard movement of viruses relative to groundwater flow, providing time for inactivation processes to affect viruses.

Mack et al. (1972) observed that poliovirus traveled at least 90 m underground. Keswick and Gerba (1980) report that viruses have penetrated to depths of 67 m and travelled horizontally for distances as great as 408 m. When wastewater was applied by rapid infiltration at a land application site, tracer phage and pathogenic animal viruses were found in groundwater at a horizontal distance of 183 m from the site (Schaub and Sorber,

1977). The tracer virus penetrated to the groundwater at the same rate as the effluent and was isolated from an 18.3-m well beneath the application site within 48 hours.

Coliphage, used as a tracer for groundwater movement in carbonate rock terrain in Missouri, traveled 1600 m in 16 hours (Fletcher and Meyers, 1974; Gerba, 1984b). Noonan and McNabb (1979b) observed a rate of movement of ~300 m/day (covering ~920 m) for phages in groundwater at a land disposal site in New Zealand. In shallow groundwater in South Wales, phage used as a tracer moved at a velocity of 36-180 m/day to be isolated in monitoring wells 690 m from the site (Martin and Thomas, 1974).

A number of models have been developed to predict the fate/survival and transport of microorganisms in the subsurface (Grosser, 1984; Vilker et al., 1978; Corapcioglu and Haridas, 1985; Yates and Yates, 1988; and Matthess and Pekdeger, 1985). Most of these models address the transport of viruses under conditions of steady-state flow and saturated soil. Tim and Mostaghimi (1991) developed a model that predicts the transport of viruses with soil water in transient flow conditions in unsaturated soil. The model incorporates mass conservation equations for simultaneous transport of water and viruses through variably saturated media, an equilibrium relationship representing the rapid, instantaneous, and reversible adsorption of virus by the soil matrix and a first-order reaction describing viral inactivation in the subsurface environment (Tim and Mostaghimi, 1991). There is a phase distribution of virus particles--between the liquid phase, or suspended virus particles associated with water moving through the soil, and a solid phase, the adsorbed particles in the soil matrix--that determines the mass available for transport in groundwater. Thus, the model represents the three key elements describing viral transport in the subsurface: (1) transport process, including convection and hydrodynamic dispersion; (2) phase distribution of the viruses between soil and water; and (3) inactivation of the viruses, which ultimately determines whether viruses persist long enough in the subsurface to become a problem by entering the groundwater or reaching surface water.

Tim and Mostaghimi (1991) point out that models addressing virus transport in the subsurface have typically been limited to conditions of steady-state flow in saturated soil, whereas their model addresses transient flow conditions of the unsaturated zone. In a transient flow system, changes in the system can alter the equilibrium. Therefore, to

determine the long-term risk of virus accumulation in soil, those parameters affecting virus interactions in soils must be identified and the resulting fate and transport of the viruses modeled in variably saturated media under the condition of transient, instead of steady-state, flow.

3.4.4. Transport by Wind. Although the dispersion of aerosol-borne viruses is partially dependent on the size of the particle, the time the particles can remain suspended and the distance they can travel are influenced by the airflow, or wind speed, and turbulence (Sattar and Ijaz, 1987).

There is significant overlap in the information on persistence and on transport of viruses in aerosols. For that reason, the studies discussed in Section 3.3.3. will only be summarized with respect to virus transport.

Johnson et al. (1978) found that spray irrigation of wastewater produced a geometric mean aerosol concentration of poliovirus of 0.002/m^3 of air at 50 m downwind of the spray site and a geometric mean concentration of other enteroviruses of 0.014/m^3 of air. Camann et al. (1980) recovered no enteroviruses in the total air volume (1980 m^3) sampled 30 m downwind of a wastewater aeration basin, giving an enterovirus aerosol concentration of <0.002 PFU/m^3. As part of the Lubbock study, enteroviruses were recovered at a geometric mean level of 0.05 PFU/m^3 and a maximum level of 16 PFU/m^3 at 44-60 m downwind from a spray irrigation site using municipal wastewater (Camann et al., 1988). Shuval et al. (1989), performing field investigations on the spread of enteric viruses by wastewater sprinkler irrigation at kibbutzim in Israel, reported that of 15/152 air samples taken at 30-730 m downwind of irrigation sites were presumptively positive for enteric viruses. Even at the distant 730-m station, 3/24 were positive. It is evident from these results that viruses can be transported in aerosols, but quantifying the health risk from these airborne viruses requires more information than is currently available.

4. PARAMETERS FOR MODEL RUNS

4.1. RATIONALE FOR PARAMETER SELECTION

The assessment of human health risk from pathogenic viruses as a result of land application of sewage sludge requires a realistic description of the fate and transport of the pathogens. The preceding chapter describes information that has been found in the published literature describing infectious doses, viral density in treated sludge, and survival and transport of viruses in soil, surface water, groundwater, and aerosols. Limited data were found for several of the many viruses capable of causing disease. Generally, the ranges of reported inactivation rates in different media were wide, varying among reports on the same virus as well as among media. In addition, descriptions of transport in soil were not easily converted to parameters useful in the Pathogen Risk Assessment Model. For this analysis, the most conservative values observed were included among the test runs to determine their effect on the modeled outcome.

Earlier reports in this series (U.S. EPA, 1990; 1991) identified a number of alterations that would improve the operation of the model. However, for this study, the model was used without changes. Therefore, the computations made during model runs are comparable to those in the previous studies. In cases where default values presented in the initial description of the model were judged to be unrealistic, the default values were replaced on the basis of best scientific judgement.

In these model runs, it is assumed that viruses are transported into subsurface soil and subsequently into groundwater and are included in any droplet aerosols formed by spray application, as well as in any particulate aerosols formed by disturbance of the soil by wind or by cultivation. It is also assumed that they will die at a characteristic rate that depends on the ambient temperature and the medium in which they are found; thus, there are different inactivation rates for the same organism in moist soil, dry particulates, droplet aerosols and water. Default parameter values are included in the program's code. If it is necessary to update these values as indicated by new information or to revise them to conform to specific conditions, new values are entered during the initiation of the model run (U.S. EPA, 1989a). In a number of cases, the default values used are known to be

unreasonable for the average case but are chosen to be protective; in some cases, no data are available to support more than a best scientific judgement; in other cases, modeling and research are available to document the values used. More detailed discussion of the choice of parameter values can be found in U.S. EPA (1989a).

The most significant parameters for risk of infection have been shown to be density of pathogens in sludge, inactivation or die-off rates, transfers among exposure media, and infectious dose (U.S. EPA, 1989a; 1990; 1991). The values chosen for initial density represent a reasonable upper bound for density of viruses in treated sludge for the applications indicated (Table 3-3). Because of new information, the base value for viruses in composted sludge, 2500/kg dry weight, is markedly larger than the value recommended in the model conceptualization documents (U.S. EPA, 1989a). In some tests of the limits of sensitivity of the model, virus concentration was set at 500/kg. The other values remain the same as described previously (U.S. EPA, 1989a). Transfers among exposure media are not well characterized and are estimates only. The infectious dose of many viruses may not be known, and even for well-characterized viral types, infectivity may depend on many conditions. Therefore, because experimental infections have indicated that a single virus may cause infection, an infectious dose of 1 was used as the default value. Reported inactivation rates vary widely, depending on both the virus and the physical and chemical conditions; additional research is necessary to characterize inactivation rates accurately. In the test model runs, each of the crucial parameters was varied, usually above and below the default value, to determine the importance of that parameter in model outcome. Parameter values were tested extensively for the first and second sites, and only the sensitive parameters so identified were tested in subsequent model runs for the other sites.

The temperature-specific rate used in the model for inactivation of viruses in moist soil is calculated by an algorithm derived from a line fitted to a logarithmic transform of survival data found in published literature (U.S. EPA, 1989a). This equation is as follows:

$$RHO = 10^{-10^{(A*TEMP^2-B)}}$$

Where:

RHO	=	Fractional survival
A	=	SLOPES [P(37)]
TEMP	=	Temperature of soil (°C)
B	=	NTRCPS [P(38)]

To generate alternative values for the present sensitivity analysis, the slope was increased or decreased by a factor of two, and the intercept was adjusted by trial until the inactivation rate at 0°C remained approximately the same. Alternatively, values were chosen by trial to fit information presented in Hurst (1989). The relative logarithmic inactivation per hour at various temperatures for moist soil was calculated; the results are shown in Table 4-1. It is clear from this table that the slope and intercept of the inactivation curve are important in determining the extent of inactivation by temperature.

The lowest temperature reported for observations of inactivation of viruses in water was -20°C. This temperature is unrealistically low to be used throughout the operation of a model designed to represent the growing season. However, it could be used to calibrate an algorithm for temperature-dependent inactivation similar to the one described above for soil. The model does not provide for temperature-dependent inactivation of viruses in water, because there were insufficient data to parameterize the relevant algorithm.

As alternatives to these values, temperature-insensitive values were substituted during some of the model runs. Reported inactivation rates range from 7.1×10^{-5} to 1.6×10^{-1} logs/hour in soil (Table 3-4), 1.6×10^{-4} to 1.4×10^{-1} logs/hour in water (Table 3-5), and 4.9×10^{-5} to 8×10^{-7} logs /second in aerosols (Table 3-6); these values were substituted to test the inactivation algorithm.

A number of parameters are interdependent. For example, the rate and depth of irrigation should not combine to exceed the infiltration rate and moisture-holding capacity of the soil; otherwise, runoff of irrigation water will occur. However, in the present test, no attempt was made to prevent runoff by irrigation. For parameters directly affecting specific crops or animal feeding practices, the relevant practices must be specified when those parameters are varied. For example, when testing FCROP1 [P(46)] and FCROP4 [P(49)],

TABLE 4-1

Temperature Algorithm for Inactivation of Viruses

	Standard	Test Values				
A=	0.00145	0.0006	0.002	0.0007	0.0029	0.0015
B=	2.957	2.957	2.957	3.0	3.0	3.3
Temp.		Log_{10} Inactivation per Hour				
0	-0.00110	-0.00110	-0.00110	-0.00100	-0.00100	-0.00050
4	-0.00116	-0.00112	-0.00118	-0.00102	-0.00111	-0.00052
8	-0.00136	-0.00120	-0.00148	-0.00110	-0.00153	-0.00062
12	-0.00178	-0.00134	-0.00214	-0.00126	-0.00261	-0.00082
16	-0.00259	-0.00157	-0.00358	-0.00151	-0.00552	-0.00121
20	-0.00419	-0.00191	-0.00696	-0.00190	-0.01445	-0.00199
24	-0.00755	-0.00244	-0.01566	-0.00253	-0.04681	-0.00366
28	-0.01512	-0.00326	-0.04083	-0.00353	-0.18775	-0.00751
32	-0.03371	-0.00454	-0.12331	-0.00520	-0.93239	-0.01721
36	-0.08359	-0.00661	-0.43151	-0.00807	-5.73323	-0.04405

both of which determine transfers to an aboveground crop, the aboveground crop option (CROP [P(66)]=1) must be used. Otherwise, the exposures affected by FCROP1 and FCROP4 will not be calculated. Similarly, some transfers are not relevant to all practices, so the parameters governing them need not be tested in all practices.

4.2. PARAMETER VALUES

The values of the main program parameters used in the initial study are given in Table 4-2. The values of the parameters are based on the model description (U.S. EPA, 1989a), which explains their meaning and use in more detail. The parameters include a number of transfer factors, which regulate the fraction of the pathogens in a particular compartment that are transferred to another compartment each hour or at specific times. For example, parameter 46 (FCROP1) specifies the fraction of viruses in the soil surface that are transferred to the surface of the aboveground crop each hour the crop is present. Other parameters may be flags to indicate that a specific subroutine should be included. In cases in which the parameter is a number with a unit, the unit is indicated in the table.

Values for Subroutine RISK, which calculates direct onsite exposures and modifications related to processing of crops and meat, are given in Table 4-3. Parameters for Subroutine GRDWTR are given in Table 4-4. Site-specific descriptive parameters for rainfall are given in Section 5, Sites for Model Runs. All five practices were tested with appropriate combinations of these variables to determine which ones are significant for infection. In addition, application of sludge to municipal parks and golf courses was modeled by use of Practice V combined with characteristic parameters for lawns in Practice III to assess runoff to an onsite pond. Parameters shown to have no effect on the outcome of model runs at the first site were not tested extensively in model runs for the other sites.

4.2.1. Main Program Parameters. The names, definitions, and values of the main program parameters are listed in Table 4-2. The default values are listed in **bold-face** type, and the rationale for choice of each value is summarized. Each value of each parameter was used in model runs for all practices at Site 1. Some variables were then eliminated from the sensitivity analysis because they seemed to cause no significant effect on the model outcome. In one set of model runs for Site 2, more conservative inactivation rates were used; they

TABLE 4-2

Main Program Parameters

Variable #	Name	Definition	Values	Rationale for Values
1	ASCRS	Pathogen density (pathogens/kg dry wt)	$2x10^3$ $1x10^5$ $2.5x10^3$ $1x10^6$	Lower values expected to be insignificant. Default value, Practices I-III. Default value, Practices IV-V. Above highest reported values.
2	APRATE	Application rate (kg/ha)	$2x10^3$ $10x10^3$ $12.5x10^3$ $25x10^3$ $1x10^5$	Low reported value (U.S.EPA, 1983). Default value, practice-specific: Practice I Practices II and III Practices IV and V Above highest reported values.
3	ASCIN	Pathogen concentration (Pathogens/ha)	--	Calculated by the program from ASCRS and APRATE.
4	TREG	Waiting limit (proposed U.S. EPA Pathogen Reduction Regulations)	0	Make pathogens available immediately for exposure as a worst case.
6	APMETH	Application method	-1 0 +1	Demonstrate the relative effects of spray, subsurface, and surface application, respectively.

TABLE 4-2 (continued)

Variable #	Name	Definition	Values	Rationale for Values
7	AREA	Area of field, lawn or garden	1 **10** 400	Low value for application in small fields, high value for large-scale farming. (Values are specific for Practices I, II and III.)
			0.005 **0.015** 0.2	Values specific for Practice IV, small to large residential lawns.
			0.01 **0.05** 0.2	Values specific for Practice V, small to large residential gardens.
8	TEMP	Air temperature (° C)	--	Calculated daily by the program from input values.
9	AQUIFR	Aquifer thickness (m)	1 **10** 40	Shallow, confined aquifer. Default value. Deep, extensive aquifer.
10	POROS	Fractional water content of aquifer	0.1 **0.3** 0.7	Dry or highly consolidated aquifer. Default value. Highly saturated aquifer.
11	FILTR8	Infiltration rate (cm/hr) (unsaturated soil)	0.5 **2.5** 10	Consolidated, hard-packed or saturated soil. Default value. Rapidly draining soil.

TABLE 4-2 (continued)

Variable #	Variable Name	Definition	Values	Rationale for Values
12	MID	Infective dose (number of pathogens)	**1** 10 100	Lowest possible infective dose, default value. Realistic intermediate value. Higher values probably insignificant.
13	TRAIN	Time of rainfall (hr)	Site-specific	Rainfall is the most significant factor in surface runoff/sediment transport to the onsite pond.
14	RDEPTH	Depth or rainfall (cm)	Event-specific	Rainfall depth, entered with each rainfall event.
15	TK	Time since last rain began (hr)	--	Counter for time since last rainfall began.
16	TIRRG	Time since last irrigation began (hr)	--	Counter for time since last irrigation began.
17	IRMETH	Irrigation method	**0** 1	Compare the effects of spray irrigation and ditch irrigation.
18	DILIRR	Fraction of irrigation water that is contaminated	**0** 1	Demonstrate the effect of irrigation with sludge as compared to uncontaminated water.
19	NIRRIG	Number of irrigations per week	0 **2** 7	No irrigation. Default, twice weekly. Daily irrigation.

TABLE 4-2 (continued)

Variable #	Name	Definition	Values	Rationale for Values
20	IRRATE	Rate of irrigation (cm/hr)	0.1 **0.5** 10	Low-rate of irrigation (cm/hr). Default value. Very high rate of irrigation.
21	DEPTH	Depth of irrigation (cm)	1 **2.5** 10	Limited depth of irrigation (cm). Default value. Extensive irrigation.
22	COUNT	Pathogen concentration in irrigation water	--	Defaults to ASCRS [P(1)] for irrigation.
23	TWIND	Time when windstorm begins (hr)	36 **60** 4320	Very early windstorm. Default early windstorm. Delay windstorm to 180 days.
24	DWIND	Duration of windstorm (hr)	2 **6** 300	Brief windstorm. Default value. Windstorm of long duration (12.5 days).
25	WINDSP	Speed of wind during windstorm (m/sec)	7 **18** 27	Very mild windstorm (~16 mph). Default value (~40 mph). Strong windstorm (~60 mph).

TABLE 4-2 (continued)

Variable #	Name	Definition	Values	Rationale for Values
26	EPSMLT	Particle size multiplier for soil	0.1 **0.33** 0.5	Very fine soil. Default value. Coarse soil.
27	ESILT	Fractional silt content of soil	0.1 **0.4** 0.8	Silt content of sand. Default value, loam or clay loam. Silt content of silt loam.
28	EHT	Height of particulate cloud during tilling (m)	1 **2** 5	Very low particulate cloud (m). Default value. High, dispersed tilling emissions.
29	SCRIT	Critical windspeed (m/sec)	2 **7.5** 20	Very loose soil. Default value. Crusted or damp soil.
30	COVER	Fraction of soil surface covered by vegetation	**0** 0.9	Compare surface runoff/sediment transport for bare soil and soil with vegetation (matched with values in Subroutine RAINS).
31	AEREFF	Efficiency of aerosol formation	$1x10^{-4}$ $\mathbf{1x10^{-3}}$ $1x10^{-2}$	Compare default value to an unrealistically high value to determine whether the model is sensitive to offsite aerosols.

TABLE 4-2 (continued)

Variable #	Variable Name	Definition	Values	Rationale for Values
32	BREEZE	Windspeed during irrigation (m/sec)	2 **4** 7 10	Vary to determine sensitivity of air transport model during irrigation to windspeed.
33	HT	Height of downwind off-site receptor (m)	0.2 **1.6** 10	Receptor near ground level. Default value. Receptor on tower, tree or roof.
34	ANDAY	Current day of the year	--	Calculated by program.
35	TMAX	Maximum monthly average air temperature (° C)	10 **Site-specific** 40	Very cool site. Very warm site.
36	TMIN	Minimum monthly average air temperature (° C)	-30 **Site-specific** 15	Very cold site. Warm site.
37	SLOPES	Slope of inactivation vs temperature curve, moist soil	0.0007 **0.00145** 0.0029	Arbitrary lower value. Default value. Arbitrary upper value.
38	NTRCPS	Intercept of inactivation vs temperature curve, moist soil	3.0 **2.957** 3.0	Adjusted to slope for no change at 20° C. Default value. Adjusted to slope for no change at 20° C.

TABLE 4-2 (continued)

Variable #	Variable Name	Definition	Values	Rationale for Values
41	ASLSUR	Transfer fraction, application to soil surface	0 **0.9** 1	Unrealistic, minimum possible. Default value. Maximum possible transfer.
42	FSSUR	Transfer fraction, application to subsurface soil	0 **1**	Unrealistic, minimum possible. Default value, maximum possible transfer.
43	FRRAIN	Transfer fraction, soil surface to surface runoff	--	Calculated by the program.
44	SUBSOL	Transfer fraction, soil surface to subsurface soil	$1x10^{5}$ $\mathbf{1x10^{3}}$ $1x10^{1}$	Very low transfer to subsurface. Default value. Very high transfer to subsurface.
45	SUSPND	Transfer fraction, soil surface to soil surface water	$1x10^{3}$ $\mathbf{1x10^{2}}$ $1x10^{1}$	Vary to determine sensitivity of model to resuspension from surface soil into soil surface water.
46	FCROP1	Transfer fraction, soil surface to crop 1	$\mathbf{1x10^{8}}$ $5x10^{6}$ $5x10^{5}$	Default value. Calculated as 0.1 g soil/crop unit for default yield. Calculated as 1.0 g soil/crop unit for default yield.

TABLE 4-2 (continued)

Variable #	Name	Definition	Values	Rationale for Values
47	FCROP2	Transfer fraction, soil surface to crop 0	$1x10^{-6}$	Calculated as 0.02 g soil/crop unit for default yield.
			$2.5x10^{-5}$	Calculated as 0.5 g soil/crop unit for default yield.
			$6x10^{-3}$	Default value.
48	FCROP3	Transfer fraction, soil surface to crop -1	$5x10^{-7}$	Calculated as 0.05 g soil/ crop unit for default yield.
			$2.5x10^{-5}$	Calculated as 2.5 g soil/crop unit for default yield.
			$1x10^{-3}$	Default value.
49	FCROP4	Transfer fraction, crop 1 to soil surface	$1x10^{-5}$	Arbitrary lower value.
			$1x10^{-4}$	Default value.
			$1x10^{-3}$	Arbitrary upper value.
50	FCROP5	Transfer fraction, crop 0 to soil surface	$1x10^{-4}$	Arbitrary lower value.
			$1.2x10^{-2}$	Default value.
			$1x10^{-1}$	Arbitrary upper value.
51	FCROP6	Transfer fraction, crop -1 to soil surface	**0**	Default value.
			$1x10^{-3}$	Arbitrary upper value.
52	FCROP7	Transfer fraction, subsurface soil to crop -1	$2.5x10^{-9}$	Arbitrary value; most of crop is above subsurface.
			$2x10^{-4}$	Default value.

TABLE 4-2 (continued)

Variable #	Name	Definition	Values	Rationale for Values
53	FRGRND	Transfer fraction, subsurface soil to groundwater	$1x10^{-5}$ **$1x10^{-3}$** $1x10^{-1}$	Arbitrary lower value. Default value. Arbitrary upper value.
54	SSWTCS	Transfer fraction, soil surface water to crop surface	0.01 **0.1**	Arbitrary lower value. Default value.
55	PSTMG	Transfer fraction, grass removed during mowing	0.2 **0.5** 0.7	Arbitrary lower value. Default value. Arbitrary upper value.
56	CSTSS	Transfer fraction, crop surface to soil surface	$1x10^{-4}$ **$1x10^{-2}$** $1x10^{-1}$	Arbitrary lower value. Default value. Arbitrary upper value.
57	SSTCS	Transfer fraction, soil surface to crop surface	$1x10^{-7}$ **$1x10^{-5}$** $1x10^{-2}$	Arbitrary lower value. Default value. Arbitrary upper value.
58	CSTSSW	Transfer fraction, crop surface to soil surface water	0.1 **0.5** **0.75** 1	Arbitrary lower value. Default value, Practices II and III. Default value, Practices I, IV and V. Arbitrary upper value.
59	DTCTMT	Transfer fraction, animal consumption to meat	**0** 1	No transfer of viruses to meat (default value). Invasion of edible tissue by every infective virion ingested.

TABLE 4-2 (continued)

Variable #	Variable Name	Definition	Values	Rationale for Values
60	DTCTMK	Transfer fraction, animal consumption to milk	**0** 1	No transfer of viruses to milk. Invasion of milk by every infective virion ingested.
61	TMTSS	Transfer fraction, manure to soil surface	0 **0.7** 1	Arbitrary lower value. Default value. Arbitrary upper value.
62	TMTH	Transfer fraction, manure to hide	0 **0.001** 0.1	Arbitrary lower value. Default value. Arbitrary upper value.
63	TMTU	Transfer fraction, manure to udder	0 **0.001** 0.01	Arbitrary lower value. Default value. Arbitrary upper value.
64	HTM	Transfer fraction, hide to meat	0 **0.1** 1	Arbitrary lower value. Default value. Arbitrary upper value.
65	UTM	Transfer fraction, udder to milk	0 **0.05** 1	Arbitrary lower value. Default value. Arbitrary upper value.
66	CROP	Type of crop (-1 for below-ground, 0 for on-ground, 1 for aboveground)	-1 0 **+1**	Type of crop is important in exposure during consumption of crop.

TABLE 4-2 (continued)

Variable #	Name	Definition	Values	Rationale for Values
67	TCULT	Cultivation time (hr) or flag for no cultivation (-2) or cultivation every 2 weeks (0)	0 -2	Include regular cultivation to determine whether model is sensitive to generation of particulate aerosols.
68	TCROP	Time crop surface is present (hr)	240	Establish early appearance of crop surface to facilitate demonstration of sensitivity of exposure to presence of crop.
69	THARV	Time crop is harvested (hr)	300	Establish early harvesting of crop to facilitate demonstration of sensitivity of exposure to presence of crop.
70	YIELD1	Yield of aboveground crop (g/ha)	$5x10^6$ **$2.5x10^7$** $1x10^8$	20% of default for low yield. Default value. 4 times default for high yield.
71	YIELD2	Yield of on-ground crop (g/ha)	$5x10^6$ **$2.5x10^7$** $1x10^8$	20% of default for low yield. Default value. 4 times default for high yield.
72	YIELD3	Yield of below-ground crop (g/ha)	$2x10^5$ **$1x10^6$** $4x10^6$	20% of default for low yield. Default value. 4 times default for high yield.
73	HAY	Yield of grass or hay (kg/m^2)	0.32 **1.6** 6.4	20% of default for low yield. Default value. 4 times default for high yield.

TABLE 4-2 (continued)

Variable #	Name	Definition	Values	Rationale for Values
74	PLNT1	Fraction of garden in aboveground crop	0.1 **0.4** 0.8	Arbitrary lower limit. Default value. Arbitrary upper limit.
75	PLNT2	Fraction of garden in on-ground crop	0.1 **0.3** 0.8	Arbitrary lower limit. Default value. Arbitrary upper limit.
76	PLNT3	Fraction of garden in below-ground crop	0.1 **0.3** 0.8	Arbitrary lower limit. Default value. Arbitrary upper limit.
77	PPG	Pathogen concentration in food (number/g)	--	Calculated by program.
78	CATTLE	Type of cattle (flag)	-1 **1**	Beef cattle option. Dairy cattle option.
79	COWS	Size of herd (cattle/ha)	3 **12** 100	Arbitrary lower value, for low-density grazing. Default value. Arbitrary upper value, for high-density grazing.
80	STORAG	Length of forage storage (hours)	0 **720** 1440	Minimum possible. Default value. Store for (arbitrary) 2 months before feeding.

TABLE 4-2 (continued)

Variable #	Name	Definition	Values	Rationale for Values
81	FORAG	Forage consumed per cow per day (kg)	**7** 25 75	Default value. Estimated value for total consumption. Estimated higher value for total consumption.
82	ALFALF	Percent of feed that is harvested crop	10 **30** 80	Arbitrary lower value. Default value. Arbitrary upper value.
83	SCNSMP	Soil consumed per cow per day (kg)	0.25 **1.1** 2.5	Arbitrary lower value. Default value. Arbitrary upper value.
84	FATTEN	Number of hours cattle are fed forage	360 **720** 5760	Half of default value. Default value. Cattle kept on forage for 8 months.
85	TSLOTR	Time of slaughter (day)	30 **-2** 240	Cattle slaughtered after 1 month. Default value, flag to prevent slaughter of dairy herd. Cattle kept on forage for 8 months before slaughter.

TABLE 4-3

Parameters for Subroutine RISK

Parameter # Name	Value*	Rationale for Choice of Values
5 DRECTC (g/day)	0.02 **0.1** 1.0	Determine effect of amount of contaminated crop surface ingested during routine daily work in the field.
6 DRECTS (g/day)	0.02 **0.1** 1.0	Determine effect of amount of contaminated soil ingested during routine daily work.
8 ICAN	**0** 1	Default, for fresh vegetables. Flag for canning sequence.
11 IFREE	**0** 1	Default, for fresh vegetables. Flag for freezing sequence.
29 TSTM2 (hours)	360 **720** 2880	Reduced storage time of meat before processing. Default value. Increased storage time of meat before processing.
32 TSTR7 (hours)	360 **720** 2880	Reduced storage time of vegetables after processing. Default value. Increased storage time of vegetables after processing.
33 VOLPND (m^3/ha)	$2x10^1$ **$1x10^2$** $1x10^3$	Very small onsite pond. Default value. Increased value for pond.
34 XDIST (m)	100 **200**	Determine effect of distance to receptor of offsite aerosol.
35 YDIST (m)	**0** 10 30	Determine effect of lateral distance of receptor from centerline of aerosol plume.

*Default values are in **bold-face** type.

TABLE 4-4

Parameters for Subroutine GRDWTR

Parameter #	Name	Definition	Value*	Rationale for Choice of Values
2	V	Velocity (cm/hr)	0.9 **3.6** 10.8	Arbitrary lower value. Default value. Arbitrary upper value.
3	D	Dispersion coefficient	20 **60** 100	Arbitrary lower value. Default value. Arbitrary upper value.
4	R	Retardation coefficient	0.2 **1.0** 2.0	Arbitrary lower value. Default value. Arbitrary upper value.
9	XI	Starting distance (m)	20 **50** 200	Arbitrary lower value. Default value. Arbitrary upper value.
10	DX	Distance increment (m)	20 **50** 200	Arbitrary lower value. Default value. Arbitrary upper value.
11	XM	Maximum distance (m)	20 **50** 200	Arbitrary lower value. Default value. Arbitrary upper value.

*Default values are in **bold-face** type.

were 0.0017 logs/hour for moist soil, 0.0015 logs/hour for water, and $8x10^{-7}$ logs/second for aerosols.

4.2.2. Parameters for Subroutine RISK. Subroutine RISK is used to calculate exposures in various exposure compartments, using data for pathogen concentration in each compartment as calculated by the model. Parameters for Subroutine RISK were varied to simulate differences in exposure to viral pathogens by direct contact with soil or crop surfaces, viruses in processed foods, the volume of the onsite pond (which had a direct effect on concentration of viral particles in surface water), or location of the receptor of offsite aerosols. Values of the parameters for Subroutine RISK are given in Table 4-3. Default values are printed in **bold-face** type.

4.2.3. Parameters for Subroutine GRDWTR. Values for this subroutine are difficult to find in the published literature. Ongoing research and development of groundwater transport models for pathogens should yield valuable new information to allow more realistic choices of parameter values for Subroutine GRDWTR. Values used in the study are given in Table 4-4.

4.2.4. Parameters for Subroutine RAINS. Because the Modified Universal Soil Loss Equation, which is the basis for Subroutine RAINS, depends on soil type, topography and land use practices, parameters for Subroutine RAINS are influenced strongly by the choice of site. The sites suggested for trial runs of the Pathogen Risk Assessment Model include one location each chosen from potential farming areas of TN, CA, FL, NM, IA and WA.

Parameters for Subroutine RAINS are defined in Table 4-5. Values for the parameters were chosen to be appropriate for soil type, topography and meteorologic patterns for these locations (see Chapter 5). Although the model is limited in its ability to represent the rainfall pattern of any location because of its restriction to $\leq$10 rainfall events, inclusion of these events early in the model run ensures that the effects of rainfall on surface runoff/sediment transport are maximized.

TABLE 4-5

Parameters for Subroutine RAINS

Parameter #	Name	Definition
2	PDUR	Duration of rainfall (hr)
3	PTOT	Total rainfall (cm)
4	BTLAG	Basin time lag (hr)
5	CN	Curve number
6	AMC	Antecedent moisture conditions
7	STAD	Storm advancement coefficient
8	USLEK	USLE K value (soil erodibility factor)
9	USLEL	USLE L value (slope length factor)
10	USLES	USLE S value (slope steepness factor)
11	USLEC	USLE C value (cover management factor)

5. SITES FOR MODEL RUNS

Six sites were chosen to provide a variety of soil types, topography and meteorologic patterns. Other than Anderson County, TN, for which more detailed meteorologic data were available to the authors, specific sites were chosen arbitrarily with the goal of geographic diversity. Data on soil properties were taken from U.S. Soil Conservation Service soil surveys, which have been developed for each county in the United States. Meteorologic data were taken from the National Oceanic and Atmospheric Administration Local Climatological Data Annual Summaries for 1981 (NOAA, 1981). The sites chosen for the model runs are described below.

5.1. SITE 1: ANDERSON COUNTY, TN

Values of site-specific variables were chosen to reflect conditions at an agricultural location in the Clinch River Valley of East Tennessee.

5.1.1. Description of Soil. The soil chosen for the model run is the Claiborne series, which comprises fine-loamy, siliceous, mesic Typic Paleudults. It is further described as follows (USDA, 1981a):

> The Claiborne series consists of deep, well drained soils that formed in sediment deposited by water or in residuum of dolomite. These soils are on ridgetops, on hillsides, and at the base of slopes. The slope range is 5 to 45 percent, but in most areas the gradient is 12 to 30 percent....
>
> The solum is more than 60 inches thick. Depth to dolomite bedrock is more than 72 inches. The soil is strongly acid or very strongly acid throughout except for the surface layer where limed. The content of coarse chert fragments ranges from 5 to 25 percent in each horizon. These fragments commonly increase in size and abundance with increasing depth.

Claiborne soils are of hydrologic group B, characterized by moderately low runoff potential, moderate infiltration rates and moderate rates of water transmission.

For this analysis, it was assumed that sites with slopes >10% (6°) would not be used because of the likelihood of excessive runoff.

5.1.2. Narrative Climatologic Summary. The following climatologic summary for Oak Ridge, Anderson County, TN, was taken from NOAA (1981):

Oak Ridge is located in a broad valley between the Cumberland Mountains, which lie to the northwest of the area, and the Great Smoky Mountains, to the southeast. These mountain ranges are oriented northeast-southwest and the valley between is corrugated by broken ridges 300 to 500 feet high and oriented parallel to the main valley. The local climate is noticeably influenced by topography. Prevailing winds are usually either up-valley, from west to southwest, or down-valley, from east to northeast. During periods of light winds daytime winds are usually southwesterly, nighttime winds usually northeasterly. Wind velocities are somewhat decreased by the mountains and ridges. Tornadoes rarely occur in the valley between the Cumberlands and the Great Smokies. In winter the Cumberland Mountains have a moderating influence on the local climate by retarding the flow of cold air from the north and west.

The coldest month is normally January but differences between the mean temperatures of the three winter months of December, January, and February are comparatively small. The lowest mean monthly temperature of the winter has occurred in each of the months December, January, or February in different years. The lowest temperature recorded during the year has occurred in each of the months November, December, January, or February in various years. July is usually the hottest month but differences between the mean temperatures of the summary months of June, July, and August are also relatively small. The highest mean monthly temperature may occur in either of the months June, July, or August and the highest temperature of the year has occurred in the months of June, July, August, and September in different years. Mean temperatures of the spring and fall months progress orderly from cooler to warmer and warmer to cooler, respectively, without a secondary maximum or minimum. Temperatures of 100° [38°C] or higher are unusual, having occurred during less than one-half of the years of the period of record, and temperatures of zero or below are rare. The average number of days between the last freeze of spring and the first freeze of fall is approximately 200. The average daily temperature range is about 22° [12°C] with the greatest average range in spring and fall and the smallest in winter. Summery nights are seldom oppressively hot and humid. Low level temperature inversions occur during approximately 57 percent of the hourly observations. Fall is usually the season with the greatest number of hours of low level inversion with the number decreasing progressively through spring and winter to a summertime minimum but seasonal differences are small.

5.1.3. Temperature. The monthly average temperatures at this location ranged between a low of 2.8° C and a high of 24.8° C.

5.1.4. Rainfall. An hourly rainfall record for April and May, 1989, was obtained from the Atmospheric Turbulence and Diffusion Laboratory, National Oceanic and Atmospheric

Administration, Oak Ridge, TN. Profiles of the first ten rain events beginning April 1, the time the model run is initiated, were constructed from this record. Profiles consisted of the duration of the event (PDUR), the total amount of precipitation in the event (PTOT) and the storm advancement coefficient (STAD), which was determined by inspection of the hourly precipitation. The resulting parameters were as follows:

Event No.	START (hr)	PDUR (hr)	PTOT (cm)	STAD
1	77	5	1.60	0.65
2	174	8	1.52	0.36
3	726	11	1.55	0.12
4	826	7	3.30	0.27
5	924	4	1.50	0.56
6	1180	5	2.31	0.19
7	1340	12	4.06	0.52
8	1549	2	1.63	0.46
9	1590	9	2.52	0.52
10	1650	14	3.48	0.45

5.1.5. Parameters for Subroutine RAINS. Parameters for Subroutine RAINS were modified to describe local rainfall and soil conditions for Anderson County, TN. Values were calculated as described in Appendix B of U.S. EPA (1989a). The values used in the model run were based on a field with dimensions 500 m by 200 m, sloping at an angle of 6° (10.5%). It was assumed for Practice I that before a crop was present, the cover management factor was not modified, whereas after the crop was present, a canopy cover of 30%, a canopy height of 0.5 m and a relative root network factor of 30% were provided; for Practices II and III, the canopy cover was taken to be 90%, the canopy height was taken to be <0.5 m and a relative root network factor of 90% was assumed. The resulting values were:

Parameter No.	Name	PRACTICE NUMBER I	II	III
4	BTLAG	0.2	0.31	0.31
5	CN	78	64	64
6	AMC	3 (<TCROP) 2 (≥TCROP)	2	2
8	USLEK	0.32	0.32	0.32
9	USLEL	4.76	4.76	4.76
10	USLES	1.25	1.25	1.25
11	USLEC	0.45 (<TCROP) 0.30 (≥TCROP)	0.02	0.02

The initial value (0.02) for USLEC in Practices II and III was subsequently shown to cause errors that halted operation of the program, so in subsequent runs and for all other sites that parameter value was changed to 0.05.

5.2. SITE 2: CHAVES COUNTY, NM

Values for site-specific variables for Site 2 were chosen to represent an agricultural area near Roswell, a city in southeast NM.

5.2.1. Description of Soil. The soil chosen for the model run is the Pecos Series, which comprises fine, mixed, thermic Torrertic Haplustolls. It is further described as follows (USDA, 1980):

> The Pecos series are deep, moderately well drained, very slowly permeable soils on flood plains. The soils formed in calcareous, saline, stratified, clayey alluvium. Slope is 0 to 1 percent.
>
> Typically, the surface layer is reddish brown silty clay loam about 12 inches thick. The upper 10 inches of the substratum is reddish brown clay, the next 20 inches is reddish brown silty clay and silty clay loam, and the lower part to a depth of 60 inches or more is brown loam and fine sandy loam. Salinity is moderate. Available water capacity is high.

Pecos soils are of hydrologic group D, characterized by a very slow infiltration rate (high runoff potential) when thoroughly wet. They consist chiefly of clays that have a high shrink-swell potential, soils that have a permanent high water table, soils that have a claypan or clay layer at or near the surface, and soils that are shallow over nearly impervious material. These soils have a very slow rate of water transmission.

5.2.2. Narrative Climatologic Summary.

> The climate at Roswell conforms to the basic trend of the four seasons, but shows certain deviations related to geography. A location south and west of the main part of major weather activity affords a degree of climatic seclusion. There are also topographic effects that are inclined to alter the course of the weather in this area. Higher landmasses almost surround the valley location, with a long, gradual descent from points southwest through west and north. The topography acts to modify air masses, especially the cold outbreaks in wintertime. Downslope warming of air, as well as air interchange within a tempering environment, often prevents sharp cooling. Moreover, the elevation of 3,600 feet in common with the geographic situation, discourages a significant part of the heat and humidity that originates in the south in summer. In winter, subfreezing at night is tempered by considerable warming during the day. Zero [°F] or lower temperatures occur as a rule a time or two each winter. Subzero cold spells are of short duration. Winter is the season of least precipitation (NOAA, 1981).

5.2.3. Temperature. The monthly average temperatures at this location ranged between a low of 4.2° C and a high of 26.2° C.

5.2.4. Rainfall. Times, duration and total rainfall for each rainfall event were constructed from the rainfall record for 1980 at the location (NOAA, 1981). The record provided the date and amount of the largest rainfall during a 24-hour period each month, as well as the total amount of rainfall each month. The largest rainfall (greater than the subroutine's lower limit of 1 cm) was always used, and the remaining rainfall during the period was divided into events placed at arbitrary times. The storm advancement coefficient was chosen to reflect the nature of rainfall in the region; the low number used reflects a preponderance of thunderstorms and sudden showers, whereas larger numbers were used for some other sites to reflect a more gradual buildup of the rainstorm. The resulting parameters were as follows:

Event No.	START (hr)	PDUR (hr)	PTOT (cm)
1	328	5	1.17
2	784	8	4.5
3	966	6	4.55
4	1280	3	1.5
5	1830	8	3.05
6	2174	10	7.75
7	2366	10	12.47
8	2800	5	3.45
9	3328	10	4.19
10	3518	6	3.0

5.2.5. Parameters for Subroutine RAINS. Parameters for Subroutine RAINS were modified to describe local rainfall and soil conditions. The slope value used in the model run was 1 degree (1.7%). The resulting values were:

Parameter No.	Name	PRACTICE NUMBER I	II	III
4	BTLAG	0.32	0.38	0.38
5	CN	89	84	84
7	STAD	0.25	0.25	0.25
8	USLEK	0.32	0.32	0.32
9	USLEL	2.54	2.54	2.54
10	USLES	0.16	0.16	0.16
11	USLEC	0.45 (<TCROP) 0.25 (≥TCROP)	0.05	0.05

5.3. SITE 3: CLINTON COUNTY, IA

Values for site-specific variables for Site 3 were chosen to represent an agricultural area in eastern IA in a county that borders on the Mississippi River.

5.3.1. Description of Soil. The soil chosen for the model run is the Fayette Series, which comprises fine-silty, mixed, mesic type Hapludalfs. It is further described as follows (USDA, 1981b):

> The Fayette series consists of well drained, moderately permeable soils on loess-covered uplands. These soils formed in loess that is more than 40 inches thick. Slope ranges from 2 to 40 percent.
>
> The solum ranges from 40 to 60 inches in thickness. There are no carbonates to a depth of 40 inches to 60 inches.

Fayette soils are of hydrologic group B, characterized by moderately low runoff potential, moderate infiltration rates, and moderate rates of water transmission.

5.3.2. Narrative Climatologic Summary. Because a meteorologic report for Clinton County was not included in NOAA (1981), the climatologic summary and data reported for nearby Dubuque, IA, (NOAA, 1981) were used:

> The principal feature of the climate in Dubuque is its variety. Standing, as it does, at the crossroads of the various air masses that cross the continent, the Dubuque area is subject to weather ranging from that of the cold, dry, arctic air masses in the winter with readings as low as 32° below [-36°C], when the ground is snow covered, to the hot, dry weather of the air masses from the desert southwest in the summer when the temperatures reach as high as 110° [43°C]. More often the area is covered by mild Pacific air that has lost considerable moisture in crossing the mountains far to the west, or by cool, dry Canadian air, or by warm, moist air from the Gulf regions. Most of the year the latter three types of air masses dominate Dubuque weather, with the invasions of Gulf air rarely occurring in the winter.
>
> The seasons vary widely from year to year at Dubuque; for example, successive invasions of cold air from the north may just reach this far one winter and bring a long, cold winter with snow-covered ground from mid-November until March, and many days of sub-zero temperatures, while another season the cold air may not reach quite this far and the winter can be mild with bare ground most of the season, and only a few sub-zero readings. The summers, too, may vary from hot and humid with considerable thunderstorm activity when the Gulf air prevails, to relatively cool, dry weather when air of northerly origin dominates the season.

5.3.3. Temperature. The monthly average temperatures at this location ranged between a low of -7.5° C and a high of 23.2° C.

5.3.4. Rainfall. Times, duration and total rainfall for each rainfall event were constructed from the rainfall record for 1980 (NOAA, 1981). The resulting rainfall parameters were as follows:

Event No.	START (hr)	PDUR (hr)	PTOT (cm)
1	180	8	2.84
2	231	6	1.0
3	396	8	1.6
4	636	6	1.2
5	970	6	1.0
6	1264	8	1.27
7	1791	10	6.12
8	1834	10	4.56
9	1934	6	3.0
10	2080	4	2.5

5.3.5. Parameters for Subroutine RAINS. Parameters for Subroutine RAINS were modified to describe soil conditions for Clinton County, IA, and rainfall for Dubuque, IA, the nearest reporting station. The slope value used in the model run was 4.6° (8%). The resulting values were:

Parameter No.	Name	PRACTICE NUMBER I	II	III
4	BTLAG	0.17	0.26	0.26
5	CN	78	61	61
6	AMC	3 (<TCROP) 2 (≥TCROP)	2	2
7	STAD	0.375	0.375	0.375
8	USLEK	0.37	0.37	0.37
9	USLEL	4.76	4.76	4.76
10	USLES	0.85	0.85	0.85
11	USLEC	0.45 (<TCROP) 0.30 (≥TCROP)	0.05	0.05

5.4. SITE 4: HIGHLANDS COUNTY, FL

Values for site-specific variables for Site 4 were chosen to represent a sandy soil in central FL. These soils can be productive for agriculture but can be improved greatly by amendment.

5.4.1. Description of Soil. The soil chosen for the model run is the Archbold Series, which comprises hyperthermic, uncoated Typic Quartzipsomments. It is further described as follows (USDA, 1989):

> The Archbold series consists of nearly level to gently sloping, moderately well drained, droughty soils that formed in marine and eolian deposits. These soils are on moderately high ridges in the ridge part of the county. The slopes range from 0 to 5 percent.
>
> Typically, the surface layer is gray sand about 4 inches thick. The underlying material to a depth of 80 inches or more is white sand.
>
> The soil reaction is slightly acid to extremely acid. The texture is sand or fine sand. The content of silt plus clay in the 10- to 40-inch control section is less than 2 percent.

Archbold soils are of hydrologic group A, characterized by having a high infiltration rate (low runoff potential) when thoroughly wet. They consist mainly of deep, well drained to excessively drained sands or gravelly sands. These soils have a high rate of water transmission.

5.4.2. Narrative Climatologic Summary. Because meteorologic information was not given in NOAA (1981) for Highlands County, the summary and data for nearby Orlando, FL, were used.

> Orlando, by virtue of its location in the central section of the Florida peninsula (which is abounding with lakes), is almost surrounded by water and, therefore, relative humidities remain high here the year round, with values hovering near 90 percent at night and dipping to 40 to 50 percent in the afternoon (sometimes to 20 percent in the winter).
>
> The rainy season extends from June through September (sometimes through October when tropical storms are near). During this period, scattered afternoon thundershowers are an almost daily occurrence, and these bring a drop in temperature to make the climate bearable (although, most summers, temperatures above 95° [35°C] are rather rare). Too, a breeze is usually present, and this also contributes towards general comfort.

5.4.3. Temperature. The monthly average temperatures at this location ranged between a low of 15.8° C and a high of 28.0° C.

5.4.4. Rainfall. Times, duration and total rainfall for each rainfall event were constructed from the rainfall record for 1980 at Orlando (NOAA, 1981). The resulting parameters were as follows:

Event No.	START (hr)	PDU (hr)	PTOT (cm)
1	1043	7	2.1
2	1166	8	3.12
3	1667	10	11.17
4	1789	9	5.7
5	1958	6	3.0
6	2536	10	3.17
7	2918	6	2.0
8	3301	6	1.6
9	3547	7	2.44
10	4025	10	9.93

In model runs from Practice I, Subroutine RAINS returned a floating-point error during computations for rainfall event 9. This error did not occur for Practices II and III, so it was probably related to both the number of organisms and the long time over which the subroutine operated. To complete the model run, it was necessary to delete rainfall events 9 and 10 for Practice I.

5.4.5. Parameters for Subroutine RAINS. Parameters for Subroutine RAINS were modified to describe local rainfall reported for Orlando, FL, and soil conditions for Highlands County, FL. The slope value used in the model run was 1.2° (2%). The resulting values were:

Parameter No.	Name	PRACTICE NUMBER I	II	III
4	BTLAG	0.45	0.8	0.8
5	CN	67	39	39
7	STAD	0.2	0.2	0.2
8	USLEK	0.1	0.1	0.1
9	USLEL	2.54	2.54	2.54
10	USLES	0.26	0.26	0.26
11	USLEC	0.45 (<TCROP) 0.25 (≥TCROP)	0.05	0.05

5.5. SITE 5: KERN COUNTY, CA

Values for site-specific variables for Site 5 were chosen to represent a soil near Bakersfield, CA, which is located in southern CA.

5.5.1. Description of Soil. The soil chosen for the model run is the Arvin series, which comprises coarse-loamy, mixed, nonacid, thermic Mollic Xerofluvents. It is further described as follows (USDA, 1981c):

> The Arvin series consist of very deep, well drained soils on alluvial fan, stream flood plains, and stream terraces. These soils formed in mixed alluvium derived from granitic rock. Slope ranges from 2 to 9 percent.
>
> Clay content ranges from 5 to 18 percent in the control section. Organic matter content is 0.9 percent or less. Reaction is slightly acid to mildly alkaline throughout.

Arvin soils are of hydrologic group B, characterized by moderately low runoff potential, moderate infiltration rates and moderate rates of water transmission.

5.5.2. Narrative Climatologic Summary.

> Bakersfield, situated in the extreme south end of the great San Joaquin Valley, is partially surrounded by a horseshoe-shaped rim of mountains with an open side to the northwest and the crest at an average distance of 40 miles.
>
> The Sierra Nevadas to the northeast shut out most of the cold air that flows southward over the continent during winter. They also catch and store snow, which provides irrigation water for use during the dry months. The Tehachapi Mountains, forming the southern boundary, act as an obstruction to northwest wind, causing heavier precipitation on the windward slopes, high wind velocity over the ridges and, at times, prevailing cloudiness in the south end of the valley after skies have cleared elsewhere. To the west are the coast ranges, and the ocean shore lies at a distance of 75 to 100 miles.
>
> Because of the nature of the surrounding topography, there are large climatic variations within relatively short distances. These zones of variation may be classified as Valley, Mountain, and Desert areas. The overall climate, however, is warm and semi-arid. There is only one wet season during the year, as 90 percent of all precipitation falls from October through April, inclusive. Snow in the valley is infrequent, with only a trace occurring in

about one year out of seven. Thunderstorms also seldom occur in the valley (NOAA, 1981).

5.5.3. Temperature. The monthly average temperatures at this location ranged between a low of 8.5° C and a high of 28.8° C.

5.5.4. Rainfall. Times, duration and total rainfall for each rainfall event were constructed from the rainfall record for 1980 at the location (NOAA, 1981). The resulting parameters were as follows:

Event No.	START (hr)	PDUR (hr)	PTOT (cm)
1	16	8	1.0
2	4378	10	1.78
3	7256	9	1.47
4	7530	6	1.07
5	8016	6	1.0
6	8320	5	1.0
7	8606	8	1.63
8	8782	8	1.0
9	13164	10	1.78
10	16022	9	1.47

5.5.5. Parameters for Subroutine RAINS. Parameters for Subroutine RAINS were modified to describe local rainfall and soil conditions. The slope value used in the model run was 1.7° (3%). The resulting values were:

Parameter No.	Name	PRACTICE NUMBER I	II	III
4	BTLAG	0.3	0.45	0.45
5	CN	78	61	61
6	AMC	3 (<TCROP) 2 (≥TCROP)	2	2
7	STAD	0.4	0.4	
8	USLEK	0.32	0.32	0.32
9	USLEL	2.54	2.54	2.54
10	USLES	0.26	0.26	0.26
11	USLEC	0.45 (<TCROP) 0.25 (≥TCROP)	0.05	0.05

5.6. SITE 6: YAKIMA COUNTY, WA

Values for site-specific variables for Site 6 were chosen to represent a soil near Yakima, WA, which is located in south-central WA along the Yakima River. This is a region of fairly low rainfall, but which is successfully farmed by irrigation.

5.6.1. Description of Soil. The soil chosen for the model run is the Kittitas Series, which comprises fine-silty, mixed (calcareous), mesic Fluvaquentic Haplaquolls. It is further described as follows (USDA, 1985):

> The Kittitas series consists of very deep, somewhat poorly drained soils on flood plains. These soils formed in mixed alluvium. Slopes range from 0 to 2 percent.

Kittitas soils are of hydrologic group C, characterized by a slow infiltration rate when thoroughly wet. They consist chiefly of soils having a layer that impedes the downward movement of water or soils of moderately fine texture or fine texture. These soils have a slow rate of water transmission.

5.6.2. Narrative Climatologic Summary.

> Yakima is located in a small east-west valley in the upper (northwestern) part of the irrigated Yakima Valley. Local topography is complex with a number of minor valleys and ridges giving a local relief of as much as 500 feet. This complex topography results in marked variations in air drainage, winds, and minimum temperatures within short distances.
>
> The climate of the Yakima Valley is relatively mild and dry. It has characteristics of both maritime and continental climates, modified by the Cascade and the Rocky Mountains, respectively. Summers are dry and rather hot, and winters cool with only light snowfall. The maritime influence is strongest in winter when the prevailing westerlies are the strongest and most steady. The Selkirk and Rocky Mountains in British Columbia and Idaho shield the area from most of the very cold air masses that sweep down from Canada into the Great Plains and eastern United States. Sometimes a strong polar high pressure area over western Canada will occur at the same time that a low pressure area covers the southwestern United States. On these occasions, the cold arctic air will pour through the passes and down the river valleys of British Columbia, bringing very cold temperatures to Yakima. That this happens infrequently is shown by the occurrence of temperatures of 0 degrees [F] or below on only 4 days a winter on the average. On about 21 days during the winter the temperature will fail to rise to the freezing point. In January and February 1950, there were 4 consecutive days colder than -20°

[-29°C], including -25° [-32°C] on February 1. However, over one-half of the winters remain above 0 degrees [F (-18°C)] (NOAA, 1981).

5.6.3. Temperature. The monthly average temperatures at this location ranged between a low of -1.5° C and a high of 22.3° C.

5.6.4. Rainfall. Times, duration and total rainfall for each rainfall event were constructed from the rainfall record for 1980 at the location (NOAA, 1981). The resulting parameters were:

Event No.	START (hr)	PDUR (hr)	PTOT (cm)
1	1628	6	1.0
2	4290	8	1.25
3	4506	10	2.06
4	5490	6	1.14
5	5722	6	1.0
6	5966	6	1.0
7	7002	10	2.65
8	7212	8	1.5
9	7498	8	1.2
10	7816	8	1.0

5.6.5. Parameters for Subroutine RAINS. Parameters for Subroutine RAINS were modified to describe local rainfall and soil conditions. The slope value used in the model run was 0.6° (1%). The resulting values were:

Parameter No.	Name	PRACTICE NUMBER I	II	III
4	BTLAG	0.4	0.5	0.5
5	CN	85	74	74
6	AMC	2	2	
7	STAD	0.4	0.4	
8	USLEK	0.43	0.43	0.43
9	USLEL	2.54	2.54	2.54
10	USLES	0.12	0.12	0.12
11	USLEC	0.45 (<TCROP) 0.25 (≥TCROP)	0.05	0.05

6. RESULTS

6.1. ALGORITHM FOR INFECTIVE DOSE

During the development of the algorithm for infection, it was assumed that the probability of infection could be described by the Poisson distribution (U.S. EPA, 1989a). According to this assumption, the probability of infection with a pathogen (whose infectious dose is M) is given by the sum of the probabilities of being exposed to M or more pathogens. While it is not feasible to calculate this number directly, an indirect method of calculation is well suited to the computer. Briefly, when the average exposure level is X, the probability of being exposed to N pathogens is given by the Poisson distribution:

$$P(N) = e^{-X}X^N/N!$$

In the exposure algorithm, the value of e^{-X} is found and multiplied by successive values of X/N as N is incremented from 1 to M-1. All values of P(N) for N<M (the MID) are summed to find the probability of not being infected, and the probability of infection is calculated as 1 minus that value, or 1-P(N).

If the infection algorithm is valid, it should generate data that would be consistent with standard methods for determining infective dose, e.g., the graphical determination of ID_{50}. To determine ID_{50}, various doses of the pathogen are administered to a population of test subjects and the number of infected and uninfected individuals at each dose is tabulated. For each dose, the number of individuals infected at that dose and higher is determined (INF_n), the number of individuals uninfected at that dose and lower is determined ($UNINF_n$), and the infection ratio $INF_n/(INF_n + UNINF_n)$ is calculated. The infection ratio is plotted for each dose, and the dose required for an infection ratio of 0.5 is interpolated graphically or calculated from doses yielding ratios just above and just below 0.5.

The infection algorithm of the model can be used to generate probabilities of infection and non-infection at various doses for a given MID. These values were generated, tabulated, and summed as described above, and the results were plotted as shown in Figure 6-1. It can be seen from this figure that the dose required for a 50% probability of infection corresponds almost exactly with the assigned MID for each value tested.

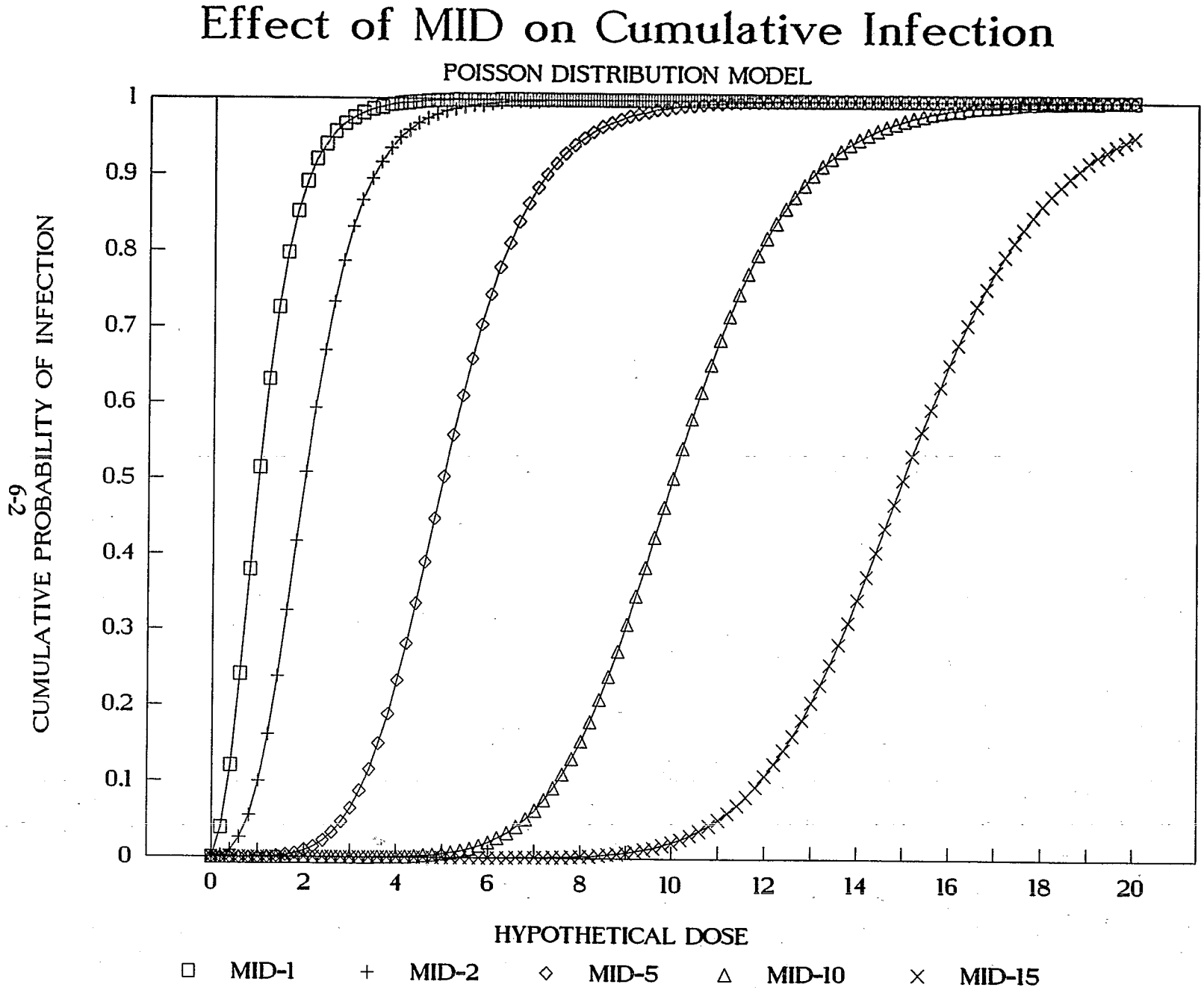
Effect of MID on Cumulative Infection
POISSON DISTRIBUTION MODEL
CUMULATIVE PROBABILITY OF INFECTION
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0
2
4
6
8
10
12
14
16
18
20
HYPOTHETICAL DOSE
MID-1
MID-2
MID-5
MID-10
MID-15

FIGURE 6-1

The model algorithm also exhibited the expected behavior with a simulated heterogeneous receptor population. It is expected that a population that is heterogeneous in its sensitivity to a pathogen will be described by a more disperse infection response than a homogeneous population, that is, the curve will rise less steeply and cover a wider range of exposure values. To test the applicability of the model's infection algorithm to this assumption, a uniform population with an MID of 10 was compared with a heterogeneous population in which the mean MID was 10. Assuming a standard deviation of 2.0 and using for guidance a table of areas of the normal distribution, the calculated distribution of MID values was: 5 and 15, 1% each; 6 and 14, 2.8% each; 7 and 13, 6.5% each; 8 and 12, 12.1% each; 9 and 11, 17.5% each; and 10, 19.7%. The infectivity ratio at each modeled dose was determined for uniform populations with these MID values, and the ratios were weighted by the population distribution and summed to yield the more disperse curve shown in Figure 6-2. This figure demonstrates that the model's infection algorithm is responsive to heterogeneity of sensitivity in the hypothetical exposed population. The disperse curve is not markedly different from the curve for a uniform population, but the entire range of sensitivity was ±50% of the MID. The range of sensitivity of >75% of the population was only ±20% of the MID, whereas the variability of sensitivity to pathogens in a typical human population is likely to be at least a few orders of magnitude. While the model could be revised to include an allowance for heterogeneity in the receptor population, such a revision is not likely to improve the accuracy of the model significantly.

6.2. SENSITIVITY TO VARIABLES

The effect of specific parameter values was tested by varying each parameter singly, using the parameter values described in Chapter 4. This required a method to compare the outcome of many model runs and express the comparison in quantitative terms. No specific day after application can be used as a good indicator of the probability of infection. The model assumes that the impact of each day's exposure is independent of any other day's exposure, despite the fact that infectious viruses could persist and accumulate, or that chronic exposure at low levels could induce immunity. The time course of exposures could be different in each compartment because of the timing of transfers of pathogens. As an

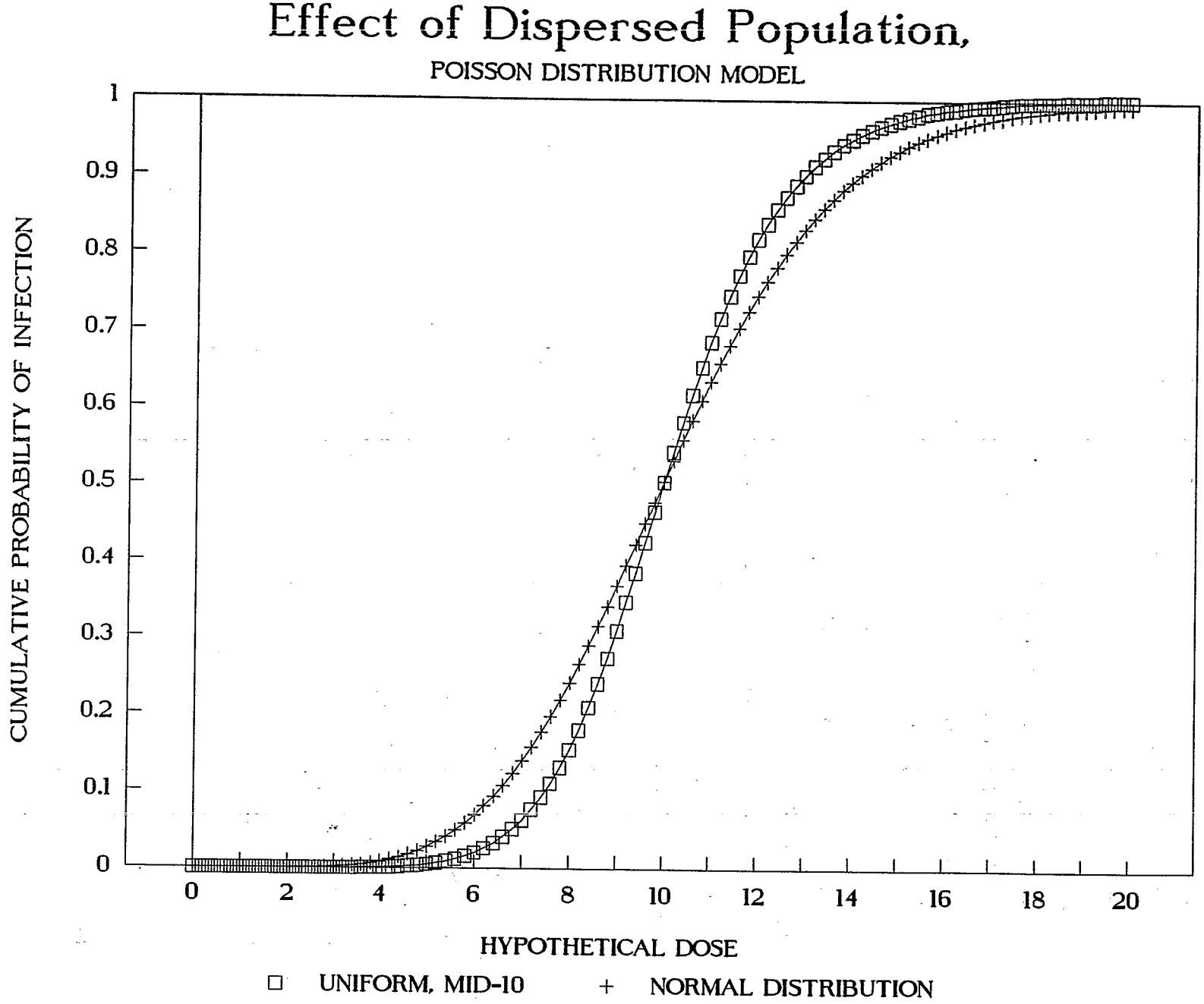
Effect of Dispersed Population,
POISSON DISTRIBUTION MODEL
CUMULATIVE PROBABILITY OF INFECTION
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0
2
4
6
8
10
12
14
16
18
20
HYPOTHETICAL DOSE
□ UNIFORM, MID-10
+ NORMAL DISTRIBUTION

FIGURE 6-2

example, in the ONSITE compartment, the maximum exposure to viruses in soil occurs immediately after incorporation. The greatest exposure to groundwater (DRINKER) depends on the rate of subsurface transport, the distance to the well, and the rate of inactivation of the viruses. Transport by surface runoff to the onsite pond (SWIMMER) cannot occur without rainfall, which determines the timing of the transfer; other parameters modify the amount of transport. Since no specific day after application can be used as a good indicator of the probability of infection, for each model run the maximum probability of infection in each exposure compartment was chosen as an appropriate indicator.

In a preliminary assessment of risk from viruses in sewage sludge, site-specific data for Site 1 and the ranges of parameters listed in Chapter 4 were tested. Approximately 1200 model runs were made, and the effects on maximum probability of infection were determined. The maximum observed probabilities of infection in each practice at Site 1, using baseline parameters, are given in Table 6-1. This table shows that the maximum probability of infection ONSITE was similar for all practices, between 1% and 7%. The probability of infection to the OFFSITE receptor was calculated as zero in every case. Risk of infection via contaminated food products (EATER) was shown only in Practice IV, and risk via offsite wellwater (DRINKER) was shown in Practice III. In contrast, infection by contact with onsite surface water (SWIMMER) was significant in all three practices that include the pond (Practices I-III), the risk level depending on site-specific as well as practice-specific variables.

In an additional detailed assessment, the inactivation rate parameters were altered as described in Section 4.2.1, and an additional set of model runs was made using site-specific data for Site 2. The parameters used as a baseline for comparisons were the default values, except that the fractional transfer from soil surface to aboveground crops [FCROP1, P(46)] was increased to 5×10^{-6}, the time at which the crop is present [TCROP, P(68)] was reduced to 240 hours, and the time of harvesting [THARV, P(69)] was reduced to 300 hours.

On the basis of these model runs, parameters showing no effect on maximum probability of infection were eliminated from further consideration. As reported for parasites (U.S. EPA, 1990) and bacterial pathogens (U.S. EPA, 1991), a large number of the

TABLE 6-1

Results of Model Runs, Baseline Conditions

Practice	Maximum Probability of Infection				
	ONSITE	OFFSITE	EATER	DRINKER	SWIMMER
I	7.02×10^{-2}	0.00	0.00	0.00	2.39×10^{-2}
II	1.10×10^{-2}	0.00	0.00	0.00	4.13×10^{-3}
III	1.10×10^{-2}	0.00	0.00	1.32×10^{-7}	4.14×10^{-3}
IV	5.54×10^{-2}	0.00	1.49×10^{-2}	--	--
V	4.67×10^{-2}	0.00	--	--	--

parameters had no effect on the maximum calculated probability of infection. Increasing the fraction of viruses transferred from surface soil to another compartment would not be expected to affect the maximum probability of infection ONSITE if the transfer occurred late in the model run, because the maximum probability of infection ONSITE typically occurs within the first few days after sludge application. Similarly in other exposure pathways, changes in the fraction of viruses transferred would have no effect on the probability of infection if the transfer occurred after the time of maximum infection and inactivation had reduced the virus population more than the transfer increased it. In some cases, the altered parameters governed processes that were insignificant compared to the main determinants of exposure, and so were not able to change the maximum probability of infection. In other cases, the exposure in a given compartment was so small that changing the time or amount of virus transfer to or from that compartment had no effect on the probability of infection.

The effects of site-specific and practice-specific differences in parameters and assumptions are illustrated by comparing the outcome of baseline model runs. The final set of model runs, in which inactivation rates were decreased in soil, water, and droplet aerosols, showed higher probabilities of infection at all sites and for most exposure compartments. The results of these model runs, using baseline parameters except for the more conservative inactivation rates, are summarized in Table 6-2. The maximum calculated probabilities of infection ONSITE were similar for each site, and again no OFFSITE infection was predicted. Infection via contaminated food products was calculated to be significant only in Practices I and IV, whereas infection via contaminated wellwater was indicated in Practices I-III at all sites. Infection to the SWIMMER was predicted at significantly higher levels than with the default inactivation parameters. The time course of infection probability in each exposure compartment in Practice I (Site 1) is illustrated in Figure 6-3.

The probability of infection by consumption of crops was proportional to the concentration of viruses in the applied sludge, but decreasing the concentration yielded a less than proportional decrease in probability of infection ONSITE and to the SWIMMER in the onsite pond. For example, reducing the concentration of viruses in sludge by a factor

TABLE 6-2

Maximum Probability of Infection
by Site and Practice

SITE	PRACTICE	ONSITE	OFFSITE	EATER	DRINKER	SWIMMER
1	I	$2.70x10^{-1}$	0.0	$5.58x10^{-4}$	$2.71x10^{-6}$	$7.40x10^{-1}$
	II	$4.63x10^{-2}$	0.0	0.0	$3.21x10^{-6}$	$2.10x10^{-1}$
	III	$4.63x10^{-2}$	0.0	0.0	$6.44x10^{-6}$	$2.12x10^{-1}$
	IV	$5.54x10^{-3}$	0.0	$9.27x10^{-1}$	0.0	0.0
	V	$2.79x10^{-3}$	0.0	0.0	0.0	0.0
2	I	$2.70x10^{-1}$	0.0	$5.11x10^{-4}$	$2.80x10^{-6}$	$1.94x10^{-1}$
	II	$4.63x10^{-2}$	0.0	0.0	$3.20x10^{-6}$	$2.88x10^{-2}$
	III	$4.63x10^{-2}$	0.0	0.0	$6.41x10^{-6}$	$2.90x10^{-2}$
	IV	$5.53x10^{-3}$	0.0	$9.28x10^{-1}$	0.0	0.0
	V	$2.79x10^{-3}$	0.0	0.0	0.0	0.0
3	I	$2.70x10^{-1}$	0.0	$5.61x10^{-4}$	$4.28x10^{-6}$	$8.03x10^{-1}$
	II	$4.63x10^{-2}$	0.0	0.0	$5.04x10^{-6}$	$3.15x10^{-1}$
	III	$4.63x10^{-2}$	0.0	0.0	$1.01x10^{-5}$	$3.15x10^{-1}$
	IV	$5.54x10^{-3}$	0.0	$9.28x10^{-1}$	0.0	0.0
	V	$2.79x10^{-3}$	0.0	0.0	0.0	0.0
4	I	$2.70x10^{-1}$	0.0	$5.11x10^{-4}$	$2.81x10^{-6}$	$3.71x10^{-3}$
	II	$4.63x10^{-2}$	0.0	0.0	$3.20x10^{-6}$	$8.03x10^{-4}$
	III	$4.63x10^{-2}$	0.0	0.0	$6.42x10^{-6}$	$8.07x10^{-4}$
	IV	$5.54x10^{-3}$	0.0	$9.25x10^{-1}$	0.0	0.0
	V	$2.79x10^{-3}$	0.0	0.0	0.0	0.0
5	I	$2.70x10^{-1}$	0.0	$5.11x10^{-4}$	$2.00x10^{-6}$	0.0
	II	$4.63x10^{-2}$	0.0	0.0	$3.20x10^{-6}$	$4.73x10^{-2}$
	III	$4.63x10^{-2}$	0.0	0.0	$6.42x10^{-6}$	4.73x10-2
	IV	$5.54x10^{-3}$	0.0	$9.25x10^{-1}$	0.0	0.0
	V	$2.79x10^{-3}$	0.0	0.0	0.0	0.0
6	I	$2.70x10^{-1}$	0.0	$5.11x10^{-4}$	$2.81x10^{-6}$	$3.15x10^{-5}$
	II	$4.63x10^{-2}$	0.0	0.0	$3.20x10^{-6}$	$4.93x10^{-6}$
	III	$4.63x10^{-2}$	0.0	0.0	$6.42x10^{-6}$	$4.94x10^{-6}$
	IV	$5.54x10^{-3}$	0.0	$9.25x10^{-1}$	0.0	0.0
	V	$2.79x10^{-3}$	0.0	0.0	0.0	0.0

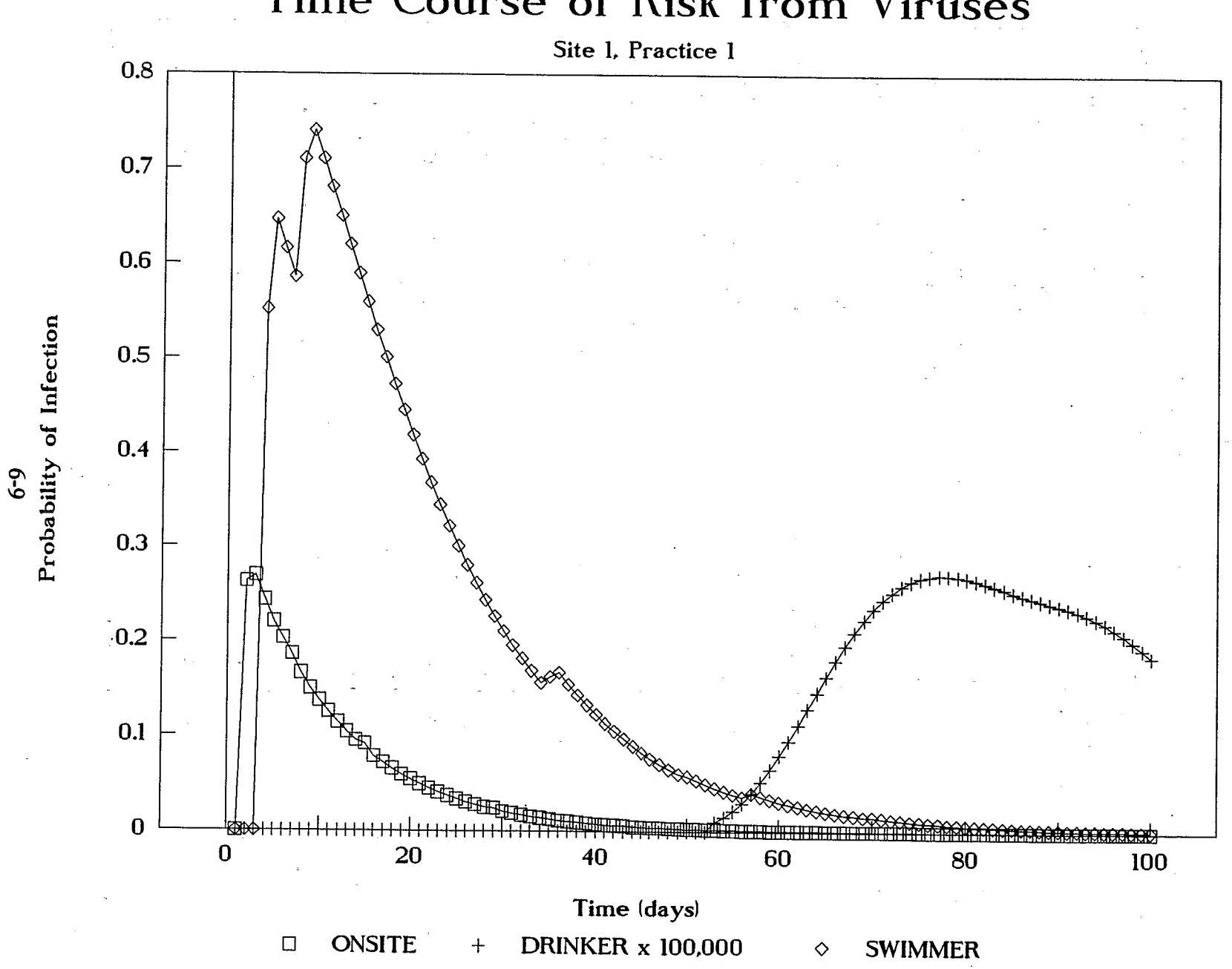
Time Course of Risk from Viruses
Site 1, Practice 1
Probability of Infection
6-9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0
20
40
60
80
100
Time (days)
ONSITE
DRINKER x 100,000
SWIMMER

FIGURE 6-3

of 200 (from 100,000/kg to 500/kg) reduced the probability of infection via crops from 5.11×10^{-4} to 2.55×10^{-6} (also a factor of 200), whereas ONSITE infection was reduced from 0.27 to 0.0016 (a factor of 172), and infection to the SWIMMER was reduced from 0.194 to 0.0011 (a factor of 180). This is consistent with the conclusions for infection by parasites (U.S. EPA, 1990). When the infectious dose is 1, the probability of infection is proportional to dose at low doses. However, because of the exponential basis of the Poisson distribution, as the dose increases the probability of infection increases less rapidly.

To test the effect of the rate of loss of infectivity or viability, a constant logarithmic inactivation rate was substituted for the temperature-dependent inactivation rates described in Table 4-1. The results for Practice III (and for EATER in Practice IV) at Site 1 are given in Table 6-3 (similar effects were seen in model runs for the other practices, but Practice III was used in this illustration because of its greater groundwater exposure). All values for inactivation rates in soil, dry particulates and water gave higher probabilities of infection than the default temperature-dependent algorithm. This suggests that the slope and intercept values used in the algorithm may be incorrect. Substitute values for inactivation of viral particles in aerosols had no effect on the probabilities of infection; the probability of infection for offsite aerosol exposure was calculated to be zero in every model run.

The effect of inactivation rate on the time course of ONSITE exposure is demonstrated in Figure 6-4. With the inactivation rates described above, the probability of infection fell rapidly as infectious particles were inactivated. However, when the inactivation rates were set to zero, the decrease in probability of infection was gradual, as virus particles in surface soil were gradually transferred to surface water, subsurface soil and groundwater. Superimposed on this pattern was a biweekly increase for one day of 4-5% in the probability of onsite infection as the farmer makes a closer inspection of the crops. Accompanying the decrease in viral particles in surface soil, there was an increase in the probability of infection to the pond swimmer as each rainfall added more runoff to the pond (Figure 6-5). At an inactivation rate of 0.0015 log/hr, the SWIMMER exposure rapidly decreased, whereas with no inactivation, the probability of infection increased to 1.0. The probability of infection by consumption of contaminated well water increased gradually, beginning 51 days after initial

TABLE 6-3

Effect of Process Functions on Probability of Infection
(Site 1, Practice III except as noted)

Run	Crop P(66)	Hourly logarithmic inactivation rate constant				Maximum Probability of Infection				
		Soil	Dry Particulates	Water	Aerosol	ONSITE	OFFSITE	EATER	DRINKER (Prac. IV)	SWIMMER
Std	1					1.10×10^{-2}	0.00	1.49×10^{-2}	1.32×10^{-7}	4.14×10^{-3}
P0001	1	-0.0005	-0.0001	-0.0003	--	5.06×10^{-2}	0.00	1.00	9.79×10^{-5}	3.40×10^{-1}
P0002	0	-0.0005	-0.0001	-0.0003	--	5.06×10^{-2}	0.00	1.00	4.54×10^{-5}	3.06×10^{-1}
P0003	-1	-0.0005	-0.0001	-0.0003	--	5.06×10^{-2}	0.00	1.00	4.54×10^{-5}	3.06×10^{-1}
P0004	1	-0.0025	-0.001	-0.002	--	4.36×10^{-2}	0.00	1.00	3.48×10^{-6}	1.60×10^{-1}
P0005	0	-0.0025	-0.001	-0.002	--	4.36×10^{-2}	0.00	1.00	3.48×10^{-6}	1.60×10^{-1}
P0006	-1	-0.0025	-0.001	-0.002	--	4.36×10^{-2}	0.00	1.00	3.48×10^{-6}	1.60×10^{-1}
P0007	1	-0.004	-0.01	-0.01	--	3.91×10^{-2}	0.00	1.00	8.95×10^{-7}	7.92×10^{-2}
P0008	0	-0.004	-0.01	-0.01	--	3.91×10^{-2}	0.00	1.00	8.95×10^{-7}	7.92×10^{-2}
P0009	-1	-0.004	-0.01	-0.01	--	3.91×10^{-2}	0.00	1.00	8.95×10^{-7}	7.92×10^{-2}
P0010	1	--	--	--	-0.0002	1.10×10^{-2}	0.00	1.51×10^{-2}	1.32×10^{-7}	4.14×10^{-3}
P0011	0	--	--	--	-0.0002	1.10×10^{-2}	0.00	1.49×10^{-2}	1.32×10^{-7}	4.14×10^{-3}
P0012	-1	--	--	--	-0.0002	1.10×10^{-2}	0.00	8.04×10^{-3}	1.32×10^{-7}	4.14×10^{-3}
P0013	1	--	--	--	-0.005	1.10×10^{-2}	0.00	1.51×10^{-2}	1.32×10^{-7}	4.14×10^{-3}
P0014	0	--	--	--	-0.005	1.10×10^{-2}	0.00	1.49×10^{-2}	1.32×10^{-7}	4.14×10^{-3}
P0015	-1	--	--	--	-0.005	1.10×10^{-2}	0.00	8.04×10^{-3}	1.32×10^{-7}	4.14×10^{-3}
P0016	1	0.00	0.00	0.00	0.00	5.62×10^{-2}	0.00	1.00	2.02×10^{-2}	7.80×10^{-1}

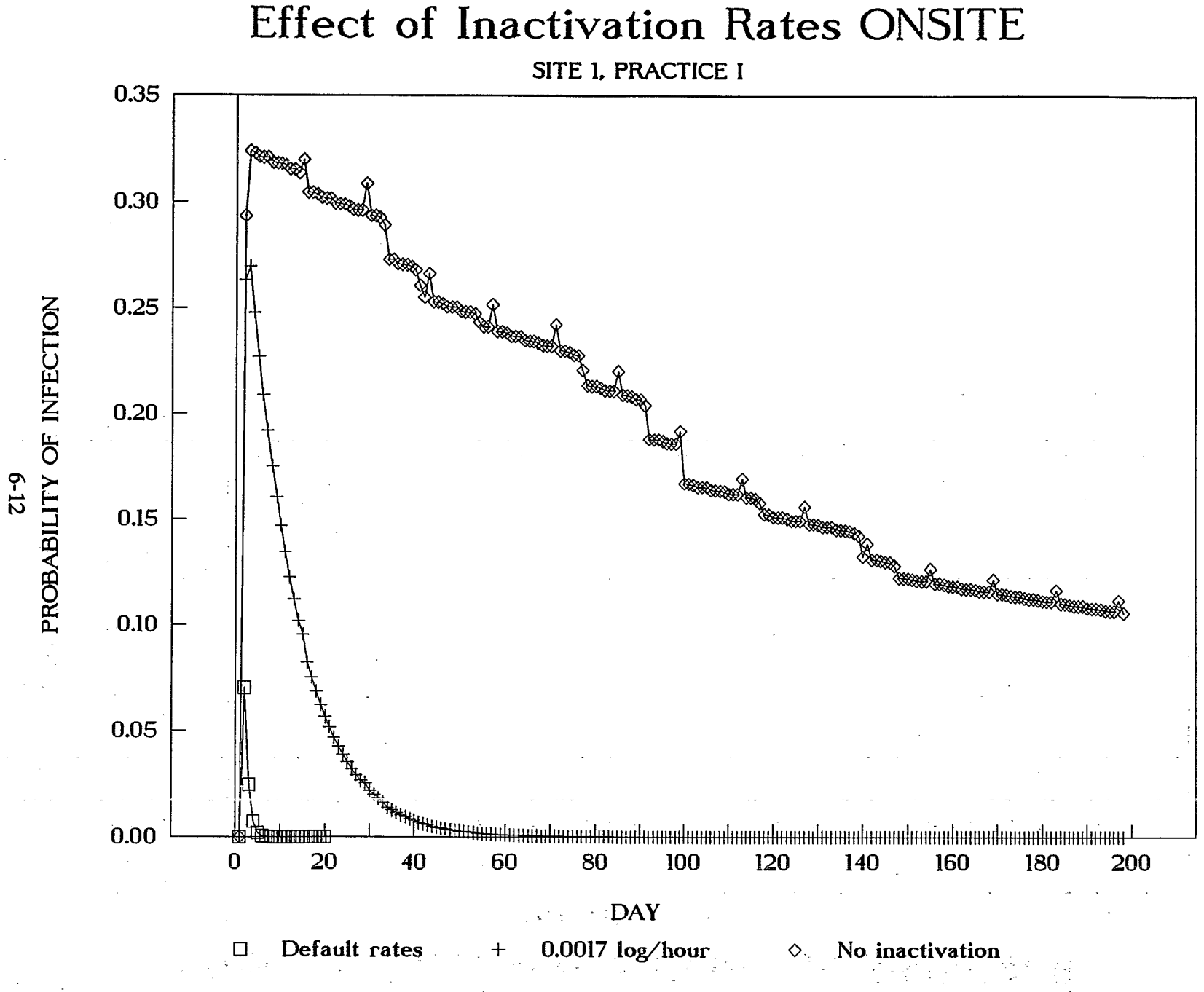
Effect of Inactivation Rates ONSITE
SITE 1, PRACTICE I
PROBABILITY OF INFECTION
0.35
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0
20
40
60
80
100
120
140
160
180
200
DAY
Default rates
0.0017 log/hour
No inactivation

FIGURE 6-4

Effect of Inactivation Rates on SWIMMER

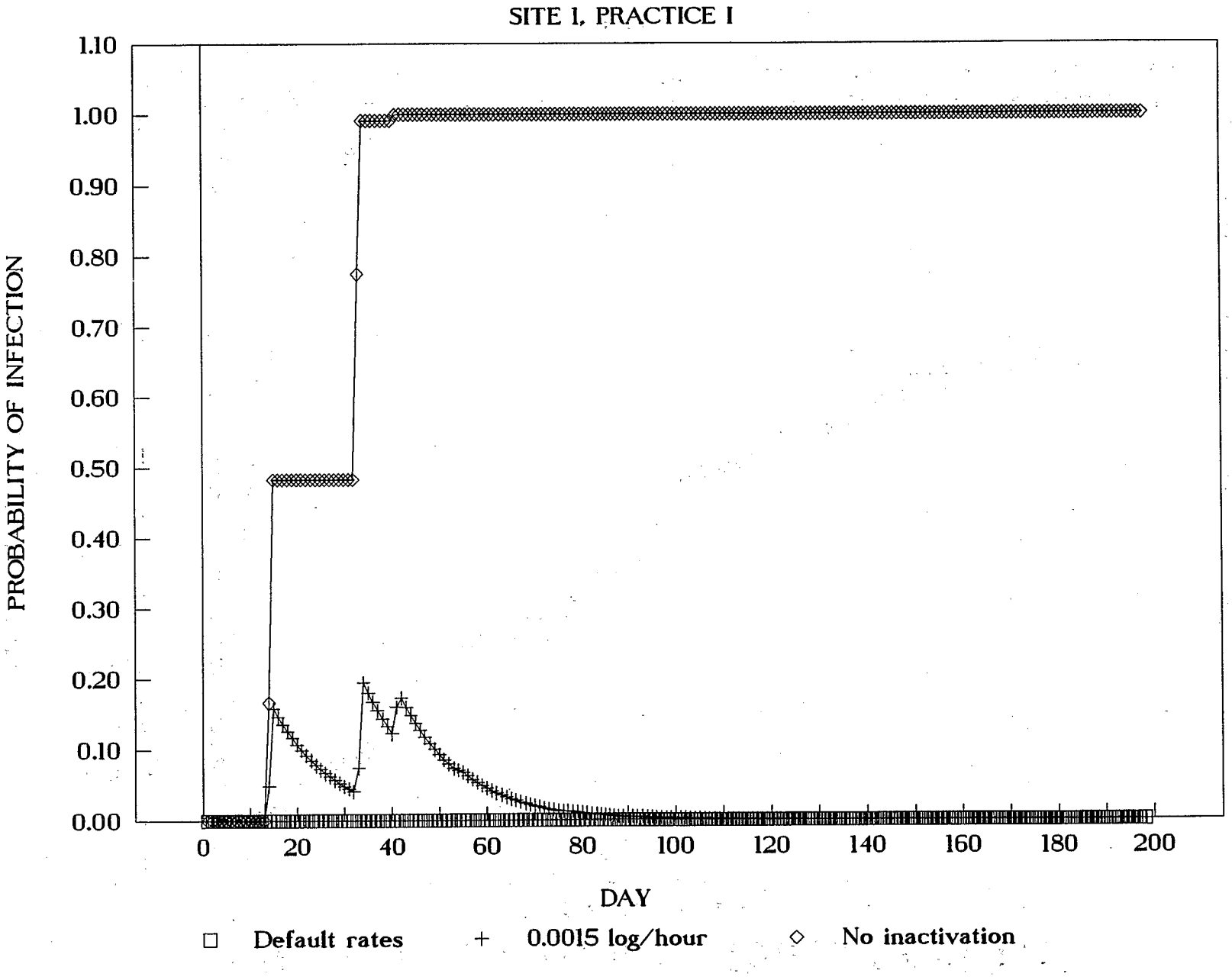

FIGURE 6-5

application of the sludge. These results show that the inactivation rate of virus particles is extremely important in determining whether a groundwater well is likely to become contaminated and in determining how long surface soil or surface water is likely to remain infectious. The results also demonstrate the importance of accurate characterization of inactivation rate for viruses of different kinds in the various transport and exposure media.

6.3. ONSITE EXPOSURES

In each model run for Practice I, in which the liquid sludge must be allowed to dry for 24 hours before it is tilled, the maximum probability of infection ONSITE occurred on day 3 and decreased as the sludge-borne viruses were inactivated and as they were transferred into other compartments. In Practices II and III, which do not require the 24-hour waiting period, the maximum probability of infection occurred on day 2. In Practice V the maximum probability of infection ONSITE occurred on day 2; in Practices IV and V, composted sludge is assumed to be spread by hand, providing a high level of exposure to the user during the application process, followed by a lower exposure as the composted sludge is incorporated and thus diluted with soil. However, exposure during subsequent tilling in Practice IV led to a maximum risk of infection at 15 days. Onsite exposures decreased rapidly as the viral particles in the soil were inactivated or transferred into other compartments. Figure 6-6 presents the time courses of ONSITE infection probabilities for Practices I-V at Site 1.

Using reference values including the conservative inactivation rates, the baseline maximum probability of infection was 0.270 for Practice I, 0.046 for Practices II and III, 0.0055 for Practice IV, and 0.0028 for Practice V. Generally, site-specific variables did not have a significant effect on the probability of ONSITE infection, because the site-specific variables alter temperature-dependent inactivation rates and rainfall-dependent runoff and sediment transport, none of which exerts major effects on the ONSITE exposure compartment before the time of maximum infection. Significant impacts on the probability of infection were observed in all application practices with changes in pathogen density in the applied sludge [ASCRS, P(1)] and sludge application rate [APRATE, P(2)], both of

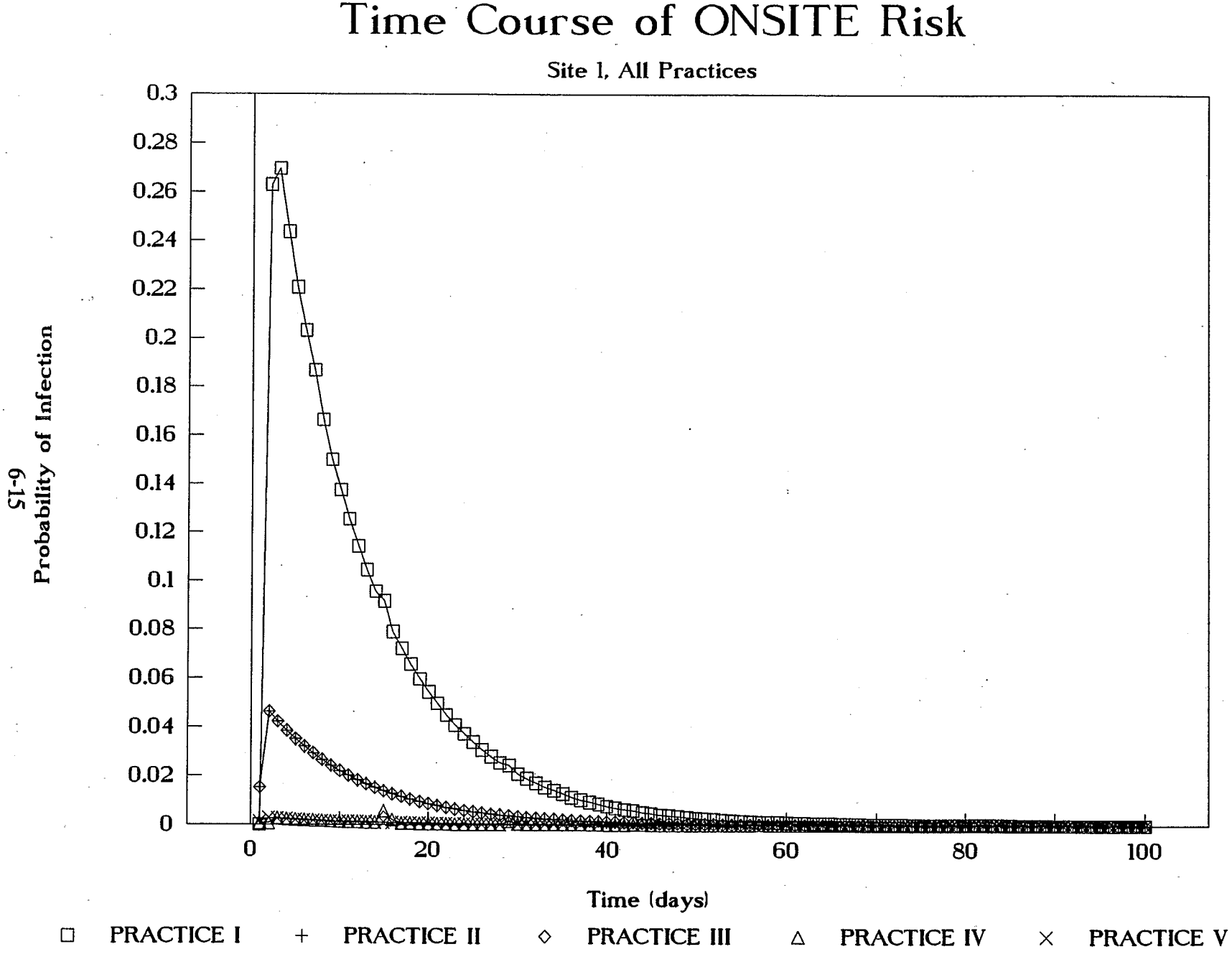
Time Course of ONSITE Risk
Site 1, All Practices
Probability of Infection
0.3
0.28
0.26
0.24
0.22
0.2
0.18
0.16
0.14
0.12
0.1
0.08
0.06
0.04
0.02
0
0
20
40
60
80
100
Time (days)
PRACTICE I
PRACTICE II
PRACTICE III
PRACTICE IV
PRACTICE V

FIGURE 6-6

which determine the number of viral particles applied. Because of the exponential nature of the probability algorithm, the changes in probability were not directly proportional to the change in parameter values, but varied as would be expected for a proportional change in exposure.

The method of application was significant in Practices I-III, subsurface application removing all probability of ONSITE infection. Increasing the infective dose [MID, P(12)] markedly reduced the probability of infection in all practices. The concentrations of particulates, as determined by the soil composition (EPSMLT [P(26)] and ESILT [P(27)]) and the height of the particulate cloud (EHT [P(28)]), had an effect on the probability of infection ONSITE. Changing the inactivation rates, either by varying the parameters SLOPES [P(37)] and NTRCPS [P(38)] or the process functions PROC1-PROC3 and HCRIT, had significant effects on the probability of infection, as illustrated by a comparison of Tables 6-1 and 6-2. The RISK parameter DRECTS, which describes the amount of soil ingested daily, also had a significant effect on probability of infection, as would be expected for a parameter directly governing exposure by contact with a contaminated medium.

6.4. OFFSITE EXPOSURE

In all model runs; the probability of infection OFFSITE was zero, indicating that although the inactivation of viruses in aerosols may be less than initially expected (compare Table 3-6 with default value of 0.002 log/sec (U.S. EPA, 1989a)), the calculated quantities of liquid and dry particulate aerosols and concentrations of viruses in the aerosols were too low to provide an infective dose to the modeled receptor.

6.5. EXPOSURE FROM CONTAMINATED FOOD

Consumption of contaminated vegetable crops was shown by model calculations to be a potential source of human infection, provided that inactivation rates were sufficiently low or harvesting times were sufficiently close to application of the sludge. The maximum probability of infection by consumption of crops in Practices I and IV at each site is shown in Table 6-2. Infection via food crops was sensitive not only to infectious dose (MID [P(12)], inactivation rates, and the parameters described in Section 6.3 that directly affect

the number of pathogens applied to the soil (ASCRS [P(1)] and APRATE [P(2)]), but also to the relative fractions of pathogens transferred among surface soil, subsurface soil, and crop surface, and to the type of crop or fraction of the total crop grown aboveground, below-ground, or on-ground (variables P(44)-P(58)). The probability of infection by consumption of vegetable crops was weakly correlated with the crop yield and strongly correlated with crop type, the fraction of garden crops as on-ground crops being most strongly positively correlated with infection and the fraction as aboveground crops being negatively correlated with infection. The first group of rows in Table 6-4 shows the effect of changes in fraction of on-ground crops when the fraction of below-ground crops was held constant. As the fraction of on-ground crops increased, the calculated probability of infection increased. In the second and third groups, the fraction of aboveground crops was held constant while the fraction of on-ground crops increased. In each case, the calculated probability of infection increased. When the fraction of on-ground crops was held constant (0.1), the calculated probability of infection increased as the fraction of below-ground crops increased. The fraction of aboveground crops had a negative influence on the probability of infection.

Contamination of meat or milk by viruses from sewage sludge did not appear to pose a significant risk to human health. No evidence was found in the literature search to substantiate infection of cattle, poultry or swine by human enteric viruses and subsequent transmission of those viruses to humans by ingestion of dairy or meat products. In addition, no condition tested resulted in a calculated probability of infection $> 1x10^{-16}$ for ingestion of meat or milk.

6.6. EXPOSURE FROM CONTAMINATED GROUNDWATER

Transport of viruses via groundwater to an offsite well was not shown by this model to be a major risk, but exposure by contaminated groundwater was shown to be likely if the rate of inactivation of viruses in water was less than the default values. The probability of infection was related to the periodic introduction of pathogens to groundwater by the infiltration of rainwater (Figure 6-7). The most important parameter related to subsurface transport of viruses appeared to be the inactivation rate of viruses in water. The results also

TABLE 6-4

Effect of Distribution of Crop Types
on Risk to Consumers of Garden Vegetables[a]

Value of Variable			Probability
PLNT1[b] P(74)	PLNT2[c] P(75)	PLNT3[d] P(76)	of EATER Infection
0.8	0.1	0.1	0.0014
0.4	0.5	0.1	0.0067
0.1	0.8	0.1	0.0108
0.4	0.1	0.5	0.0109
0.4	0.3	0.3	0.0149
0.1	0.1	0.8	0.0229
0.1	0.4	0.5	0.0417

[a]Results for Site 1, Practice IV, default inactivation rates
[b]Aboveground crops
[c]On-ground crops
[d]Below-ground crops

Time Course of Risk to DRINKER

Site 1, Practices I-III

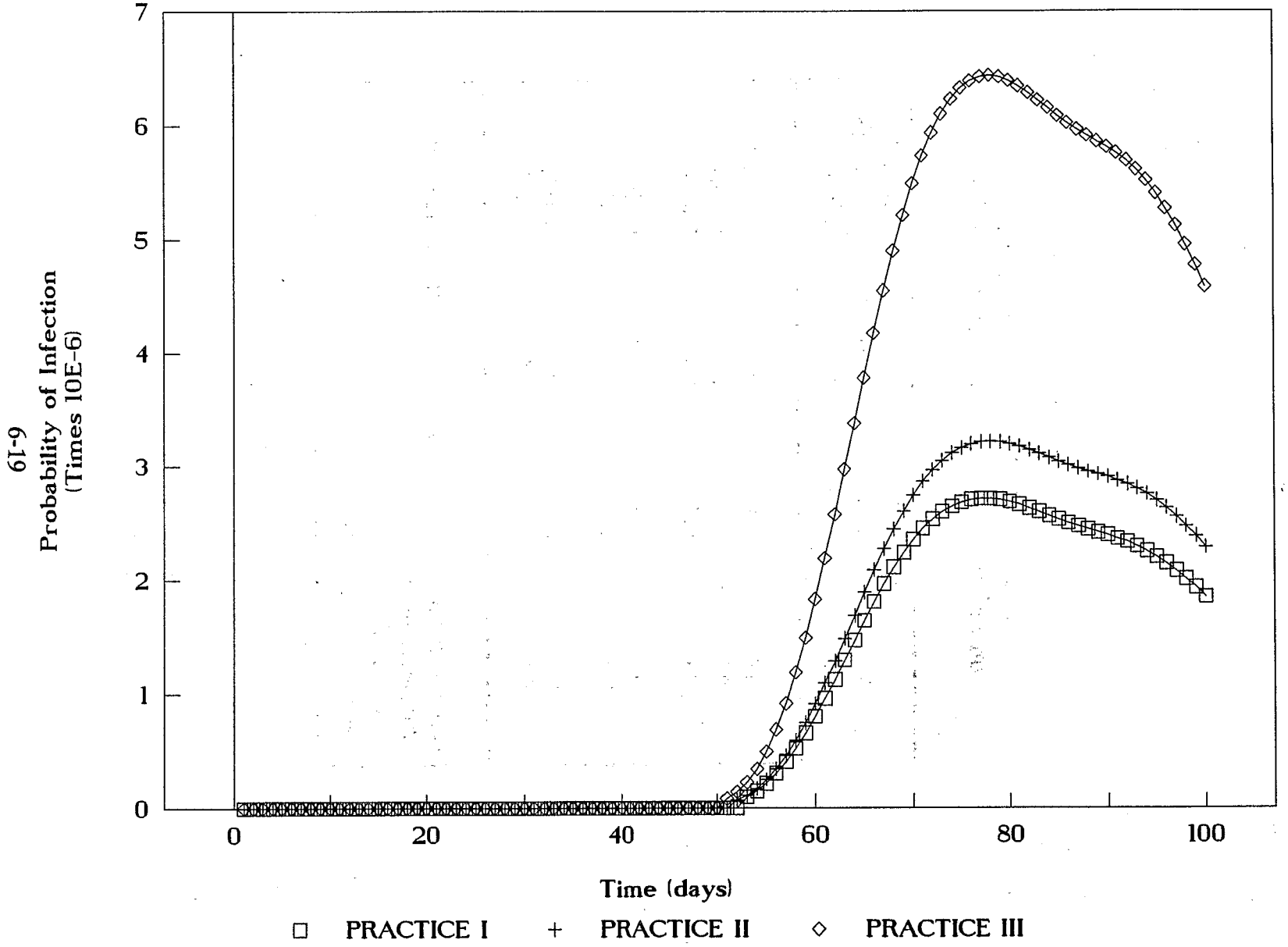

FIGURE 6-7

showed an increase in probability of infection at the offsite well whenever the time required for the viruses to reach the well was decreased. Thus, increasing groundwater velocity or reducing the dispersion or retardation coefficients or the distance to the well moderately increased the probability of infection in Practices II and III. Conversely, exposures were reduced when these parameters were changed in the opposite direction. No effect was observed in Practice I using the default inactivation rates because the concentration of viral particles entering the groundwater was too small to be significant. However, when the reduced, more conservative inactivation rates were used, the number of pathogens reaching the offsite well increased and the effects of GRDWTR parameters decreased. The effects of differences in GRDWTR parameters in model runs for Site 1 are summarized in Table 6-5.

6.7. SURFACE WATER EXPOSURE

Contaminated surface water, represented by the SWIMMER in an onsite pond, was the most significant source of exposure. A peak in probability of infection occurred after each rainfall, when additional contaminated soil surface water and soil were washed into the pond. The successive pulses of pathogens released to the onsite pond are illustrated in Figure 6-3. Site-specific parameters for Subroutine RAINS reflect differences in timing and amount of rainfall and in properties of the soil that affect surface runoff and sediment transport. Sites at which rainfall is slow and steady would be expected to have less runoff and sediment transport than sites at which rainfall is infrequent but heavy. Similarly, sites that are nearly level and have a good soil and extensive ground cover should have less runoff than steeply sloped sites with poor soil and little ground cover. A comparison of the effects of site-specific parameters for Subroutine RAINS on probability of infection are demonstrated in Figure 6-8, which presents results for Practice I at all sites. Risk of infection decreases with time before the first rainfall because of inactivation of the viral pathogens in soil. Table 6-2 summarizes the maximum risk of infection to the pond swimmer at each site for each of the three practices that includes an onsite pond.

TABLE 6-5

Effect of Variables for Subroutine GRDWTR
on Infection via Groundwater

Variable #	Name	Definition	Value	Maximum Infection Risk Probability	Day
2	V	Velocity (cm/hr)	0.9	1.20×10^{-6}	254
			3.6	2.71×10^{-6}	77
			10.8	4.15×10^{-6}	39
3	D	Dispersion coefficient	0	2.79×10^{-6}	77
			60	2.71×10^{-6}	77
			100	2.63×10^{-6}	78
4	R	Retardation coefficient	0.2	3.99×10^{-6}	31
			1.0	2.71×10^{-6}	77
			2.0	2.46×10^{-6}	139
11	XM	Maximum distance (m)	20	2.78×10^{-6}	42
			50	2.71×10^{-6}	77
			200	2.47×10^{-6}	255

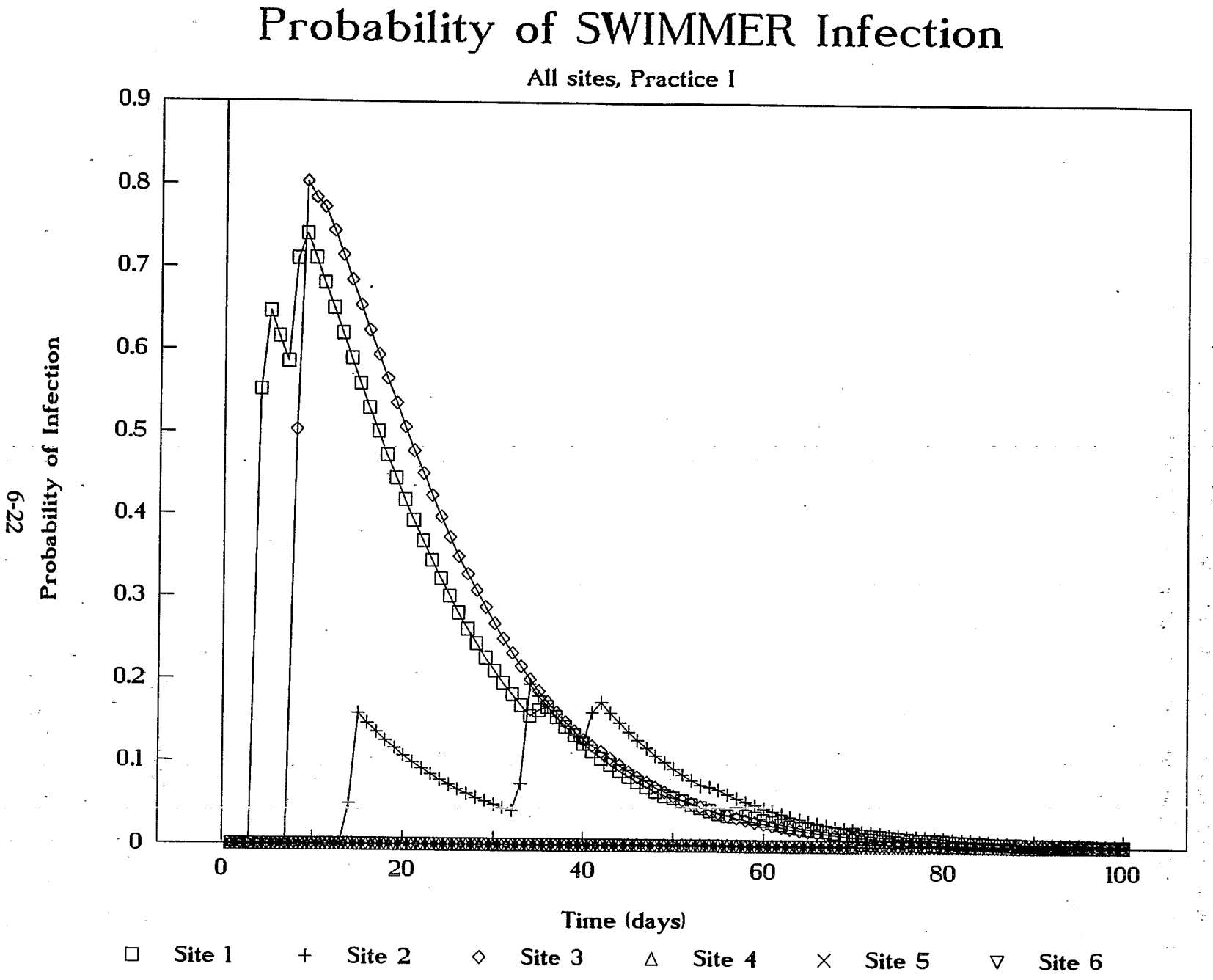
Probability of SWIMMER Infection
All sites, Practice I
Probability of Infection
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0
20
40
60
80
100
Time (days)
Site 1
Site 2
Site 3
Site 4
Site 5
Site 6

FIGURE 6-8

Practice-specific differences in runoff can also occur. Figure 6-9 shows the calculated probability of infection at Site 1 for practices I, II and III. This figure shows that the maximum probability of infection is less and the highest probability of infection occurs later in Practices II and III, in which there is more ground cover and therefore less surface runoff.

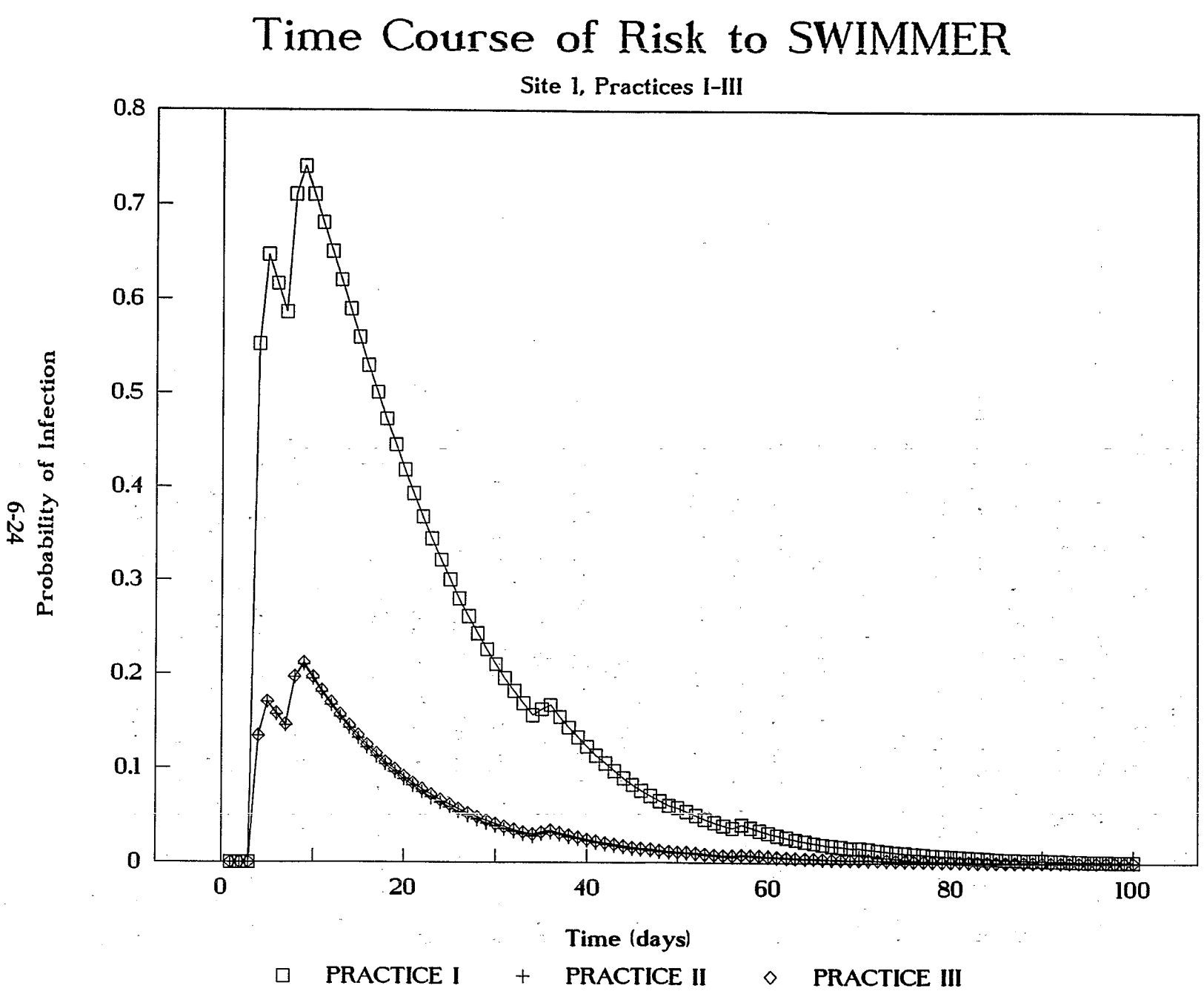
Time Course of Risk to SWIMMER
Site 1, Practices I-III
Probability of Infection
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0
20
40
60
80
100
Time (days)
PRACTICE I
PRACTICE II
PRACTICE III

FIGURE 6-9

7. CONCLUSIONS

7.1. LITERATURE REVIEW

Literature values for virus density in treated sludge were so variable, both by treatment methodology and by virus type, that no single number could be selected as typical. However, 2000 virus particles/kg was chosen as representative of viral density in composted sludge and 100,000 particles/kg in digested sludge. Infective doses, while varying by detection method and by virus type, have been reported to be as low as 1 infective particle. As a conservative assumption, this minimum value was used for the model runs. Reported inactivation rates range from 7.1×10^{-5} to 1.6×10^{-1} logs/hour in soil, 1.6×10^{-4} to 1.4×10^{-1} logs/hour in water, and 4.9×10^{-5} to 8×10^{-7} logs per second in aerosols. Like the density values, these rates are quite variable. Information on dispersion of viruses in the environment is limited in its applicability to generating a rate of transport in environmental media. Development of a variety of transport models has been an attempt to quantify the movement of viruses, especially in the subsurface and in groundwater.

7.2. MODELING RESULTS

Although detailed data on survival and transport of viruses in soil are limited, the model appears to confirm the general observations in the literature that viruses in treated sewage sludge present a potential health risk, justifying land-use restrictions. However, model runs implied that restrictions may be overly conservative. Reports of offsite infection by viruses in sludge-amended soil or in aerosols from liquid treated sludge were not found; model runs confirm the low probability of offsite infection except by uncontrolled surface runoff.

A test of the infection algorithm yielded results that are consistent with experimentally and epidemiologically observed responses of populations to pathogen exposure. The algorithm could be revised to include allowances for individual variation in sensitivity in the population, but because of the uncertainties inherent in the model and in the data used in its operation, it was concluded that the additional complexity was not warranted.

7.2.1. Sensitivity Analysis. Model runs showed that the probability of infection by viruses as a result of exposure to soil contaminated with sewage sludge is related to the concentration of organisms in the sludge, the amount of sludge applied and the amount of contaminated soil to which the individual is exposed, either by casual contact or by ingestion of food grown in the contaminated soil. The method of application was also significant. Subsurface application resulted in no exposure to any individual onsite, either by direct contact or in the onsite pond, although exposure via groundwater was not eliminated.

Direct proportionality of response to exposure level was not observed, because the probability of infection is calculated by a Poisson distribution, which is an exponential function of exposure rather than a proportional one. It was shown previously (U.S. EPA, 1990) that when the MID = 1, variations in the probability of infection can be extrapolated from variations in virus concentrations up to a probability of about 0.1. Many of the parameters of the model seemed to have little bearing on the probability of infection, apparently because they ultimately had no effect on the number of viral particles to which the human receptor was exposed in each exposure compartment, or because they exerted their effect on survival or transport after the maximum probability of infection had occurred.

The probability of infection was sensitive to the rate of inactivation of the viruses. This was the most significant property of the viruses themselves, the other most sensitive properties being practice-specific or related to host response as well.

7.2.2. Onsite Exposures. Significant onsite exposures were calculated in all practices. The greatest ONSITE risk, ~0.27 per day, was associated with Practice I, application of sludge for production of commercial crops. These calculations imply that the field worker who inadvertently ingests soil during daily activity on the sludge application site is at significant risk of infection by viruses. The results imply that there should be a waiting period before routine daily activity on the site. The length of the waiting period should depend on the initial application rate and pathogen concentration as well as the inactivation rate of the virus. A benchmark risk of $1x10^{-4}$ per day was suggested previously (U.S. EPA, 1990) on the basis of proposed U.S. EPA pathogen reduction regulations (U.S.EPA, 1989b). In the model runs reported here, the probability of infection ONSITE in Practice I, using a conservative inactivation rate, was $>1x10^{-2}$ for 37 days and $>1x10^{-4}$ for 82 days.

The maximum risks of infection calculated in domestic applications were lower (0.0055 for Practice IV and 0.0028 for Practice V), but not low enough to be protective. However, it must be noted that the concentration used in the model runs, 2500 viruses/kg, is a maximum value derived from a limit of detection reported in the literature, and lower levels may routinely be found in composted sludge. It is assumed that incorporation in these practices is done by hand or with power tools rather than by farm machinery; however, the calculated exposures do not include direct exposure to non-incorporated sludge.

In summary, it appears that a probability of infection greater than the arbitrary benchmark value of $1x10^{-4}$ is likely during application and incorporation of liquid treated sludge for agricultural practices. If the initial viral concentrations in composted sludge are >50/kg, the user is likely to be at risk of infection. A person engaged in these activities could probably reduce the risk by wearing a protective mask and washing thoroughly before handling food.

7.2.3. Sediment Transport and Surface Runoff. The most significant potential source of infection was exposure to runoff water and transported sediment after rainfall. Rainfall events were modeled as being able to transport contaminated soil from the field to the onsite pond, where the suspended or particle-bound viruses accumulated. A swimmer in the pond was therefore exposed to the viruses, by ingestion of either contaminated water or sediments. Model runs indicated that it would be prudent to limit access to runoff water and sediment from a sludge-amended field, either by mulching to reduce runoff, ditching and/or diking to contain the runoff or restricting access to any onsite ponds receiving runoff.

7.2.4. Offsite Exposures. No health hazard was indicated as a result of offsite transport of viruses by droplet aerosols or by wind-blown dust. This may be a model limitation caused by over-simplification of the Gaussian-plume aerosol transport subroutine in the model, or it may reflect a very low concentration or probability of transport of infective viruses in aerosols. Modeling results were consistent with abundant literature reports that groundwater can be a significant source of viral transport. However, because of dilution and inactivation during subsurface transport, the highest probabilities of infection predicted for consumption of groundwater at an offsite well were $<1x10^{-5}$ (Table 6-2). Variations in the parameters used for operation of the groundwater subroutine had little effect on the

maximum probability of infection when conservative inactivation rates were used, although the time at which the maximum probability was observed did depend strongly on these parameters (Table 6-5). Because infiltration to groundwater is treated by the subroutine as a point source rather than as a large area (U.S. EPA, 1989a), the subroutine probably greatly overestimates the concentration of viruses reaching the offsite well.

7.2.5. Waiting Period. Practice-specific waiting periods are proposed in the U.S. EPA Pathogen Reduction Requirements (U.S. EPA, 1989b) before access to sludge-amended land or consumption of crops grown thereon. For exposure comparisons, a probability of infection of $1x10^{-4}$ was tentatively chosen as a benchmark for sufficient protection of human health (U.S. EPA, 1990). Using this benchmark value, the default values for application rate and inactivation rate, and a virus density of $1x10^{5}$/kg in Practice I, the initial maximum probability of infection for aboveground crops harvested 13 days after sludge application was $5\text{-}6x10^{-4}$ (Table 6-2); at the conservative inactivation rate for viruses in soil, a waiting period of at least 45 days would be required to reduce the probability of infection below $1x10^{-4}$ for aboveground crops contaminated with 0.1 g soil per crop unit. A waiting period of 5 months appeared to be adequate (probability of infection $<1x10^{-16}$) for below-ground crops, whose consumption is currently forbidden for 5 years after sludge application, as well as for on-ground crops (probability of infection $3.6x10^{-7}$). In neither case did a 4-month wait appear to be adequate. In Practice IV, the calculated probability of infection by food consumption was >90% when the crops were harvested beginning at 13 days; a 4-month waiting period reduced the probability of infection below $1x10^{-4}$.

In all cases, the probability of infection depended on the amount of soil consumed with the crop. The default value for fraction of soil adhering to the aboveground crop is very low; using this value (~20 mg/crop unit), the calculated probability of infection of the food consumer was $<10^{-16}$ in Practice I, indicating that a waiting period would not be required for a low level of surface contamination. Similarly, the probability of infection depends on the concentration of pathogens in the sludge when it is applied. Therefore, the appropriate waiting period should probably be variable, depending not only on intended land use, as is currently true, but also on sludge application rate and pathogen

concentration. In calculating a safe waiting period, conservative assumptions should be made about amounts of soil ingested with crops.

U.S. EPA restrictions (U.S. EPA, 1989b) on growing food crops in sludge-amended soil, while necessary for protection against potential health hazards from parasites, appear to be more stringent than required by typical or even worst-case inactivation rates for viruses on crops.

8. RESEARCH NEEDS

8.1. INFORMATION NEEDS FOR VIRUSES

Major requirements to improve the understanding of viruses are standardized methods for detecting and enumerating viruses and for studying their survival. Ideally, any standard method for testing virus occurrence and infectivity should be able to (1) detect all pathogenic viruses; (2) allow differentiation among the viruses in the sample; (3) provide a correlation between assay and infective dose to humans; (4) accurately characterize infective plaques, i.e., neither overestimate nor underestimate the number of infective plaque forming units (Wellings, 1987; Bertucci et al., 1983).

Unfortunately, standardized methods for detecting and characterizing viruses in soil, sediments, and both groundwater and surface water are not yet available (Rao and Melnick, 1987). For example, many of the methods used for concentrating and assaying viruses in water do not recover that portion of the viruses adsorbed on suspended solids (Rao, 1987) or adhering to sample containers (Ward and Winston, 1985). Wellings (1987) describes methods for recovering viruses from different soil types (Berg and Berman, 1984; Farrah and Bitton, 1984; Goyal, 1984) but emphasizes the absence of an efficient, standard method.

The recent publication (ASTM, 1990) of a standard method for recovery of viruses from wastewater sludge is a significant improvement in methodology, although Cliver (1987) points out that there is no single recovery method that is optimal for all viruses in all types of sludges. Likewise, the development by Ijaz et al. (1987) of a methodology for studying the aerobiology of viruses is an important contribution to laboratory techniques, because the lack of standardization has been a problem in comparing research results. Sobsey et al. (1985) have developed a method for recovery and quantitation of hepatitis A virus (HAV) from water, and Payment and Armon (1989) have reviewed detection methods for viruses in drinking water.

Citing the need to develop more reliable techniques for recovering more types of enteroviruses from contaminated water, Jansons and Bucens (1986) describe a method for concentrating rotavirus by hollow fiber ultrafiltration, thereby overcoming some of the drawbacks of filter adsorption-elution methods. Guttman-Bass et al. (1987) compared

methods for detecting rotavirus in water but found that none of the methods was sensitive enough to detect rotaviruses in Jerusalem wastewater. Hurst et al. (1988) compare the efficiency of three methods for detecting adenoviruses.

Gerba et al. (1989) suggest that perhaps one of the most promising techniques for detecting viruses in water and other environmental samples may be the use of gene probes, a methodology that is rapid, inexpensive, sensitive, does not rely on viral cultivation, and could allow development of field test kits. Limitations are that the technique cannot distinguish between infectious and noninfectious viruses without use of cell culture, and it currently requires radioactive labels for increased sensitivity. More research is needed to evaluate the sensitivity and reliability of this technique in environmental media.

Another major information need is a better understanding of the epidemiology and relative infectivity under varied environmental conditions of enteric viruses, particularly rotaviruses, coronaviruses, picornaviruses, and the other viruses that have been less studied than poliovirus and HAV. Although infection by droplet aerosols has been studied extensively for many enteroviruses, much less is known about infection via dry particulate aerosols, which appear to be of more concern for sludge application practices.

Additional data are needed on persistence and transport of viruses, particularly those that have not been as well characterized, in soil, surface water and groundwater. A better understanding of viral transport and persistence in relation to physical conditions and predatory microorganisms would provide necessary data for improved modeling. Likewise, development of predictive parameters for densities of viruses in treated sludge would be of value in determining initial concentrations for adjusting application rates.

In summary, the following information is needed to improve the usefulness of the Pathogen Risk Assessment Model and to allow for a more reliable risk assessment of land application of sewage sludge:

- Simple and accurate standardized methods for detecting and quantifying, by type, pathogenic viruses in treated sludge destined for land application, in final composted sludge products, and in environmental media;
- Improved understanding of minimum infective doses, particularly low-dose effects and MIDs for sensitive subjects;

- More accurate persistence and transport data on all pathogenic viruses of major concern in sludge;
- Development of an index of soil types that would correlate capacity for solute transport and suitability for sludge application (also valuable for onsite waste disposal or solid waste disposal);
- Research on subsurface injection of sludge and the relative probability of virus transport in groundwater; and
- Epidemiologic studies evaluating enteric virus transmission.

8.2. MODEL DEVELOPMENT

The literature review yielded a considerable body of information that invalidated some of the recommended starting parameters for the model. Among these are density of viruses in treated sludge, inactivation rates in aerosols, temperature-dependent inactivation rates in soil and water, and fractional transfers among compartments. Many of these parameters are represented by default values included in the model code (U.S. EPA, 1989a). An update of the model should include revision of default parameters for parasites (U.S. EPA, 1990) and bacterial pathogens (U.S. EPA, 1991) as well as viruses. Exposure equations for Practices IV and V should be revised to allow for exposure to undiluted composted sludge during application.

Equations describing temperature-dependent inactivation of viruses in environmental media should be revised in light of new information. The slope and intercept of each inactivation function is fitted empirically to literature values. Regression analysis of the data in Table 3-5 resulted in a correlation coefficient of 0.32. A log transform of the inactivation rates was used in derivation of other temperature-dependent rate equations (U.S. EPA, 1989a). When the data of Table 3-5 were analyzed similarly, a correlation coefficient of 0.67 was found. The data were then grouped by virus type and analyzed separately. The results of this analysis are shown in Table 8-1. The correlation coefficient for poliovirus and echovirus are not good enough to justify use of the parameters derived from the data. The

TABLE 8-1

Parameters for Temperature-Dependent Inactivation of Viruses in Aquatic Systems*

Virus Type	Number of Samples	Slope (m)	Intercept (b)	Correlation Coefficient
Polio	18	-2.44	0.076	0.28
Coxsackie	20	-2.61	0.049	0.87
Echo	11	-2.51	0.009	0.17

*Fitted to the equation $Y = mX + b$, where
X = Temperature
Y = Log_{10} of the inactivation rate
m = Slope
b = Intercept

data for coxsackie viruses represent several studies, although in many of them the temperature was given as a range rather than a specific value (the mid-point of the range was used in Figure 3-1 and in the regression analysis). Therefore, the slope and intercept derived from the data may be less valid than indicated. More data are necessary for derivation of reliable virus-specific slope and intercept factors.

The model has no temperature and relative humidity parameters linked with inactivation of viruses in air. As Ijaz et al. (1985a,b,c; 1987) have demonstrated, these factors are significant determinants in virus persistence. Viruses have been shown to survive longer than bacterial indicators under hostile environmental conditions (Shuval et al., 1989), and some viruses live significantly longer than others (Ijaz et al., 1985b).

Likewise, according to Yates and Yates (1988), subsurface transport models do not incorporate effects of temperature, pH, moisture content, organic matter, etc. on viral inactivation, and it appears no research is in process to incorporate such environmental effects into subsurface transport models. Some researchers, however, have developed alternate methods for model solute transport that implicitly include such factors but avoid the input of environmental data needed by more explicit models (Jury, 1982, 1983; Jury et al., 1982, 1986; Sposito et al., 1986).

Tim and Mostaghimi (1991) have developed a numerical model for predicting virus fate and transport through variably saturated porous medium in the subsurface under transient flow conditions. Simulation of the vertical movement of viruses applied in wastewater effluents and sewage sludges is accomplished by combining expressions describing transient water flow and subsurface solute transport of virus particles. The current model does not now have an adequate component for describing subsurface transport. Consequently, continued model development suggests the importance of evaluating existing aerosol and subsurface transport models to determine if improvements based on recent modeling efforts can be applied to the Pathogen Risk Assessment Model.

As Gerba (1987) points out, the existing models for predicting virus transport and survival should be field-validated. Likewise, the Pathogen Risk Assessment Model should also be field-validated. Perhaps it is some combination of the most useful and effective portions of several models that will ultimately prove to be the best predictor of pathogen

risk. However, the size limitations of a PC-based model make it imperative to refine the selection of the parameters of most significance in assessing pathogen risk from land application of sludge.

In summary, the following revisions would improve the accuracy of the model:

- Revision of default parameter values, especially for inactivation rates in aerosols and temperature-dependent inactivation rates in soil and water;
- Revision of temperature-dependent inactivation algorithms;
- Incorporation of factors for humidity and temperature in inactivation equations for aerosols;
- Incorporation of subroutines for subsurface transport under conditions of transient flow; and
- Incorporation of factors to allow for subsurface transport through solution channels, cracks, etc.

In addition, field validation of the model's predictions is necessary before the model can be considered an accurate predictor of health risk. The model would be easier to use if it were revised to operate from a menu rather than the current lengthy questionnaire format.

9. REFERENCES

Adams, D.J., J.C. Spendlove, R.S. Spendlove and B.B. Barnett. 1982. Aerosol stability of infectious and potentially infectious reovirus particles. Appl. Environ. Microbiol. 44(4): 903-908.

Akin, E.W. and J.C. Hoff. 1978. Human Viruses in the Aquatic Environment: A Status Report with Emphasis on the EPA Research Program. Report to Congress. EPA-570/9-78-006. U.S. EPA, Cincinnati, OH. (Cited in Kowal, 1985)

Akin, E.W., W. Jakubowski, J.B. Lucas and H.R. Pahren. 1978. Health hazards associated with wastewater effluents and sludge: Microbiological considerations. In: Proceedings of the Conference on Risk Assessment and Health Effects of Land Application of Municipal Wastewater and Sludge, B.P. Sagik and C.A. Sorber, Ed. Center for Applied Research and Technology, The University of Texas at San Antonio, San Antonio, TX. p. 9-26.

ASTM (American Society for Testing and Materials). 1990. Standard Practice for Recovery of Viruses from Wastewater Sludges. D 4994-89. In: Annual Book of ASTM Standards.

Bagdasar'yan, G.A. 1964. Survival of viruses of the enterovirus group (poliomyelitis, echo, coxsackie) in soil and on vegetables. J. Hyg. Epidemiol. Microbiol. Immunol. 8: 497. (Cited in Gerba, 1987)

Berg, G. 1978. Viruses in the environment: Criteria for risk. In: Proceedings of the Conference on Risk Assessment and Health Effects of Land Application of Municipal Wastewater and Sludges, B.P. Sagik and C.A. Sorber, Ed. Center for Applied Research and Technology, The University of Texas at San Antonio, San Antonio, TX. p. 216-229.

Berg, G. and D. Berman. 1980. Destruction by anaerobic mesophilic and thermophilic digestion of viruses and indicator bacteria indigenous to domestic sludges. Appl. Environ. Microbiol. 39(2): 361-368.

Berg, G. and D. Berman. 1984. Test Method Submitted to ASTM Task Group Responsible for Developing Standard Methods for Virus Recovery from Soils, D19:24:04:04/05, American Society for Testing and Materials, Philadelphia. (Cited in Wellings, 1987)

Berg, G., R.M. Clark, D. Berman and S.L. Chang. 1965. Aberrations in survival curves. In: Transmission of viruses by the water route, G. Berg, Ed. Interscience Publishers, NY. p. 235-240.

Berk, R.S. 1983. An introduction to the world of microorganisms. In: Microbiology: Basic Principles and Clinical Applications, N.R. Rose and A.L. Barron, Ed. Macmillan Publishing Company, NY. p. 3-10.

Bertucci, J.J., C. Lue-Hing, D. Zenz and S.J. Sedita. 1977. Inactivation of viruses during anaerobic sludge digestion. J.- Water Pollut. Control Fed. 49: 1642-1651.

Bertucci, J.J., S.H. Abid, C. Lue-Hing, C.S. Clark, J.D. Fenters and K.F. Fannin. 1983. Confirmed viruses versus unconfirmed plaques in sewage. J. Environ. Eng. 109(2): 351-368.

Bitton, G. 1978. Survival of enteric viruses. In: Water Pollution Microbiology, Vol. 2, R. Mitchell, Ed. John Wiley and Sons, NY. p. 273-299.

Bitton, G., N. Masterson and G.E. Gifford. 1976. Effect of a secondary treated effluent on the movement of viruses through a cypress dome soil. J. Environ. Qual. 5(4): 370-375.

Bitton, G., O.C. Pancorbo, A.R. Overman and G.E. Gifford. 1978. Retention of viruses during sludge application to soils. Prog. Water Technol. 10: 597-606.

Bitton, G., R. Fraxedas and G.E. Gifford. 1979a. Effect of solar radiation on poliovirus: Preliminary experiments. Water Res. 13(3): 225-228.

Bitton, G., J.M. Davidson and S.R. Farrah. 1979b. On the value of soil columns for assessing the transport pattern of viruses through soils: A critical outlook. Water Air Soil Pollut. 12: 449-457.

Bitton, G., S.R. Farrah, A.R. Overman, G.E. Gifford, O.C. Pancorbo and J.M. Charles. 1980. Fates of Viruses Following Application of Municipal Sludge to Land. Unpublished. (Cited in Pedersen, 1981)

Bitton, G., S.R. Farrah, O.C. Pancorbo and J.M. Davidson. 1981. Fate of viruses following land application of sewage sludge. I. Survival and transport patterns in core studies under natural conditions. In: Viruses and Wastewater Treatment, M. Goddard and M. Butler, Ed. Pergamon Press, Oxford. (Cited in Gerba and Bitton, 1984)

Bitton, G., O.C. Pancorbo and S.R. Farrah. 1983a. Effect of hydrostatic pressure on poliovirus survival in ground water. Ground Water 21(6): 756-758.

Bitton, G., S.R. Farrah, R.H. Ruskin, J. Butner and Y.J. Chou. 1983b. Survival of pathogenic and indicator organisms in ground water. Ground Water 21(4): 405-410.

Bitton, G., O.C. Pancorbo and S.R. Farrah. 1984. Virus transport and survival after land application of sewage sludge. Appl. Environ. Microbiol. 47(5): 905-909.

Bitton, G., J. E. Maruniak and F.W. Zettler. 1987. Virus survival in natural ecosystems. In: Survival and Dormancy of Microorganisms, Y. Henis, Ed. John Wiley and Sons, NY. p. 301-332.

Bixby, R.L. and D.J. O'Brien. 1979. Influence of fulvic acid on bacteriophage adsorption and complexation in soil. Appl. Environ. Microbiol. 38: 840-845.

Booz-Allen and Hamilton, Inc. 1983. A Background Document on Pathogenic Organisms Commonly Found in Municipal Sludge. Prepared for U.S. EPA, Office of Water Regulations and Standards, Washington, D.C. Contract No. 68-01-4930. (Cited in U.S. EPA, 1988)

Brandt, C.D., H.W. Kim, W.J. Rodriguez, J.O. Arrobio, B.C. Jeffries and R.H. Parrott. 1982. Rotavirus gastroenteritis and weather. J. Clin. Microbiol. 16(3): 478-482.

Brashear, D.A. and R.L. Ward. 1983. Inactivation of indigenous viruses in raw sludge by air drying. Appl. Environ. Microbiol. 45: 1943-1945.

Burge, W.D. and N.K. Enkiri. 1978. Virus adsorption by five soils. J. Environ. Qual. 7(1): 73-76.

Burge, W.D. and P.B. Marsh. 1978. Infectious disease hazards of landspreading sewage wastes. J. Environ. Qual. 7(1): 1-9.

Burge, W.D., W.N. Cramer and E. Epstein. 1978. Destruction of pathogens in sewage sludge by composting. Trans. ASAE 21: 510-514.

Burnet, F.M and M. McKie. 1930. Balanced salt action as manifested in bacteriophage phenomena. Aust. J. Exp. Biol. Med. Sci. 7: 183. (Cited in Yates and Yates, 1988)

Camann, D.E., C.A. Sorber, B.P. Sagik, J.P. Glennon and D.E. Johnson. 1978. A model for predicting pathogen concentrations in wastewater aerosols. In: Proceedings of the Conference on Risk Assessment and Health Effects of Land Application of Municipal Wastewater and Sludges, B.P. Sagik and C.A. Sorber, Ed. Center for Applied Research and Technology, The University of Texas at San Antonio, San Antonio, TX. p. 240-271.

Camann, D.E., H.J. Harding and D.E. Johnson. 1980. Wastewater aerosol and school attendance monitoring at an advanced wastewater treatment facility: Durham Plant, Tigard, Oregon. In: Wastewater Aerosols and Disease, H.R. Pahren and W. Jakubowski, Ed. Health Effects Research Laboratory, Office of Research and Development, U.S. EPA, Cincinnati, OH. EPA-600/9-80-028. NTIS PB81-169864.

Camann, D.E., B.E. Moore, H.J. Harding and C.A. Sorber. 1988. Microorganism levels in air near spray irrigation of municipal wastewater: The Lubbock infection surveillance study. J.- Water Pollut. Control Fed. 60(11): 1960-1970.

Carnow, B., R. Northrop, R. Wadden, et al. 1979. Health Effects of Aerosols Emitted from an Activated Sludge Plant. Health Effects Research Laboratory, Office of Research and Development, U.S. EPA, Cincinnati, OH. EPA-600/1-79-019.

CDC (Centers for Disease Control). 1989. Historical Summary Tables Covering the Period 1939-1988. Morbid. Mortal. Wkly. Rep. 37(54): 36, 49-57.

Clark, C.S., G.L. Van Meer, C.C. Linnemann, et al. 1980. Health effects of occupational exposure to wastewater. In: Wastewater Aerosols and Disease, H.R. Pahren and W. Jakubowski, Ed. Health Effects Research Laboratory, Office of Research and Development, U.S. EPA, Cincinnati, OH. EPA-600/9-80-028.

Clarke, N.A., G. Berg, P.W. Kabler and S.L. Chang. 1964. Human enteric viruses in water: Source, survival and removability. In: Advances in Water Pollution Research. Proceedings of the International Conference, London, September 1962, W.W. Eckenfelder, Ed. Macmillan, NY. p. 523-541.

Clarke, S.K.R., G.T. Cook, S.I. Egglestone, et al. 1972. A virus from epidemic vomiting disease. Br. Med. J. 3: 86-89.

Cliver, D.O. 1975. Virus association with wastewater solids. Environ. Lett. 10(3): 215-223.

Cliver, D.O. 1987. Fate of viruses during sludge processing. In: Human Viruses in Sediments, Sludges, and Soils, V.C. Rao and J.L. Melnick, Ed. CRC Press, Boca Raton, FL. p. 111-127.

Corapcioglu, M.Y. and A. Haridas. 1985. Microbial transport in soils and groundwater: A numerical model. Adv. Water Resour. 8: 188-200.

Cords, C.E., C.G. James and L.C. McLaren. 1975. Alteration of capsid proteins of coxsackievirus A13 by low ionic conditions. J. Virol. 15(2): 244-252.

Cubbage, C.P., J.J. Gannon, K.W. Cochran and G.W. Williams. 1979. Loss of infectivity of poliovirus 1 in river water under simulated field conditions. Water Res. 13: 1091-1099.

Davidson, G.P., R.F. Bishop, R.R.W. Townley, I.H. Holmes and B.J. Ruck. 1975. Importance of a new virus in acute sporadic enteritis in children. Lancet 1: 242-246.

de Jong, J.C., T. Trouwborst and K.C. Winkler. 1973. Mechanisms of virus decay in aerosols. In: Airborne Transmission and Airborne Infection, J.F.P. Hers and K.C. Winkler, Ed. John Wiley & Sons, Inc., NY. p. 124-130.

Dimmock, N.J. 1967. Differences between the thermal inactivation of picornaviruses at "high" and "low" temperatures. Virology 31: 338-353.

Dolin, R., N.R. Blacklow, H. DuPont, et al. 1971. Transmission of acute infectious nonbacterial gastroenteritis to volunteers by oral administration of stool filtrates. J. Infec. Dis. 123(3): 307-312.

Dorland's Illustrated Medical Dictionary. 1981. 26th ed. W.B. Saunders Company, Philadelphia.

Duboise, S.M., B.P. Sagik, B.E.D. Moore and J.F. Malina. 1974. Virus migration through soils. In: Virus Survival in Water and Wastewater Systems, J.F. Malina and B.P. Sagik, Ed. Center for Research in Water Resources, Univ. of Texas. p. 233-240. (Cited in Reddy et al., 1981)

Duboise, S.M., B.E. Moore, C.A. Sorber and B.P. Sagik. 1979. Viruses in soil systems. CRC Crit. Rev. Microbiol. 7: 245-285.

Eisenhardt, A., E. Lund and B. Nissen. 1977. The effect of sludge digestion on virus infectivity. Water Res. 11: 579-581.

Elliott, L.F. and J.R. Ellis. 1977. Bacterial and viral pathogens associated with land application of organic wastes. J. Environ. Qual. 6(3): 245-251.

Englande, A.J. 1983. Incidence and fate of viruses in sludges. In: Sludge Characteristics and Behavior, J.B. Carberry and A.J. Englande, Ed. Martinus Nijhoff Pub., Boston.

Estes, M.J., E.L. Palmer and J.F. Obijeski. 1983. Rotaviruses: A review. Curr. Top. Microbiol. Immunol. 105: 123-184.

Fannin, K.F., S.C. Vana and W. Jakubowski. 1985. Effect of an activated sludge wastewater treatment plant on ambient air densities of aerosols containing bacteria and viruses. Appl. Environ. Microbiol. 49(5): 1191-1196.

Farrah, S.R. and G. Bitton. 1984. Test Method Submitted to ASTM Task Group Responsible for Developing Standard Methods for Virus Recovery from Soils, D19:24:04:04/05, American Society for Testing and Materials, Philadelphia. (Cited in Wellings, 1987)

Fattal, B., Y. Wax, M. Davies and H.I. Shuval. 1986a. Health risks associated with wastewater irrigation: An epidemiological study. Am. J. Public Health 76(8): 977-979.

Fattal, B., P. Yekutiel, Y. Wax and H.I. Shuval. 1986b. Prospective epidemiological study of health risks associated with wastewater utilization in agriculture. Water Sci. Technol. 18: 199-209.

Feachem, R.G., D.J. Bradley, H. Garelick and D.D. Mara. 1983. Sanitation and Disease: Health Aspects of Excreta and Wastewater Management. World Bank/John Wiley & Sons, NY. 501 p.

Fletcher, M.W. and R.L. Meyers. 1974. Groundwater tracing in karst terrain using bacteriophage T-4. Abstr. Annual Meeting of the Am. Soc. Microbiol. p. 52.

Foster, S.O., E.L. Palmer, G.W. Gary, et al. 1980. Gastroenteritis due to rotavirus in an isolated Pacific island group: An epidemic of 3439 cases. J. Infect. Dis. 141(1): 32-39.

Funderburg, S.W., B.E. Moore, B.P. Sagik and C.A. Sorber. 1981. Viral transport through soil columns under conditions of saturated flow. Water Res. 15: 703-711.

Gerba, C.P. 1983a. Pathogens. In: Utilization of Municipal Wastewater and Sludge on Land, A.L. Page, T.L. Gleason, J.E. Smith, I.K. Iskandar and L.E. Sommers, Ed. University of California, Riverside, CA. p. 147-187. (Cited in Kowal, 1985)

Gerba, C.P. 1983b. Virus survival and transport in groundwater. Dev. Indust. Microbiol. 24: 247-251.

Gerba, C.P. 1984a. Applied and theoretical aspects of virus adsorption to surfaces. Adv. Appl. Microbiol. 30: 133-168.

Gerba, C.P. 1984b. Microorganisms as groundwater tracers. In: Groundwater Pollution Microbioloby, G. Bitton and C.P. Gerba, Ed. John Wiley and Sons, NY. p. 225-233.

Gerba, C.P. 1984c. Strategies for the Control of Viruses in Drinking Water, Report to the American Association for the Advancement of Science. Washington, D.C. (Cited in Yates, 1990)

Gerba, C.P. 1985. Microbial contamination of the subsurface. In: Ground Water Quality, C.H. Ward, W. Giger and P.L. McCarty, Ed. John Wiley and Sons, NY. p. 53-67.

Gerba, C.P. 1987. Transport and fate of viruses in soils: Field studies. In: Human Viruses in Sediments, Sludges and Soils, V.C. Rao and J.L. Melnick, Ed. CRC Press, Boca Raton, FL. p. 141-153.

Gerba, C.P. and G. Bitton. 1984. Microbial pollutants: Their survival and transport pattern to groundwater. In: Groundwater Pollution Microbiology, G. Bitton and C.P. Gerba, Ed. John Wiley and Sons, NY. p. 65-80.

Gerba, C.P. and J.C. Lance. 1978. Poliovirus removal from primary and secondary sewage effluent by soil filtration. Appl. Environ. Microbiol. 36(2): 247-251.

Gerba, C.P. and G.E. Schaiberger. 1975. Effect of particulates on virus survival in seawater. J.- Water Pollut. Control Fed. 47(1): 93-103.

Gerba, C.P., S.M. Goyal, C.J. Hurst and R.L. LaBelle. 1980. Type and strain dependence of enterovirus adsorption to activated sludge, soils and estuarine sediments. Water Res. 14: 1197-1198.

Gerba, C.P., S.M. Goyal, I. Cech and G.F. Bogdan. 1981. Quantitative assessment of the adsorptive behavior of viruses to soils. Environ. Sci. Technol. 15(8): 940-944.

Gerba, C.P., A.B. Margolin and M.J. Hewlett. 1989. Application of gene probes to virus detection in water. Water Sci. Technol. 21(3): 147-154.

Goddard, M.R., J. Bates and M. Butler. 1981. Recovery of indigenous enteroviruses from raw and digested sewage sludges. Appl. Environ. Microbiol. 42(6): 1023-1028.

Goddard, M.R., J. Bates and M. Butler. 1982. Isolation of indigenous enteroviruses from chemically treated and dewatered sludge samples. Appl. Environ. Microbiol. 44(5): 1042-1046.

Gold, E. and G.A. Nankervis. 1989. Cytomegalovirus. In: Viral Infections of Humans: Epidemiology and Control, A.S. Evans, Ed. Plenum Medical Book Company, NY. p. 169-189.

Goyal, S.M. 1984. Test Method Submitted to ASTM Task Group Responsible for Developing Standard Methods for Virus Recovery from Soils, D19:24:04:04/05, American Society for Testing and Materials, Philadelphia. (Cited in Wellings, 1987)

Goyal, S.M. and C.P. Gerba. 1979. Comparative adsorption of human enteroviruses, simian rotavirus, and selected bacteriophages to soils. Appl. Environ. Microbiol. 38(2): 241-247.

Grigor'eva, L.V., A.S. Gorodetskii, T.G. Omel'yanets and L.A. Bogdanenko. 1965. Survival of bacteria and viruses on vegetable crops irrigated with infected water. Hyg. Sanit. 30: 357-361.

Grohmann, G., R.I. Glass, J. Gold, et al. 1991. Outbreak of human calicivirus gastroenteritis in a day-care center in Sydney, Australia. J. Clin. Microbiol. 29(3): 544-550.

Grosser, P.W. 1984. A one-dimensional mathematical model of virus transport. 2nd International Conference on Groundwater Quality Research, Tulsa, OK. (Cited in Tim and Mostaghimi, 1991)

Guttman-Bass, N., Y. Tchorsh and E. Marva. 1987. Comparison of methods for rotavirus detection in water and results of a survey of Jerusalem wastewater. Appl. Environ. Microbiol. 53(4): 761-767.

Haas, C.N. 1986. Wastewater disinfection and infectious disease risks. CRC Crit. Rev. Environ. Control 17(1): 1-20.

Herrmann, J.E., K.D. Kostenbader and D.O. Cliver. 1974. Persistence of enteroviruses in lake water. Appl. Microbiol. 28(5): 895-896.

Hurst, C.J. 1988a. Effect of environmental variables on enteric virus survival in surface freshwaters. Water Sci. Technol. 20(11/12): 473-476.

Hurst, C.J. 1988b. Influence of aerobic microorganisms upon virus survival in soil. Can. J. Microbiol. 34: 696-699.

Hurst, C.J. 1989. Fate of virus during wastewater sludge treatment processes. CRC Crit. Rev. Environ. Control 18(4): 317-343.

Hurst, C.J., S.R. Farrah, C.P. Gerba and J.L. Melnick. 1978. Development of quantitative methods for the detection of enteroviruses in sewage sludges during activation and following land disposal. Appl. Environ. Microbiol. 36(1): 81-89.

Hurst C.J., C.P. Gerba and I. Cech. 1980a. Effects of environmental variables and soil characteristics on virus survival in soil. Appl. Environ. Microbiol. 40(6): 1067-1079.

Hurst, C.J., C.P. Gerba, J.C. Lance and R.C. Rice. 1980b. Survival of enteroviruses in rapid-infiltration basins during the land application of wastewater. Appl. Environ. Microbiol. 40(2): 192-200.

Hurst, C.J., K.A. McClellan and W.H. Benton. 1988. Comparison of cytopathogenicity, immunofluorescence and *in situ* DNA hybridization as methods for the detection of adenoviruses. Water Res. 22(12): 1547-1552.

Hurst, C.J., W.H. Benton and K.A. McClellan. 1989. Thermal and water source effects upon the stability of enteroviruses in surface freshwaters. Can. J. Microbiol. 35: 474-480.

IAWPRC. 1983. The health significance of viruses in water. Water Res. 17: 121-132.

Ijaz, M.K., A.H. Brunner, S.A. Sattar, R.C. Nair and C.M. Johnson-Lussenburg. 1985a. Survival characteristics of airborne human coronavirus 229E. J. Gen. Virol. 66: 2743-2748.

Ijaz, M.K., S.A. Sattar, C.M. Johnson-Lussenburg and V.S. Springthorpe. 1985b. Comparison of the airborne survival of calf rotavirus and poliovirus type 1 (Sabin) aerosolized as a mixture. Appl. Environ. Microbiol. 49(2): 289-293.

Ijaz, M.K., S.A. Sattar, C.M. Johnson-Lussenburg, V.S. Springthorpe and R.C. Nair. 1985c. Effect of relative humidity, atmospheric temperature, and suspending medium on the airborne survival of human rotavirus. Can. J. Microbiol. 31: 681-685.

Ijaz, M.K., Y.G. Karim, S.A. Sattar and C.M. Johnson-Lussenburg. 1987. Development of methods to study the survival of airborne viruses. J. Virol. Methods 18: 87-106.

Jansons, J. and M.R. Bucens. 1986. Concentration of rotavirus by ultrafiltration. Water Res. 20(1): 79-83.

Jansons, J., L.W. Edmonds, B. Speight and M.R. Bucens. 1989. Survival of viruses in groundwater. Water Res. 23(3): 301-306.

Jewell, W.J., R.M. Kabrick and J.A. Spada. 1980. Autoheated Aerobic Thermophilic Digestion with Air Aeration. Draft Report. Municipal Environmental Research Laboratory, U.S. EPA, Cincinnati, OH. (Cited in Pedersen, 1981)

Johnson, D.E., D.E. Camann, C.A. Sorber, B.P. Sagik and J.P. Glennon. 1978. Aerosol monitoring for microbial organisms near a spray irrigation site. In: Proceedings of the Conference on Risk Assessment and Health Effects of Land Application of Municipal Wastewater and Sludge, B.P. Sagik and C.A. Sorber, Ed. Center for Applied Research and Technology, The University of Texas at San Antonio, TX. p. 231-239.

Johnson, D.E., et al. 1980a. The Evaluation of Microbiological Aerosols Associated with the Application of Wastewater to Land: Pleasanton, California. EPA-600/1-80-015, U.S. EPA, Cincinnati, OH. (Cited in Camann et al., 1988)

Johnson, D.E., D.E. Camann, K.T. Kimball, R.J. Prevost and R.E. Thomas. 1980b. Health effects from wastewater aerosols at a new activated sludge plant: John Egan Plant, Schaumburg, Illinois. In: Wastewater Aerosols and Disease, H.R. Pahren and W. Jakubowski, Ed. Health Effects Research Laboratory, Office of Research and Development, U.S. EPA, Cincinnati, OH. EPA-600/9-80-028. NTIS PB81-169864.

Jorgensen, P.H. and E. Lund. 1985. Detection and stability of enteric viruses in sludge, soil, and groundwater. Water Sci. Technol. 17: 185. (Cited in Gerba, 1987)

Joyce, G. and H.H. Weiser. 1967. Survival of enteroviruses and bacteriophage in farm pond waters. J. Am. Water Works Assoc. 59: 491-501.

Jury, W.A. 1982. Simulation of solute transport using a transfer function model. Water Resour. Res. 18(2): 363-368.

Jury, W.A. 1983. Chemical transport modeling: Current approaches and unresolved problems. In: Chemical Mobility and Reactivity in Soil Systems, D.W. Nielson, D.E. Elrick and K.K. Tanji, Ed. Am. Soc. Agron., Madison, WI. (Cited in Yates and Yates, 1988)

Jury, W.A., L.H. Stolzy and P. Shouse. 1982. A field test of the transfer function model for predicting solute transport. Water Resour. Res. 18(2): 369-375.

Jury, W.A., G. Sposito and R.E. White. 1986. A transfer function model of solute transport through soil. I. Fundamental concepts. Water Resour. Res. 22(2): 243-247.

Kabrick, R.M., W.J. Jewell, B.V. Salotto and D. Berman. 1979. Inactivation of Viruses, Pathogenic Bacteria and Parasites in the Autoheated Thermophilic Digestion of Sewage Sludges. Proc. 34th Annual Industrial Waste Conference, Purdue University, West Lafayette, IN., May 8-10, 1979. p. 3-24. (Cited in U.S. EPA, 1988)

Kapuscinski, R.B. and R. Mitchell. 1980. Processes controlling virus inactivation in coastal water. Water Res. 14: 363-371.

Katzenelson, E. and D. Mills. 1984. Contamination of vegetables with animal viruses via the roots. Monogr. Virol. 15: 216-220. (Cited in Lasowski and Kott, 1990)

Katzenelson, E., I. Buium and H.I. Shuval. 1976. Risk of communicable disease infection associated with wastewater irigation in agricultural settlements. Science 194: 944-946.

Keswick, B.H. and C. P. Gerba. 1980. Viruses in groundwater. Environ. Sci. Technol. 14(11): 1290-1297.

Keswick, B.H., C.P. Gerba, S.L. Secor and I. Cech. 1982. Survival of enteric viruses and indicator bacteria in groundwater. J. Environ. Sci. Health A17: 903-912.

Keswick, B.H., T.K. Satterwhite, P.C. Johnson, et al. 1985. Inactivation of Norwalk virus in drinking-water by chlorine. Appl. Environ. Microbiol. 50(2): 261-264.

Koch, K. and D. Strauch. 1981. Removal of polio- and parvovirus in sewage sludge by lime treatment. Zentralbl. Bakteriol Mikrobiol. Hyg., Abt. 1 Orig. B 174: 335-347.

Konowalchuk, J. and J.I. Speirs. 1975a. Survival of enteric viruses on fresh vegetables. J. Milk Food Technol. 38(8): 469-472.

Konowalchuk, J. and J.I. Speirs. 1975b. Survival of enteric viruses on fresh fruit. J. Milk Food Technol. 38: 598-600. (Cited in Feachem et al., 1983)

Konowalchuk, J. and J.I. Speirs. 1977. Virus detection on grapes. Can. J. Microbiol. 23: 1301-1303.

Kowal, N.E. 1985. Health Effects of Land Application of Municipal Sludge. Health Effects Research Laboratory, Office of Research and Development, U.S. EPA, Research Triangle Park, NC. EPA/600/1-85/015.

Kowal, N.E., H.R. Pahren and E.W. Akin. 1981. Microbiological health effects associated with the use of municipal wastewater for irrigation. In: Municipal Wastewater in Agriculture, F.M. D'Itri, J.A. Martinez and M.A. Lambarri, Ed. Academic Press, NY. p. 271-342.

Kucera, L.S. 1983. Morphology, classification, multiplication, and genetics of animal viruses. In: Microbiology: Basic Principles and Clinical Applications, N.R. Rose and A.L. Barron, Ed. Macmillan Publishing Company, NY. p. 311-334.

Kurtz, J.B., T.W. Lee and D. Pickering. 1977. Astrovirus associated gastroenteritis in a children's ward. J. Clin. Pathol. 30: 948-952.

Kutz, S.M. and C.P. Gerba. 1988. Comparison of virus survival in freshwater sources. Water Sci. Technol. 20(11/12): 467-471.

Lance, J.C. and C.P. Gerba. 1980. Poliovirus movement during high rate land filtration of sewage water. J. Environ. Qual. 9: 31-34.

Lance, J.C. and C.P. Gerba. 1982. Virus removal with land filtration. In: Water Reuse, E.J. Middlebrooks, Ed. Ann Arbor Science, Ann Arbor, MI. p. 641-660.

Lance, J.C. and C.P. Gerba. 1984. Virus movement in soil during saturated and unsaturated flow. Appl. Environ. Microbiol. 47(2): 335-337.

Lance, J.C., C. P. Gerba and J.L. Melnick. 1976. Virus movement in soil columns flooded with secondary sewage effluent. Appl. Environ. Microbiol. 32(4): 520-526.

Lance, J.C. C.P. Gerba and D.-S. Wang. 1982. Comparative movement of different enteroviruses in soil columns. J. Environ. Qual. 11(3): 347-351.

Landry, E.F., J.M. Vaughn, M.Z. Thomas and C.A. Beckwith. 1979. Adsorption of enteroviruses to soil cores and their subsequent elution by artificial rainwater. Appl. Environ. Microbiol. 38: 680-687.

Landry, E.F., J.M. Vaughn and W.F. Penello. 1980. Poliovirus retention in 75-cm soil cores after sewage and rainwater application. Appl. Environ. Microbiol. 40: 1032-1038.

Larkin, E.P., J.T. Tierney, and R. Sullivan. 1976a. Persistence of poliovirus 1 in soil and on vegetables irrigated with sewage wastes: Potential problems. In: Virus Aspects of Applying Municipal Waste to Land, L.B. Baldwin et al., Ed. Univ. of Florida, U.S. EPA. p. 119-130. (Cited in Reddy et al., 1981)

Larkin, E.P., J.T. Tierney and R. Sullivan. 1976b. Persistence of virus on sewage-irrigated vegetables. J. Environ. Eng. Div. (Am. Soc. Civ. Eng.) 102(EE1): 29-35.

Lasowski, Y.I. and Y. Kott., 1990. Inactivation of enteric viruses in chlorinated wastewater sprayed on vegetables. In: Water Chlorination Chemistry, Environmental Impact and Health Effects, Vol. 6, R.L. Jolley, L.W. Condie, J.D. Johnson, et al., Ed. Proceedings of the Sixth Conference on Water Chlorination: Environmental Impact and Health Effects, Oak Ridge, TN, May 3-8, 1987. Lewis Pub. p. 945-953.

Lefler, E. and Y. Kott. 1974. Virus retention and survival in sand. In: Virus Survival in Water and Wastewater Systems, J.F. Malina and B.P. Sagik, Ed. Center for Research in Water Resources, Austin, Texas. p. 84. (Cited in Gerba, 1987)

Levine, W.C. and G.F. Craun. 1990. Waterborne disease outbreaks, 1986-1988. Morbid. Mortal. Wkly. Rep. 39(SS-1): 1-13.

Levy, M. and S.E. Read. 1990. Erythema infectiosum and pregnancy-related complications. Can. Med. Assoc. J. 143(9): 849-858.

Lew, J.F., R.I. Glass, M. Petric, et al. 1990. Six-year retrospective surveillance of gastroenteritis viruses identified at ten electron microscopy centers in the USA and Canada. Pediatr. Infect. Dis. J. 9(10): 709-714. BIOSIS Number: 91009916.

Loehr, R.C., W.J. Jewell, J.D. Novak, W.W. Clarkson and G.S. Friedman. 1979. Land Application of Wastes, Vol. II. Van Nostrand Reinhold Company, NY.

Lund, E. 1971. Observations on the virus binding capacity of sludge. In: Proceedings of the Fifth International Water Pollution Research Conference, S.H. Jenkins, Ed. Pergamon Press, Ltd., Oxford. p. 1-24/1-5. (Cited in Traub et al., 1986)

Lund, E. 1978. Human pathogens as potential health hazards in the reuse of water. Ambio 7(2): 56-61.

Mack, W.N., Y.-S. Lu and D.B. Coohan. 1972. Isolation of poliomyelitis virus from a contaminated well. Health Serv. Rep. 87: 271-274. (Cited in Lance and Gerba, 1982)

Martin, R. and A. Thomas. 1974. An example of the use of bacteriophage as a groundwater tracer. J. Hydrol. 23: 73-78.

Matson, D.O., M.K. Estes, R.I. Glass, et al. 1989. Human calicivirus-associated diarrhea in children attending day care centers. J. Infect. Dis. 159(1): 71-78.

Matthess, G. and A. Pekdeger. 1985. Survival and transport of pathogenic bacteria and viruses in ground water. In: Ground Water Quality, C.H. Ward, W. Giger and P.L. McCarty, Ed. John Wiley & Sons, NY. p. 472-482.

McDaniels, A.E., K.W. Cochran, J.J. Gannon and G.W. Williams. 1983. Rotavirus and reovirus stability in microorganism-free distilled and wastewaters. Water Res. 17(10): 1349-1353.

Melnick, J.L. 1987. Human enteric viruses in sediments, sludges, and soils: An overview. In: Human Viruses in Sediments, Sludges and Soils, V.C. Rao and J.L. Melnick, Ed. CRC Press, FL. p. 1-2.

Melnick, J.L. and C.P. Gerba. 1980. The ecology of enteroviruses in natural waters. CRC Crit. Rev. Environ. Control 10(1): 65-93.

Menna, J.H. and L.S.F. Soderberg. 1983. Viral pathogenesis and host defense against viral infections, viral oncogenesis. In: Microbiology: Basic Principles and Clinical Applications, N.R. Rose and A.L. Barron, Ed. Macmillan Publishing Company, NY. p. 335-351.

Metcalf, T.G. 1987. Bioaccumulation and disposition of solids-associated virus by shellfish. In: Human Viruses in Sediments, Sludges, and Soils, V.C. Rao and J.L. Melnick, Ed. CRC Press, Boca Raton, FL. p. 77-89.

Modlin, J.F. 1988. Perinatal echovirus and group B coxsackievirus infections. Clin. Perinatol. 15(2): 233-246. MEDLINE Number 88242122.

Moore, B.E., B.P. Sagik and C.A. Sorber. 1978. Land application of sludges: Minimizing the impact of viruses on water resources. In: Proceedings of the Conference on Risk Assessment and Health Effects of Land Application of Municipal Wastewater and Sludges, B.P. Sagik and C.A. Sorber, Ed. Center for Applied Research and Technology, The University of Texas, San Antonio, TX. p. 154-167.

Moore, R.S., D.H. Taylor, L.S. Sturman, M.M. Reddy and G.W. Fuhs. 1981. Poliovirus adsorption by 34 minerals and soils. Appl. Environ. Microbiol. 42(6): 963-975.

Moore, R.S., D.H. Taylor, M.M. Reddy and L.S. Sturman. 1982. Adsorption of reovirus by minerals and soils. Appl. Environ. Microbiol. 44(4): 852-859.

Moore, B.E., D.E. Camann, G.A. Turk and C.A. Sorber. 1988. Microbial characterization of municipal wastewater at a spray irrigation site: The Lubbock infection surveillance study. J.- Water Pollut. Control Fed. 60(7): 1222-1230.

Morris, E.J and H.M. Darlow. 1971. Inactivation of viruses. In: Inhibition and Destruction of the Microbial Cell, W.G. Hugo, Ed. Academic Press, NY. p. 687-702.

MSDGC (Metropolitan Sanitary District of Greater Chicago) and IIT Research Institute. 1979. Viral and Bacterial Levels Resulting from the Land Application of Digested Sludge. Health Effects Research Laboratory, Office of Research and Development, U.S. EPA, Cincinnati, OH. EPA-600/1-79-015.

Murray, J.P. and S.J. Laband. 1979. Degradation of poliovirus by adsorption on inorganic surfaces. Appl. Environ. Microbiol. 37(3): 480-486.

Murray, J.P. and G.A. Parks. 1980. Poliovirus adsorption on oxide surfaces--correspondence with the DLVO-Lifshitz theory of colloid stability. Am. Chem. Soc. Adv. Chem. Ser. 189: 97-133.

Nahmias, A.J., H. Keyserling and F.K. Lee. 1989. Herpes Simplex Viruses 1 and 2. In: Viral Infections of Humans: Epidemiology and Control, A.S. Evans, Ed. Plenum Medical Book Company, NY. p. 393-417.

Niemi, M. 1976. Survival of Escherichia coli phage T7 in different water types. Water Res. 10: 751-755.

NOAA (National Oceanic and Atmospheric Administration). 1981. Local Climatological Data: Annual Summaries for 1981. Environmental Data and Information Service, National Climatic Center, Asheville, NC.

Noonan, M.J. and J.F. McNabb. 1979a. Contamination of Canterbury groundwater by viruses. In: The Quality and Movement of Ground Water in Alluvial Aquifers, M.J. Noonan, Ed. Tech. Publication No. 2. Lincoln College, Christ Church, New Zealand. p. 195-201. (Cited in Rao et al., 1986)

Noonan, M.J. and J.F. McNabb. 1979b. Movement of bacteriophage in groundwater in New Zealand. Abs. Ann. Mtg. Am. Soc. Microbiol. p. 221.

O'Brien, R.T. and J. S. Newman. 1977. Inactivation of poliovirus and coxsackieviruses in surface water. Appl. Environ. Microbiol. 33(2): 334-340.

Ohara, G.T. and J.E. Colbaugh. 1975. A summary of observations in thermophilic digester operations. In: Proceedings of the 1975 National Conference on Municipal Sludge Management and Disposal. Anaheim, CA. August 18-20, 1975. p. 218-222. Information Transfer, Rockville, MD. (Cited in Englande, 1983)

Palfi, A. 1972. Survival of enteroviruses during anaerobic sludge digestion. In: Advances in Water Pollution Research, Proceedings of the 6th International Conference, S.H. Jenkins, Ed. Jersusalem, Israel. p. 99-107.

Pancorbo, O.C., B.G. Evanshen, W.F. Campbell, S. Lambert, S.K. Curtis and T.W. Woolley. 1987. Infectivity and antigenicity reduction rates of human rotavirus strain Wa in fresh waters. Appl. Environ. Microbiol. 53(8): 1803-1811.

Pancorbo, O.C., G. Bitton, S.R. Farrah, G.E. Gifford and A.R. Overman. 1988. Poliovirus retention in soil columns after application of chemical- and polyelectrolyte-conditioned dewatered sludges. Appl. Environ. Microbiol. 54(1)118-123.

Parsons, D., C. Brownlee, D. Wetler et al. 1975. Health aspects of sewage effluents irrigation. Pollut. Control Branch, British Columbia Water Resour. Serv. Dept. of Lands, Forests, and Water Resour., Victoria, B.C. (Cited in Burge and Marsh, 1978)

Payment, P. 1989. Presence of human and animal viruses in surface and ground waters. Water Sci. Technol. 21(3): 283-285.

Payment, P. and R. Armon. 1989. Virus removal by drinking water treatment processes. CRC Crit. Rev. Environ. Control 19(1): 15-31.

Payne, C.M., C.G. Ray, V. Borduin, L.L. Minnich and M.D. Lebowitz. 1986. An eight-year study of the viral agents of acute gastroenteritis in humans: Ultrastructural observations and seasonal distribution with a major emphasis on coronavirus-like particles. Diagn. Microbiol. Infect. Dis. 5(1): 39-54. BIOSIS Number: 82035465.

Pedersen, D.C. 1981. Density Levels of Pathogenic Organisms in Municipal Wastewater Sludge--A Literature Review. Prepared under contract # 68-03-2803 at Camp Dresser & McKee Inc., Boston, MA. Municipal Environmental Research Laboratory, Office of Research and Development, U.S. EPA, Cincinnati, OH. EPA-600/2-81-170.

Prince, D.S., C. Astry, S. Vonderfecht, G. Jakab, F.M. Shen and R.H. Yolken. 1986. Aerosol transmission of experimental rotavirus infection. Pediatr. Infect. Dis. J. 5(2): 218-222. (Cited in Sattar and Ijaz, 1987)

Rao, V.C. 1987. Virus association with suspended solids. In: Human Viruses in Sediments, Sludges, and Soils, V.C. Rao and J.L. Melnick, Ed. CRC Press, Boca Raton, FL. p. 57-75.

Rao, V.C. and G. Bitton. 1987. Methods of recovering viruses from aquatic sediments. In: Human Viruses in Sediments, Sludges, and Soils, V.C. Rao and J.L. Melnick, Ed. CRC Press, Boca Raton, FL. p. 21-35.

Rao, V.C. and J.L. Melnick. 1987. Human Viruses in Sediments, Sludges, and Soils, V.C. Rao and J.L. Melnick, Ed. CRC Press, Boca Raton, FL. 240 p.

Rao, V.C., T.G. Metcalf and J.L. Melnick. 1986. Human viruses in sediments, sludges, and soils. Bull. WHO 64(1): 1-14.

Raphael, R.A., S.A. Sattar and V.S. Springthorpe. 1985. Long-term survival of human rotavirus in raw and treated river water. Can. J. Microbiol. 31: 124-128.

Reddy, K.R., R. Khaleel and M.R. Overcash. 1981. Behavior and transport of microbial pathogens and indicator organisms in soils treated with organic wastes. J. Environ. Qual. 10(3): 255-266.

Righthand, V.F. 1983. Picornaviruses and viruses associated with gastroenteritis. In: Microbiology: Basic Principles and Clinical Applications, N.R. Rose and A.L. Barron, Ed. Macmillan Publishing Company, NY p. 383-391.

Rosenberg, H.S. 1987. Cardiovascular effects of congenital infections. Am. J. Cardiovasc. Pathol. 1(2): 147-156. MEDLINE Number 89087676.

Sagik, B.P., B.E. Moore and C.A. Sorber. 1978. Infectious disease potential of land application of wastewater. In: State of Knowledge in Land Treatment of Wastewater, H.L. McKim, Ed. U.S. Army Corps of Engineers, Cold Regions Research and Engineering Laboratory, Hanover, NH. p. 35. (Cited in Sobsey and Shields, 1987)

Salo, R.J. and D.O. Cliver. 1976. Effect of acid, pH, salts, and temperature on the infectivity and physical integrity of enteroviruses. Arch. Virol. 52: 269-282.

Sanders, D.A., J.F. Malina, B.E. Moore, B.P. Sagik and C. A. Sorber. 1979. Fate of poliovirus during anaerobic digestion. J.- Water Pollut. Control Fed. 51(2): 333-343.

Sattar, S.A. 1981. Virus survival in receiving waters. In: Viruses and Wastewater Treatment, M. Goddard and M. Butler, Ed. Pergamon Press, NY. (Cited in Yates et al., 1990)

Sattar, S.A. and M.K. Ijaz. 1987. Spread of viral infections by aerosols. CRC Crit. Rev. Environ. Control 17(2): 89-131.

Sattar, S.A., S. Ramia and J.C.N. Westwood. 1976. Calcium hydroxide (lime) and the elimination of human pathogenic viruses from sewage: Studies with experimentally-comtaminated (poliovirus type 1, Sabin) and pilot-plant samples. Can. J. Public Health 67(3): 221-226.

Schaub, S.A. and C.A. Sorber. 1977. Virus and bacteria removal from wastewater by rapid infiltration through soil. Appl. Environ. Microbiol. 33(3): 609-619.

Scheuerman, P.R. 1984. Fate of viruses during aerobic digestion of wastewater sludges. Ph.D. Dissertation, University of Florida. (Cited in U.S. EPA, 1988)

Schiff, G.M., G.M. Stefanovic, E.C. Young, D.S. Sander, J.K. Pennekamp and R.L. Ward. 1984. Studies of echovirus-12 in volunteers: Determination of minimal infectious dose and the effect of previous infection on infectious dose. J. Infect. Dis. 150(6): 858-866.

Shuval, H.I., N. Guttman-Bass, J. Applebaum and B. Fattal. 1989. Aerosolized enteric bacteria and viruses generated by spray irrigation of wastewater. Water Sci. Technol. 21(3): 131-135.

Slote, L. 1976. Viral aerosols: A potential occupationally related health threat in aerated wastewater treatment systems. J. Environ. Health 38: 310. (Cited in Sattar and Ijaz, 1987)

Sobsey, M.D. 1983. Transport and fate of viruses in soils. In: Microbial Health Considerations of Soil Disposal of Domestic Wastewater, L.W. Canter, E.W. Akin, J.F. Kreissl and J.F. McNabb, Ed. U.S. EPA, Cincinnati, OH. EPA-600/9-83-017. (Cited in Yates and Yates, 1988)

Sobsey, M.D. and P.A. Shields. 1987. Survival and transport of viruses in soils: Model studies. In: Human Viruses in Sediments, Sludges and Soils, V.C. Rao and J.L. Melnick, Ed. CRC Press, Boca Raton, FL. p. 155-177.

Sobsey, M.D., C.H. Dean, M.E. Knuckles and R.A. Wagner. 1980. Interactions and survival of enteric viruses in soil materials. Appl. Environ. Microbiol. 40(1): 92-101.

Sobsey, M.D., S.E. Oglesbee, E.A. Wait and A.I. Cuenca. 1985. Detection of hepatitis A virus (HAV) in drinking water. Water Sci. Technol. 17: 23-38. (Cited in Sobsey and Shields, 1987)

Sobsey, M.D., P.A. Shields, F.H. Hauchman, R.L. Hazard and L.W. Caton. 1986. Survival and transport of hepatitis A virus in soils, groundwater, and wastewater. Water Sci. Technol. 18(10): 97-106.

Sorber, C.A. and K.J. Guter. 1975. Health and hygiene aspects of spray irrigation. Am. J. Public Health 65(1): 47-52.

Sorber, C.A., B.P. Sagik and B.E. Moore. 1979. Aerosols from municipal wastewater spray irrigation. In: Utilization of Municipal Sewage Effluent and Sludge on Forest and Disturbed Land, W.E. Sopper and S.N. Kerr, Ed. University Park, Pennsylvania State University Press. p. 255-263.

Sorber, C.A., B.E. Moore, D.E. Johnson, H.J. Harding and R.E. Thomas. 1984. Microbiological aerosols from the application of liquid sludge to land. J. Water Pollut. Control Fed. 56(7): 830-836.

Spendlove, J.C. and K.F. Fannin. 1982. Methods of characterization of virus aerosols. In: Methods in Environmental Virology, C.P. Gerba and S.M. Goyal, Ed. Marcel Dekker, Inc., NY. p. 261-329. (Cited in Sattar and Ijaz, 1987)

Spillmann, S.K., F. Traub, M. Schwyzer and R. Wyler. 1987. Inactivation of animal viruses during sewage sludge treatment. Appl. Environ. Microbiol. 53(9): 2077-2081.

Sposito, G., R.E. White, P.R. Darrah and W.A. Jury. 1986. A transfer function model of solute transport through soil: 3. The convection-dispersion equation. Water Resour. Res. 22(2): 255-262.

Sproul, O.J. 1978. The efficiency of wastewater unit processes in risk reduction. In: Proceedings of the Conference on Risk Assessment and Health Effects of Land Application of Municipal Wastewater and Sludges, B.P. Sagik and C.A. Sorber, Ed. Center for Applied Research and Technology, The University of Texas at San Antonio, TX. p. 282-296.

Taylor, D.H., R.S. Moore, and L.S. Sturman. 1981. Influence of pH and electrolyte composition on adsorption of poliovirus by soils and minerals. Appl. Environ. Microbiol. 42(6): 976-984.

Tierney, J.T., R. Sullivan and E.P. Larkin. 1977. Persistence of poliovirus 1 in soil and on vegetables grown in soil previously flooded with inoculated sewage sludge or effluent. Appl. Environ. Microbiol. 33(1): 109-113.

Tim, U.S. and S. Mostaghimi. 1991. Model for predicting virus movement through soils. Ground Water 29(2): 251-259.

Traub, F., S.K. Spillmann and R. Wyler. 1986. Method for determining virus inactivation during sludge treatment processes. Appl. Environ. Microbiol. 52(3): 498-503.

USDA (U.S. Department of Agriculture), Soil Conservation Service. 1980. Soil Survey of Chaves County, New Mexico, Northern Part.

USDA (U.S. Department of Agriculture), Soil Conservation Service. 1981a. Soil Survey of Anderson County, Tennessee.

USDA (U.S. Department of Agriculture), Soil Conservation Service. 1981b. Soil Survey of Clinton County, Iowa.

USDA (U.S. Department of Agriculture), Soil Conservation Service. 1981c. Soil Survey of Kern County, California, Southeastern Part.

USDA (U.S. Department of Agriculture), Soil Conservation Service. 1985. Soil Survey of Yakima County, Washington.

USDA (U.S. Department of Agriculture), Soil Conservation Service. 1989. Soil Survey of Highlands County,Florida.

U.S. EPA. 1983. Process Design Manual for Land Application of Municipal Sludge. Municipal Environmental Research Laboratory, Cincinnati, OH. EPA/625/1-83/016.

U.S. EPA. 1986. Development of a Qualitative Pathogen Risk Assessment Methodology for Municipal Sludge Landfilling. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. EPA/600/6-88/006. NTIS PB88-198544.

U.S. EPA. 1988. Pathogen Risk Assessment Feasibility Study. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. EPA/600/6-88/003. NTIS PB88-191440.

U.S. EPA. 1989a. Pathogen Risk Assessment for Land Application of Municipal Sludge, Volumes I and II. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. EPA/600/6-90/002A,B. NTIS PB90-171901/AS and PB90-171919/AS.

U.S. EPA. 1989b. Standards for the Disposal of Sewage Sludge; Proposed Rule. Federal Register 54(23): 5886-5887.

U.S. EPA. 1990. Preliminary Risk Assessment for Parasites in Municipal Sewage Sludge Applied to Land. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. EPA/600/6-91/001. NTIS PB91-182352/AS.

U.S. EPA. 1991. Preliminary Risk Assessment for Bacteria in Municipal Sewage Sludge Applied to Land. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. EPA/600/6-91/006.

Vaughn, J.M., E.F. Landry, C.A. Beckwith and M.Z. Thomas. 1981. Virus removal during groundwater recharge: Effects of infiltration rate on adsorption of poliovirus to soil. Appl. Environ. Microbiol. 41(1): 139-147.

Vilker, V.L., L.H. Frommhagen, R. Kamdar and S. Sundarum. 1978. Applications of ion exchange/adsorption models to virus transport in percolating beds. AIChE Symp. Ser. 74: 84-92. (Cited in Tim and Mostaghimi, 1991)

Wallis, P.M., D.L. Lehmann, D.A. Macmillan and J.M. Buchanan-Mappin. 1984. Sludge application to land compared with a pasture and a hayfield: Reduction of biological health hazard over time. J. Environ. Qual. 13(4): 645-650.

Walter, R., W. Macht, J. Durkop, R. Hecht, U. Hornig and P. Schulze. 1989. Virus levels in river waters. Water Res. 23(2): 133-138.

Wang, D-S, J.C. Lance and C.P. Gerba. 1980. Removal of virus from wastewater during land disposal. Abstr. of the National Meeting of the ACS 20: ENVR 63.

Ward, R.L. 1977. Inactivation of enteric viruses in wastewater sludge. In: Sludge Management, Disposal and Utilization, Proceedings of the Third National Conference. Information Transfer, Inc., Rockville, MD. (Cited in Englande, 1983)

Ward, R.L. and E.W. Akin. 1984. Minimum infective dose of animal viruses. CRC Crit. Rev. Environ. Control 14(4): 297-310.

Ward, R.L. and C.S. Ashley. 1977. Inactivation of enteric viruses in wastewater sludge through dewatering by evaporation. Appl. Environ. Microbiol. 34(5): 564-570.

Ward, R.L. and C.S. Ashley. 1978. Heat inactivation of enteric viruses in dewatered wastewater sludge. Appl. Environ. Microbiol. 36(6): 898-905.

Ward, B.K. and L.G. Irving. 1987. Virus survival on vegetables spray-irrigated with wastewater. Water Res. 21(1): 57-63.

Ward, R.L. and P.E. Winston. 1985. Development of methods to measure virus inactivation in fresh waters. Appl. Environ. Microbiol. 50(5): 1144-1148.

Ward R.L., C.S. Ashley and R.H. Moseley. 1976. Heat inactivation of poliovirus in wastewater sludge. Appl. Environ. Microbiol. 33(3): 339-346.

Ward, R.L., G.A. McFeters and J.G. Yeager. 1984. Pathogens in Sludge: Occurrence, Inactivation, and Potential for Regrowth. Sandia National Laboratories, Albuquerque, NM. SAND83-0557, TTC-0428, UC-41. U.S. DOE Contract DE-AC04-76DP00789.

Ward, R.L., D.I. Bernstein, E.C. Young, J.R. Sherwood, D.R. Knowlton and G.M. Schiff. 1986. Human rotavirus studies in volunteers: Determination of infectious dose and serological response to infection. J. Infect. Dis. 154 (5): 871-880.

Ward, R.L., D.R. Knowlton, J. Stober, et al. 1989. Effect of wastewater spray irrigation on rotavirus infection rates in an exposed population. Water Res. 23(12): 1503-1509.

Wellings, F.M. 1987. Methods of enterovirus recovery from different types of soils. In: Human Viruses in Sediments, Sludges, and Soils, V.C. Rao and S.L. Melnick, Ed. CRC Press, Boca Raton, FL. p. 129-139.

Wellings, F.M, A.L. Lewis and C.W. Mountain. 1974. Virus survival following wastewater spray irrigation of sandy soils. In: Virus Survival in Water and Wastewater Systems, J.F. Malina and B.P. Sagik, Ed. Center for Research in Water Resources Systems, Austin, TX. p. 253-260. (Cited in Gerba, 1985)

Wellings, F.M., A.L. Lewis, C.W. Mountain and L.V. Pierce. 1975. Demonstration of virus in groundwater after effluent discharge onto soil. Appl. Environ. Microbiol. 29(6): 751-757.

Wellings, F.M., A.L. Lewis and C.W. Mountain. 1976. Demonstration of solids-associated virus in wastewater and sludge. Appl. Environ. Microbiol. 31(3): 354-358.

WHO. 1981. The Risk to Health of Microbes in Sewage Sludge Applied to Land. EURO Reports and Studies 54. World Health Organization, Copenhagen, Denmark.

Wilson, M., C.T. Hadden, E.L. Burress, J.M. Webb, R.J.F. Bruins, N.E. Kowal and C. Sonich-Mullin. 1989. Microbiological risk assessment for sludge reuse and disposal options. Joint American Water Works Association-Water Pollution Control Federation Conference, San Diego, CA, August 1989.

Yanko, W.A. 1988. Occurrence of Pathogens in Distribution and Marketing Municipal Sludges. Health Effects Research Laboratory, U.S. EPA, Research Triangle Park, NC. EPA/600/1-87/014.

Yates, M.V. 1984. Virus Persistence in Ground Water, Ph.D. dissertation. University of Arizona, Tucson. (Cited in Yates and Yates, 1988)

Yates, M.V. 1990. The Use of Models for Granting Variances from Mandatory Disinfection of Ground Water Used as a Public Water Supply. Robert S. Kerr Environmental Research Laboratory, Office of Research and Development, U.S. EPA, Ada, OK. EPA/600/2-90/010.

Yates, M.V. and S.R. Yates. 1988. Modeling microbial fate in the subsurface environment. CRC Crit. Rev. Environ. Control. 17(4): 307-344.

Yates, M.V., C.P. Gerba and L.M. Kelley. 1985. Virus persistence in groundwater. Appl. Environ. Microbiol. 49(4): 778-781.

Yates, M.V., L.D. Stetzenbach, C.P. Gerba and N.A. Sinclair. 1990. The effect of indigenous bacteria on virus survival in ground water. J. Environ. Sci. Health A25(1): 81-100.

Yeager, J.G. and R.T. O'Brien. 1979a. Enterovirus inactivation in soil. Appl. Environ. Microbiol. 38(4): 694-701.

Yeager, J.G. and R.T. O'Brien. 1979b. Structural changes associated with poliovirus inactivation in soil. Appl. Environ. Microbiol. 38(4): 702-709.

Young, D.C. and D.G. Sharp. 1977. Poliovirus aggregates and their survival in water. Appl. Environ. Microbiol. 33(1): 168-177.

APPENDIX A

MODEL OVERVIEW

MODEL OVERVIEW

Five sludge management practices, representing land application and D&M management options, are included in the present model and are numbered I-V. They are listed in Table A-1 and illustrated in Figures A-1 through A-5. Two of the practices use heat-dried or composted sludge for residential purposes and three use liquid sludge for commercial farming operations. Since each of these two types of sludge represents a wide range of sludge treatment possibilities, the extent of treatment or conditioning prior to land application must be approximated for each case (i.e., the pathogen concentration in the applied sludge must be specified). The computer model represents the compartments and transfers among compartments of the five management practices. The compartments are the various locations, states or activities in which sludge or sludge-associated pathogens exist; they vary to some extent among practices. In each compartment, pathogens either increase, decrease or remain the same in number with time, as specified by "process functions" (growth, dieoff or no population changes) and "transfer functions" (movement between compartments). The population in each compartment, therefore, generally varies with time and is determined by a combination of initial pathogen input, "transfer functions" and "process functions." The populations of pathogens in the compartments representing human exposure locations (designated with an asterisk in Figures A-1 through A-5 and in Table A-2), together with appropriate intake and infective dose data, are used to estimate human health risk.

Although each practice listed in Table A-1 is different, all five practices share common characteristics. All compartments that appear in one or more of the five sludge management practices are listed in Table A-2. Those compartments with an asterisk represent exposure sites for the human receptor:

- 3* inhalation or ingestion of emissions from application of sludge or tilling of sludge/soil;
- 5* inhalation or ingestion of windblown or mechanically generated particulates;
- 6* swimming in a pond fed by surface water runoff;

TABLE A-1

Sludge Management Practices and Descriptions in Pathogen Risk Assessment Model

PRACTICE	DESCRIPTION[b]
I	Application of Liquid Treated Sludge for Production of Commercial Crops for Human Consumption
II	Application of Liquid Treated Sludge to Grazed Pastures
III	Application of Liquid Treated Sludge for Production of Crops Processed before Animal Consumption
IV	Application of Dried or Composted Sludge to Residential Vegetable Gardens
V	Application of Dried or Composted Sludge to Residential Lawns

[a]Source: U.S. EPA, 1989a
[b]Two types of sludge are used in this model - liquid and dried/composted. The extent of treatment or conditioning prior to application is variable and must be determined for each case.

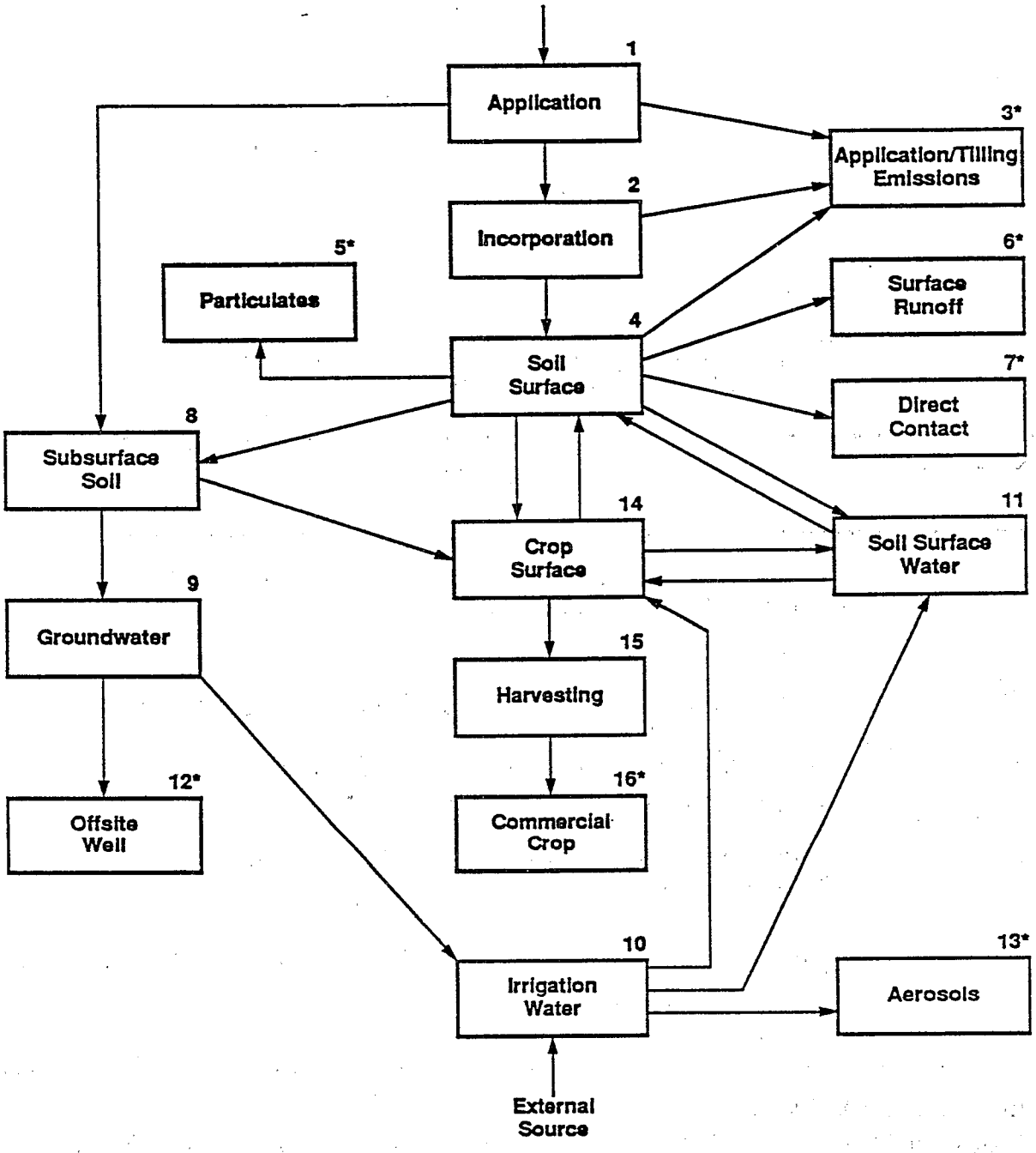

FIGURE A-1

Input/Output Diagram for Practice I – Application of Liquid Sludge for Production of Commercial Crops for Human Consumption

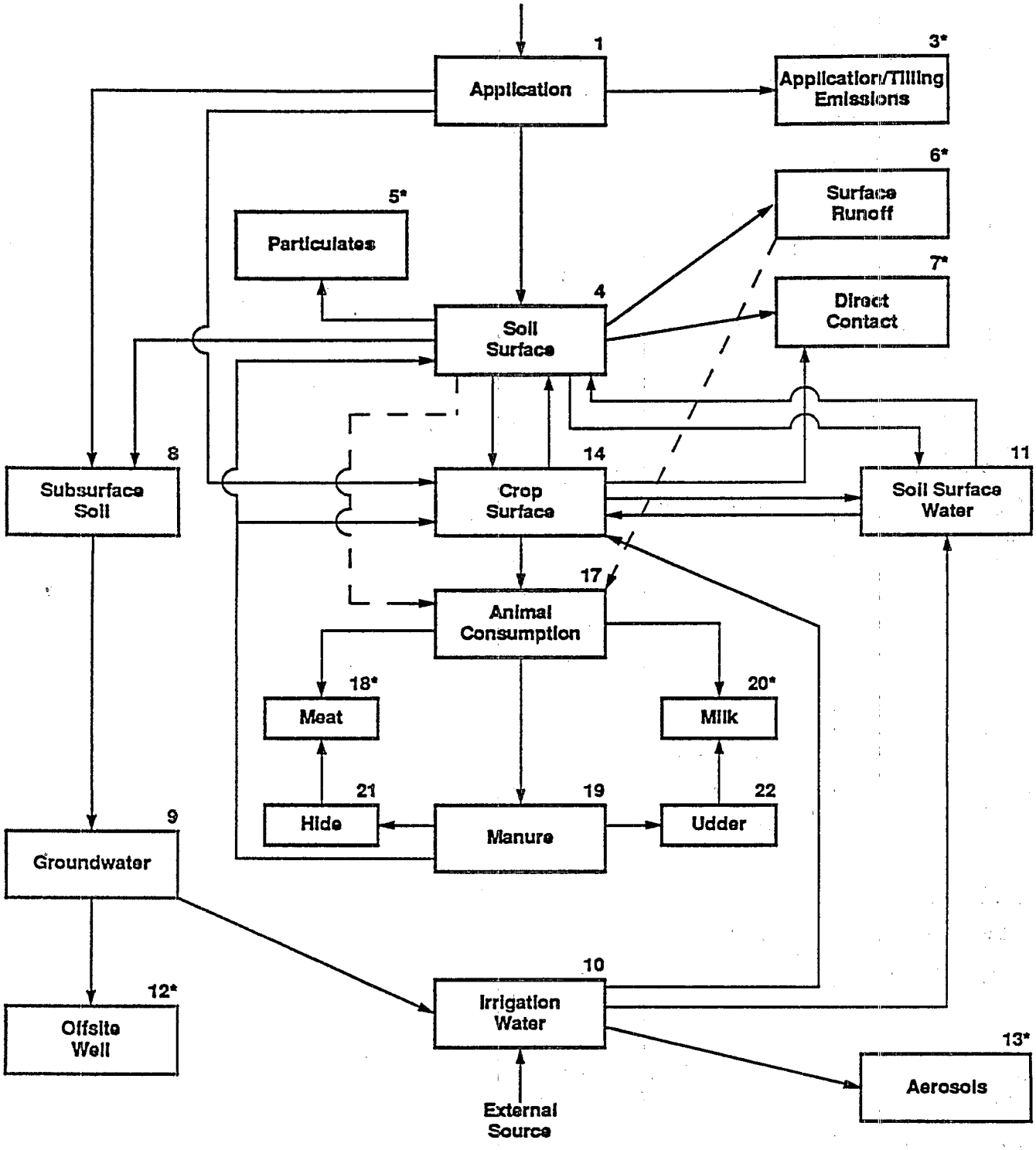

FIGURE A-2

Input/Output Diagram for Practice II – Application of Liquid Sludge to Grazed Pastures

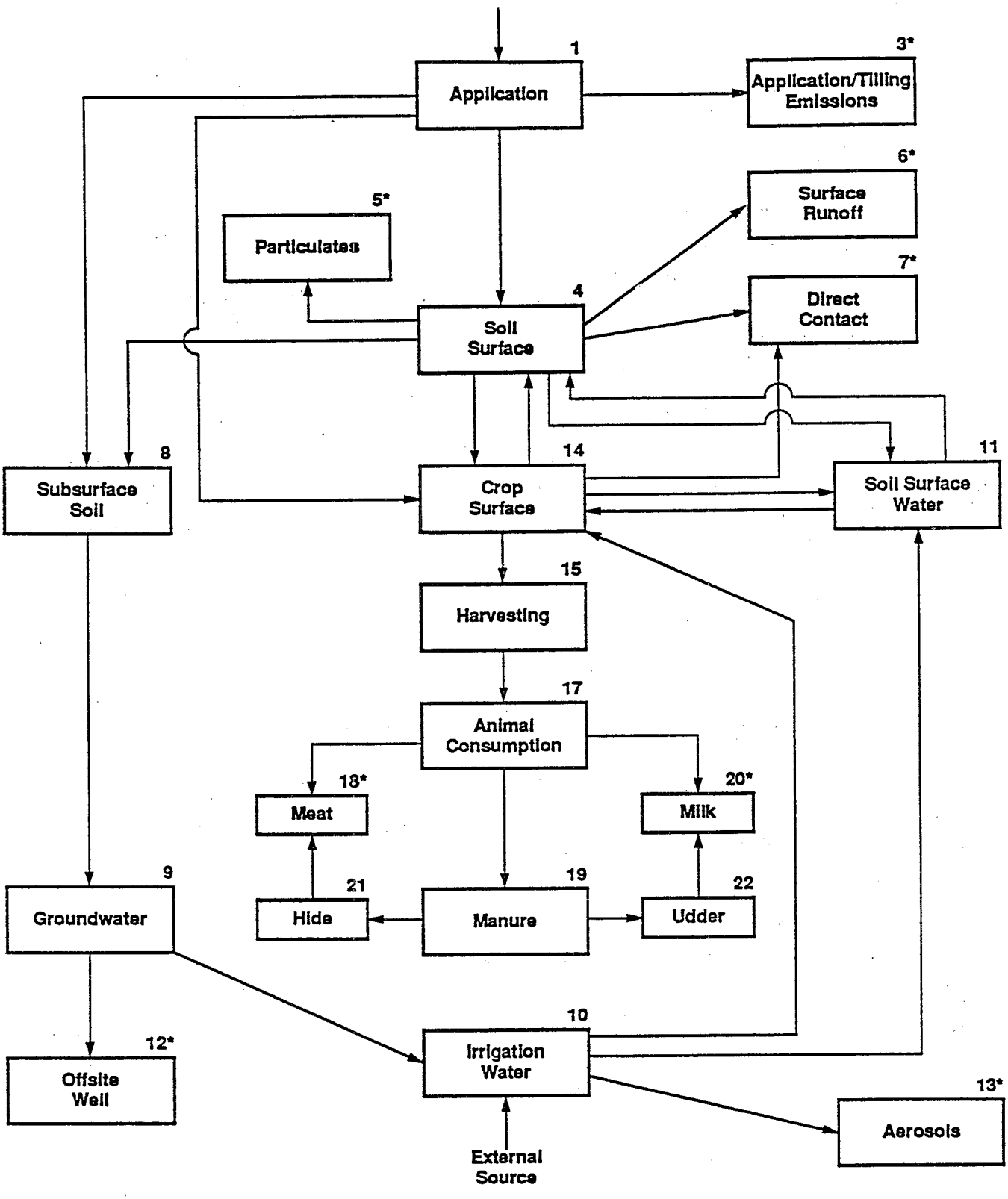

FIGURE A-3

Input/Output Diagram for Practice III – Application of Liquid Sludge for Production of Crops Processed before Animal Consumption

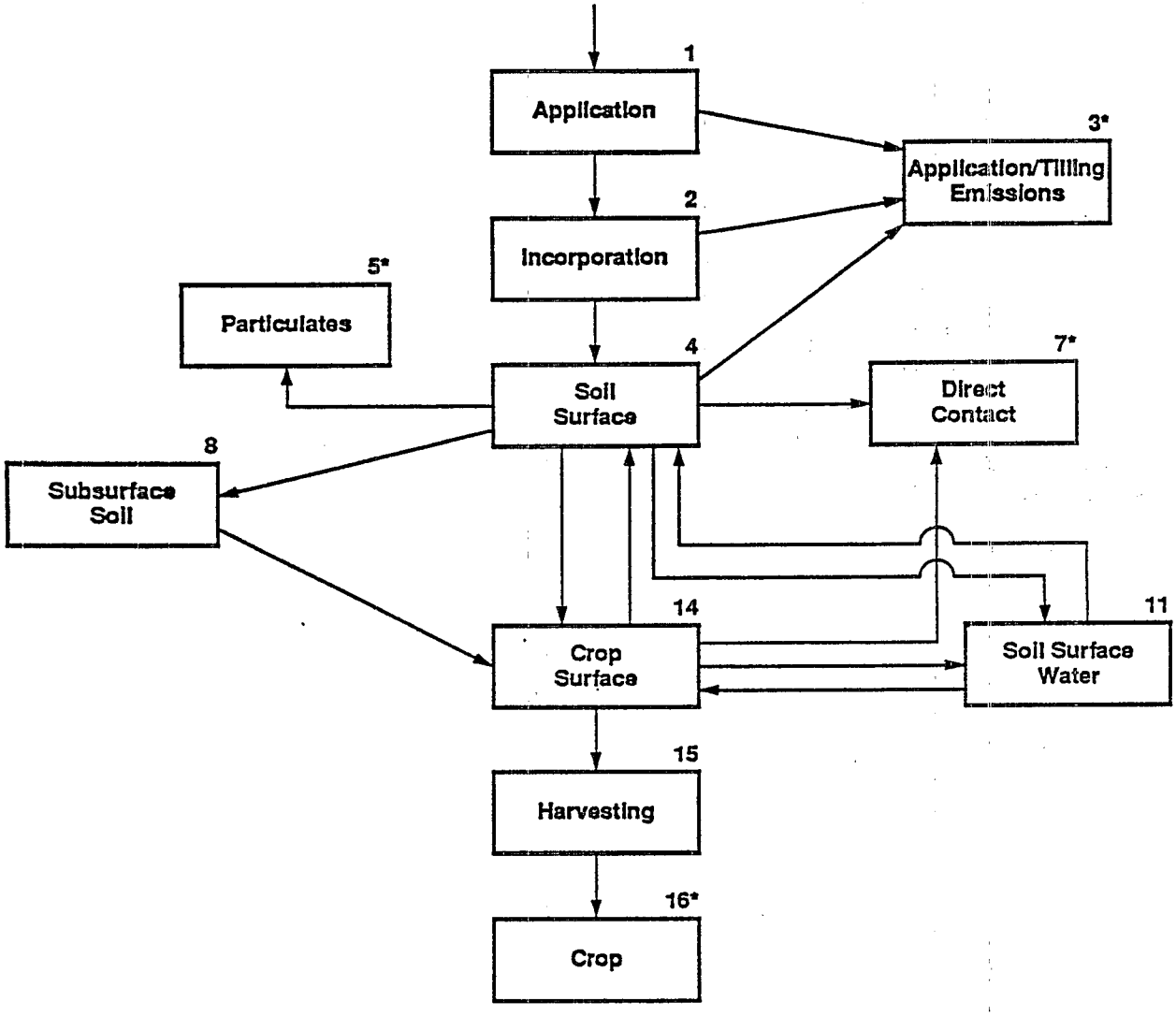

FIGURE A-4

Input/Output Diagram for Practice IV – Application of Dried or Composted Sludge to Residential Vegetable Gardens

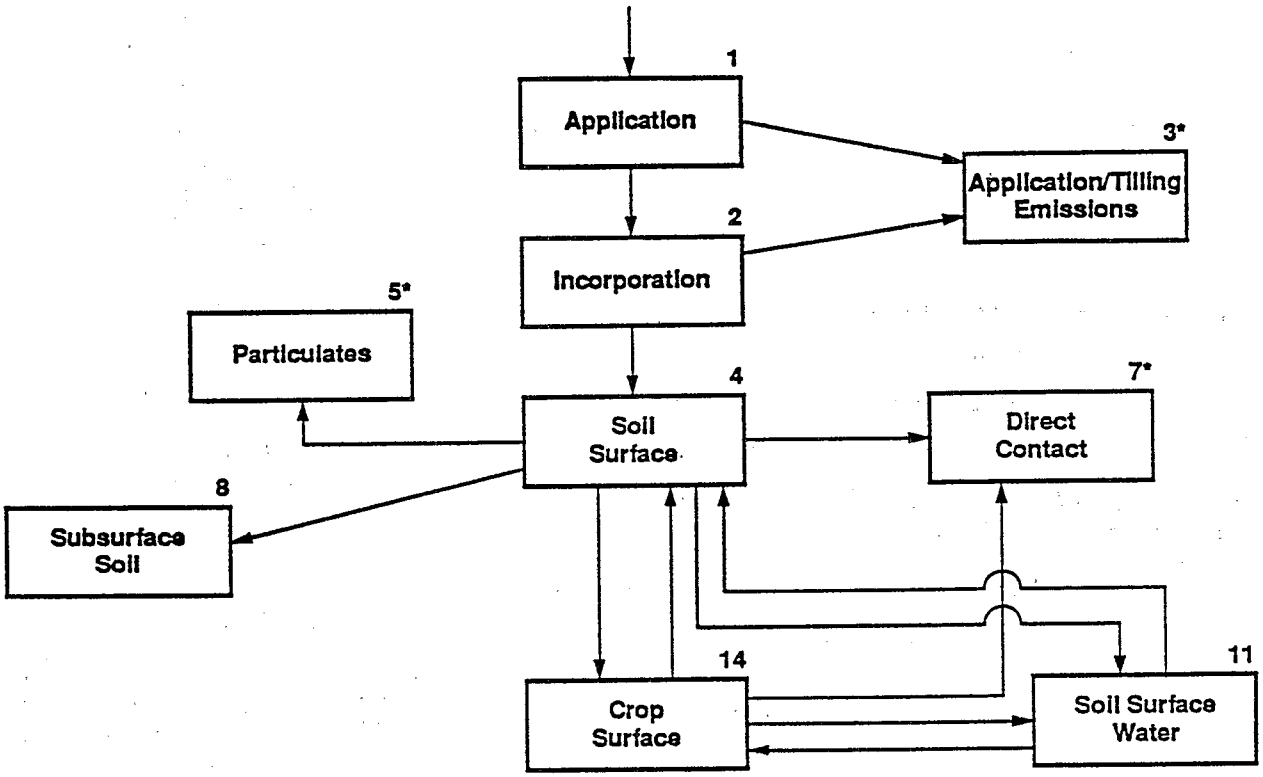

FIGURE A-5

Input/Output Diagram for Practice V – Application of Dried or Composted Sludge to Residential Lawns

TABLE A-2

Compartments Included in the Sludge Management Practices

Compartment Name and Number	Liquid Sludge Management Practices			Dried/Composted Sludge Management Practices	
	I	II	III	IV	V
Application	1	1	1	1	1
Incorporation	2	2	2		
Application/Tilling Emissions	3*[b]	3*	3*	3*	3*
Soil Surface	4	4	4	4	4
Particulates	5*	5*	5*	5*	5*
Surface Runoff	6*	6*	6*		
Direct Contact	7*	7*	7*	7*	7*
Subsurface Soil	8	8	8	8	8
Groundwater	9	9	9		
Irrigation Water	10	10	10		
Soil Surface Water	11	11	11	11	11
Offsite Well	12*	12*	12*		
Aerosols	13*	13*	13*		
Crop Surface	14	14	14	14	14
Harvesting	15		15	15	
(Commercial) Crop	16*			16*	
Animal Consumption		17	17		
Meat		18*	18*		
Manure		19	19		
Milk		20*	20*		
Hide		21	21		
Udder		22	22		

[a]Source: U.S. EPA, 1989a
[b]Asterisk indicates exposure compartments.

- 7* direct contact with sludge-contaminated soil or crops (including grass, vegetables, or forage crops);
- 12* drinking water from an offsite well;
- 13* inhalation and subsequent ingestion of aerosols from irrigation;
- 16* consumption of vegetables grown in sludge-amended soil;
- 18* consumption of meat or
- 20* consumption of milk from cattle grazing on or consuming forage from sludge-amended fields.

The first 14 compartments, most of which are common to all practices, are described below.

APPLICATION (1) represents the application of sludge to a field (default size 10 ha) or to a yard or garden of specified size. Liquid sludge may be applied by spread-flow techniques, by spray, or by subsurface injection. The application rate and pathogen concentrations are variables to be entered by the user of the model. During spread-flow and spray application, sludge will be spread thinly on the soil, where it will be subject to drying, heating and solar radiation, thus losing the protective benefits provided by bulk sludge. It is assumed, therefore, that inactivation will occur at a rate characteristic of the organism in soil at 5°C above the ambient temperature (Brady, 1974; USDA, 1975). It is also assumed that liquid sludge is absorbed by the upper 5 cm of soil surface during this time. The default time period for transfer from APPLICATION (1) to INCORPORATION (2) is 24 hours, which allows a field treated with liquid sludge to dry sufficiently to plow or cultivate. If the injection option is chosen, the liquid sludge goes directly to SUBSURFACE SOIL (8) at hour 10. During spray application of liquid sludge or application of dry composted sludge, droplets or loose particulates may become airborne. Liquid aerosols are modeled by a Gaussian-plume air dispersion model that calculates the downwind concentration of airborne particulates. Dry particulate emissions are calculated using models for generation of dust by tilling or mechanical disturbance of soil. Both are represented as transfers from APPLICATION (1) to APPLICATION/TILLING EMISSIONS (3).

INCORPORATION (2) involves the mixing, by plowing or cultivation, of the sludge and sludge-associated pathogens evenly throughout the upper 15 cm of soil. Process functions associated with this compartment are the same as for the relevant pathogen type in soil. Particulate emissions generated by cultivation are represented by a transfer from INCORPORATION (2) to APPLICATION/TILLING EMISSIONS (3) beginning at hour 24, extending for enough time to cultivate the field (at a rate of 5 ha/hr) or till the garden or lawn (at a rate of 0.002 ha/hr). At the end of this time, all remaining pathogens are transferred to SOIL SURFACE (4).

APPLICATION/TILLING EMISSIONS (3*) is an exposure compartment that receives the dust, or suspended particulates, generated by application or by the tilling of dried sludge or sludge-soil mixture. It also receives aerosols generated by spray application of liquid sludge. All process functions associated with this compartment are incorporated in the aerosol subroutines. Exposure in this compartment is by inhalation but, as in all inhalation exposures, model simplification limits the exposure to the pathogens assumed to be ingested after the inhaled dust or aerosol spray is trapped in the upper respiratory tract, swept back to the mouth by ciliary action and swallowed.

SOIL SURFACE (4) describes the processes occurring in the upper 15 cm (Practices I, IV and V) or upper 5 cm (Practices II and III) of the soil layer. Microbes are inactivated at rates characteristic for moist soil at 5°C above the chosen ambient temperature (Crane and Moore, 1986; Kibbey et al., 1978). Transfers from SOIL SURFACE (4) occur by wind to WIND-GENERATED PARTICULATES (5), at a time chosen by the user, by surface runoff and sediment transport after rainfall events to SURFACE RUNOFF (6), by a person walking through the field or contacting soiled implements or clothing or by other casual contact to DIRECT CONTACT (7), by leaching after irrigation or rainfall to SUBSURFACE SOIL (8), by resuspension during irrigation or rainfall to SOIL SURFACE WATER (11), or at harvest to CROP SURFACE (14).

WIND-GENERATED PARTICULATES (5*) describes the airborne particulates generated by wind. Process functions are the same as for the organism in air-dried soil at the ambient temperature (Crane and Moore, 1986; Kibbey et al., 1978). The exposed individual is standing in the field or at a user-specified distance downwind from the field

during a windstorm. The wind-generated exposure is calculated from user-specified values for duration and severity of the windstorm (default values, 6 hr at 18 m/sec (40 mph)).

SURFACE RUNOFF (6*) is an exposure compartment describing an onsite pond containing pathogens transferred from SOIL SURFACE (4) by surface runoff and sediment transport after rainfall. These processes are described by a separate subroutine. Inactivation rates in this compartment are characteristic of microbes in water and are much lower than rates for soil. Water is removed from the pond by infiltration and recharge of the groundwater aquifer, but it is assumed that no microbes are transferred by this process. The human receptor is an individual who incidentally ingests 0.1 L of contaminated water while swimming in the pond. This compartment is also an exposure compartment for cattle drinking 20 L of water daily from the pond (Practice II).

DIRECT CONTACT (7*) is the exposure compartment for a worker or a child less than 5 years old who plays in or walks through the field, yard or garden, incidentally ingesting 0.1 g of soil or vegetation at the daily geometric mean concentration of pathogens. This human receptor represents the worst-case example of an individual contacting contaminated soil or soiled clothing or implements. No process functions are associated with this compartment because it is strictly an exposure compartment.

SUBSURFACE SOIL (8) describes the processes and transfers for pathogens in the subsurface soil between 5 or 15 cm depth and the water table. It also serves as the incorporation site for subsurface injection of liquid sludge. Process functions in SUBSURFACE SOIL (8) are the same as for moist soil at the ambient temperature. The transfer from SOIL SURFACE (4) occurs after each rain or irrigation event as a result of leaching from the soil surface. The time of transfer is calculated by dividing the depth of rainfall or irrigation by the infiltration rate. Transfer to GROUNDWATER (9) is arbitrarily set at one hour later. At present, the relation between unsaturated water flow and subsurface transport has not been well-established. Thus, this model lacks a satisfactory subroutine to describe pathogen transport from the subsurface soil to groundwater. Instead, user-specified variables are used to describe the fraction of pathogens transferred from SOIL SURFACE (4) to SUBSURFACE SOIL (8) and from SUBSURFACE SOIL to GROUNDWATER.

GROUNDWATER (9) describes the flow of pathogens in the saturated zone. Process functions are the same as for other water compartments. Transfers occur to IRRIGATION WATER (10) if the water is needed for irrigation or to OFFSITE WELL (12*) if the water is used for drinking. The number of pathogens transferred to IRRIGATION WATER (10) is based on the concentration of pathogens in the groundwater compartment and the total depth of irrigation. The transfer to OFFSITE WELL (12) is described by a modification of the subsurface solute transport model of van Genuchten and Alves (van Genuchten and Alves, 1982). Because microbes in suspension are passively transported by bulk water flow and interact with soil particles by adsorption and desorption, they behave similarly enough to dissolved chemicals that existing solute transport models can be used to describe their fate in the saturated zone (Gerba, 1988).

IRRIGATION WATER (10) describes the transfers for pathogen-contaminated water used for irrigation. No processes are associated with this compartment because it is intended as a transition compartment. Irrigation of the field, lawn or garden takes place a user-specified number of times each week. This irrigation water may come from either an onsite well fed by GROUNDWATER (9) or from an outside source of treated, liquid sludge. The default conditions vary by practice. In either case, AEROSOLS (13) are generated unless a non-spray option is chosen. Spray irrigation is the default since it would be most likely to cause a significant exposure to workers or offsite persons. In addition to aerosol emissions, irrigation transfers pathogens to CROP SURFACE (14) and to SOIL SURFACE WATER (11).

SOIL SURFACE WATER (11) represents any irrigation water or rainfall in contact with the ground prior to infiltration. This compartment describes the temporary suspension of pathogens in such a water layer and their subsequent transfer to CROP SURFACE (14) or to SOIL SURFACE (4). Process functions are the same as for other water compartments.

OFFSITE WELL (12*) is the exposure site for a human receptor drinking 2 L/day of contaminated water whose pathogens have been transported through groundwater. Process functions are the same as for groundwater. The groundwater transport subroutine supplies the concentration of pathogens in the well at a user-specified distance from the

source. No transfers out of the compartment are specified because it is an exposure compartment only.

AEROSOLS (13*) describes fugitive emissions from spray irrigation, which occurs at a default rate of 0.5 cm/hr for 5 hr. The source of irrigation water producing AEROSOLS can be an onsite well (i.e., GROUNDWATER) or liquid sludge. A Gaussian-plume model is used to calculate concentrations of airborne microbes downwind. The human receptor is an onsite worker or a person offsite who is exposed during the time of irrigation.

CROP SURFACE (14) describes contamination of vegetable or forage crops by transfer of user-specified amounts to or from SOIL SURFACE (4), from IRRIGATION WATER (10), or to or from SOIL SURFACE WATER (11). Process functions are not well characterized but are assumed to be influenced by drying, thermal inactivation and solar radiation; they are thus most characteristic of pathogens in surface soil.

These preceding 14 compartments are common to most of the five practices modeled. The following descriptions of the five management practices help clarify the differences among the practices.

Practice I: Application of Liquid Treated Sludge for Production of Commercial Crops for Human Consumption.

Liquid sludge may be applied as fertilizer/soil conditioner for the production of agricultural crops for human consumption or for animal forage or prepared feed. Both existing (CFR, 1988) and proposed (U.S. EPA, 1989b) regulations prohibit direct application of sewage sludge to crop surfaces. Therefore, this model practice is designed for a single application of liquid sludge, which is incorporated into the soil before the crop is planted. Regulations also require various waiting periods before the planting of crops that will be consumed uncooked by humans. These restrictions, however, are optional in the model and can be tested.

Vegetables can be grown aboveground, on-ground or below-ground. These are represented by tomatoes, zucchini and carrots, respectively. At HARVESTING (15) time,

all pathogens remaining on CROP SURFACE (14) are transferred to HARVESTING (15), which represents a single harvest of all of the crop. The same process functions apply as in CROP SURFACE (14). The crop is held for 24 hours before being processed. The number of pathogens is then transferred to COMMERCIAL CROPS (16*), the compartment in which further processing takes place. The number of pathogens/crop unit following processing is calculated in this compartment and is the figure used in the vegetable-exposure risk calculations. A 24-hour pathogen exposure is computed by Subroutine VEG. Pathogen concentrations are determined as number/crop unit for each sludge management practice. Before being consumed, vegetables normally are processed in some way. Included in the program is a series of user-selectable processing steps. The user has the option of choosing any or all processing steps and of specifying some conditions within processing steps. The human receptor is a person who consumes minimally prepared vegetables (washed, but not peeled or cooked) at a rate of 81 g tomatoes, 80 g zucchini or 43 g carrots per eating occasion (Pao et al., 1982).

Practice II: Application of Liquid Sludge to Grazed Pastures.

In this practice, liquid sludge is applied as fertilizer, soil conditioner and irrigation water for the production of forage crops for pasture. This model practice is designed for repeated applications of liquid sludge, initially on a field with a standing forage crop used for pasture. It is assumed that spray irrigation will be used because this method is effective for delivering large amounts of sludge to a large area. In this way, the pasture is also used as a final treatment and disposal system for the treated sludge. The irrigation rate, the total weekly depth and the number of times per week can be specified by the user. A sludge solids concentration of 5% is assumed.

The model assumes that each hectare of pasture supports 12 head of cattle, although both area and herd size may be varied. This may be a higher density than is the common practice for fields that receive no irrigation, but with adequate irrigation, sufficient forage is expected to be produced. Current and proposed regulations require various waiting periods before animals can be grazed. These requirements can also be tested by the model.

ANIMAL CONSUMPTION (17) describes the ingestion of CROP SURFACE (14) by cattle grazing in the pasture. Transfers from ANIMAL CONSUMPTION (17) are to MEAT (18*), MANURE (19) and MILK (20*).

MEAT (18*) is the compartment describing transfer of pathogens from ANIMAL CONSUMPTION (17) to meat. The human receptor is assumed to consume 0.256 kg of meat daily (U.S. FDA, 1978). Contamination of meat by gut contents during slaughter or by systemic infection by sludge-borne pathogens can be modeled. The model allows for inactivation of pathogens in meat by cooking, assuming reasonable cooking times and temperatures.

The production and consumption of milk from cattle pastured on the sludge-amended field are modeled when the dairy cattle option is chosen. The default condition is for consumption of raw milk because commercial production of milk poses an extremely small hazard of exposure to pathogens. In the model, contamination from dirty utensils and careless handling are combined as a transfer from the manure-contaminated udder [MANURE (19)], which occurs at each milking. All three pathogens can enter milk by this route. MILK (20*) is the compartment describing production and consumption of milk from cattle pastured on the sludge-amended field when the dairy cattle option is chosen. The default condition models the consumption of raw milk that has been stored for 24 hours. In exposure calculations, it is assumed that the human receptor consumes 2 kg milk/day, roughly three times the national average milk consumption (U.S. FDA, 1978).

Practice III: Application of Liquid Treated Sludge for Production of Crops Processed before Animal Consumption.

In this practice, liquid sludge is applied as fertilizer, soil conditioner and irrigation water for the production of forage crops to be processed and stored for animal feed. This model practice is designed for repeated applications of liquid sludge, initially on a field with a standing forage crop. It is assumed that spray irrigation will be used for the application of liquid sludge, because this method is effective for delivering large amounts of sludge to a large area. In this way, the field is also used as a final treatment and disposal system for the treated sludge. The rate, the total weekly depth and the number of irrigations per week

can be changed by the user. A sludge solids concentration of 5% is assumed. The risks to the human receptor are similar to those for the preceding practice, i.e., exposure through meat or milk, in addition to direct contact with the forage grown in the field.

Practice IV: Application of Dried or Composted Sludge to Residential Vegetable Gardens.

Dried or composted treated sludge may be sold or given away to the public as a bulk or bagged product for use as fertilizer or soil conditioner for the production of domestic garden crops for human consumption. Although some studies have shown that composting is highly effective in removing pathogens from sludge (Wiley and Westerberg, 1969), other studies have shown that bacterial pathogens may grow in dried or composted sludge to concentrations of $1x10^6$ organisms/kg dry weight (U.S. EPA, 1988). Exposure of individuals to materials used in home gardening would be expected to be more frequent than exposure in a commercial agricultural setting. Therefore, this practice would be expected to pose a greater risk of infection. This model practice is designed to describe the application of dried or composted treated sludge, which is incorporated into the soil before the crops are planted.

Vegetables can be grown aboveground, on-ground or below-ground. These are represented by tomatoes, zucchini and carrots, respectively. The user may specify the proportions of above-ground, on-ground and below-ground crops in the garden. At HARVESTING (15) time, all pathogens remaining on CROP SURFACE (14) are transferred to HARVESTING (15). The same process functions apply as in CROP SURFACE (14). The crop is held for 24 hours before being processed. The number of pathogens is then transferred to CROP (16*), the compartment in which further processing takes place. The number of pathogens/crop unit following processing is calculated in this compartment and is the figure used in the vegetable-exposure risk calculations. A 24-hour pathogen exposure is computed by Subroutine VEG. Pathogen concentrations are determined as number/crop unit for each sludge management practice. Pathogen concentrations are determined as number/crop unit.

Before being consumed, vegetables normally are processed in some way. Included in the program is a series of user-selectable processing steps. The user has the option of

choosing any or all processing steps and of specifying some conditions within processing steps. In the default condition, the human receptor is a person who consumes minimally prepared vegetables (washed, but not peeled or cooked) at a rate of 81 g tomatoes, 80 g zucchini or 43 g carrots per eating occasion (Pao et al., 1982).

Practice V: Application of Dried or Composted Sludge to Residential Lawns.

Dried or composted treated sludge may be made available to the public as a bulk or bagged product to be sold or given away for use as fertilizer or soil conditioner for the preparation of a seed bed for domestic lawns. Individuals engaged in preparing a seed bed for a lawn are likely to come into contact with the soil and any additives used to improve the seed bed. If the soil or the additives contain pathogens, this practice would be expected to pose a risk of infection. This model practice is designed to describe the application of dried or composted treated sludge, which is incorporated into the soil before the lawn is seeded.

The main exposure in this practice is for the lawn worker or for a child younger than 5 years old who plays in or walks through the lawn site, incidentally ingesting soil or crop surface at the daily geometric mean concentration of pathogens. This human receptor represents the worst-case example of an individual contacting contaminated soil or soiled clothing or implements. Before all pathogens have been transferred to SOIL SURFACE (4), exposure is at the pathogen concentration found in undiluted sludge whereas, after the transfer, the concentration is that calculated for the soil-sludge mixture.

After 840 hours, the time assumed necessary for the lawn to require mowing, the lawn is mowed weekly, and a fraction of the pathogens associated with CROP SURFACE (14) are transferred to DIRECT CONTACT (7). It is assumed that the person mowing the lawn is exposed by inhalation and/or ingestion at each mowing.

REFERENCES

Brady, N.C. 1974. The Nature and Properties of Soil. Macmillan Publishing Co., Inc., New York. p. 266-276.

CFR (Code of Federal Regulations). 1988. Disease, 40 CFR 257.3-6. In: U.S. EPA, Criteria for classification of solid waste disposal facilities and practices, 40 CFR 257.3.

Crane, S.R. and J.A. Moore. 1986. Modeling enteric bacterial die-off: A review. Water Air Soil Pollut. 27: 411-439.

Gerba, C.P. 1988. Alternative Procedures for Predicting Viral and Bacterial Transport from the Subsurface into Groundwater. Draft. U.S. Environmental Protection Agency, Cincinnati, OH.

Kibbey, H.J., C. Hagedorn and E.L. McCoy. 1978. Use of fecal streptococci as indicators of pollution in soil. Appl. Environ. Microbiol. 35: 711-717.

Pao, E.M., Fleming, K.H., Guenther, P.M. and Mickle, S.J. 1982. Foods commonly eaten by individuals: Amount per day and per eating occasion. U.S. Department of Agriculture, Economics Report No. 44.

USDA (U.S. Department of Agriculture). 1975. Soil taxonomy: A basic system of soil classification for making and interpreting soil surveys. Soil Conservation Service. Agriculture Handbook No. 436.

U.S. EPA. 1988. Occurrence of Pathogens in Distribution and Marketing Municipal Sludges. Prepared by County Sanitation Districts of Los Angeles County for the Health Effects Research Laboratory, Office of Research and Development, Research Triangle Park, NC. EPA/600/1-87/014. NTIS PB88 154273.

U.S. EPA. 1989a. Pathogen Risk Assessment for Land Application of Municipal Sludge, Volumes I and II. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. EPA/600/6-90/002A,B. NTIS PB90-171901/AS and PB90-171919/AS.

U.S. EPA. 1989b. Standards for the Disposal of Sewage Sludge; Proposed Rule. Federal Register 54(23): 5886-5887.

U.S. FDA (U.S. Food and Drug Administration). 1978. FY78 Total Diet Studies.

van Genuchten, M.T. and W.J. Alves. 1982. Analytical solutions on the one-dimensional convective-dispersive solute transport equation. USDA Technical Bulletin No.1661.

Wiley, B.B. and S.C. Westerberg. 1969. Survival of human pathogens in composted sewage. Appl. Microbiol. 18: 994-1001.

☆U.S. GOVERNMENT PRINTING OFFICE: 1992 -648 -003/ 41860

United States
Environmental Protection
Agency

Center for Environmental Research
Information
Cincinnati, OH 45268

BULK RATE
POSTAGE & FEES PAID
EPA
PERMIT No. G-35

Official Business
Penalty for Private Use, $300

Please make all necessary changes on the above label, detach or copy, and return to the address in the upper left-hand corner.

If you do not wish to receive these reports CHECK HERE ☐; detach, or copy this cover, and return to the address in the upper left-hand corner.

EPA/600/R-92/064