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Pathogen Risk Assessment Methodology for Municipal Sewage Sludge Landfilling and Surface Disposal

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PREFACE

Section 405 of the Clean Water Act requires the U.S. Environmental Protection Agency (U.S. EPA) 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 assess the potential risk to human health posed by parasites, bacteria and viruses in municipal sewage sludge and to develop preliminary risk assessments for each of these classes of pathogens. This document describes a methodology and computer model designed to assess human health risks from pathogens in landfilled or surface disposed sewage sludge.

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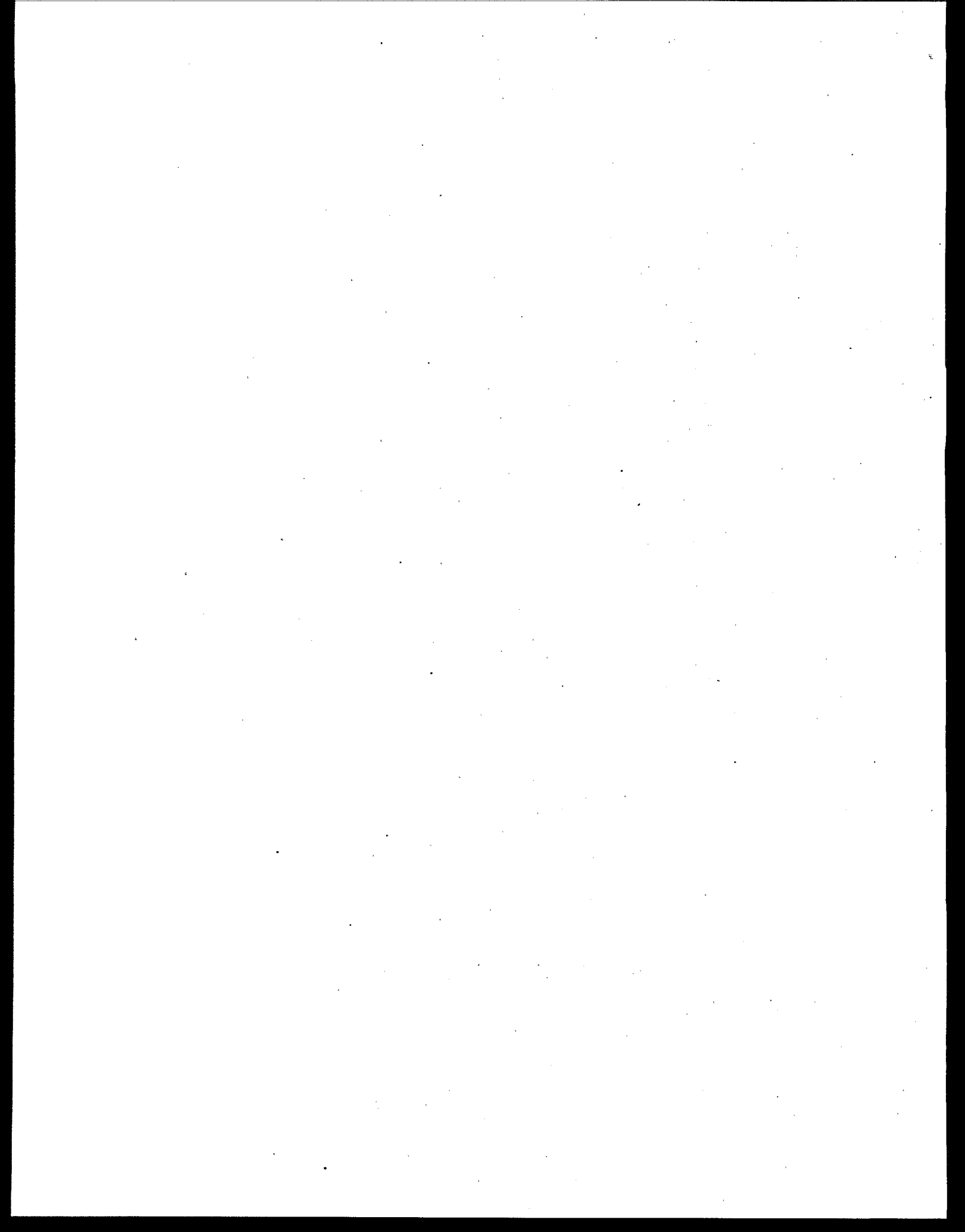
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ABBREVIATIONS AND SYMBOLS

CEC	Cation exchange capacity
CFU	Colony-forming units
D&M	Distribution and marketing
dia	Diameter
ffu	Focus-forming units
GWDR	Ground-Water Disinfection Rule
HAV	Hepatitis A virus
HID	Human infective dose
hr	Hour
ID	Infective dose
K _D	Soil-water partition coefficient
MPN	Most probable number
MPNCU	Most probable number of cytopathogenic units
MULTIMED	Multimedia Exposure Assessment Model for Evaluating the Land Disposal of Wastes
NR	Not reported
PFU	Plaque-forming Units
SRV	Small round viruses
TCID	Tissue culture infectious dose
TSS	Total suspended solids
USDA	U.S. Department of Agriculture
U.S. EPA	U.S. Environmental Protection Agency
WHO	World Health Organization
wt	Weight



1. EXECUTIVE SUMMARY

This document describes a methodology and associated computer model, SLDGFILL (sludge-only landfill or surface disposal), for assessing the risk to humans of pathogens from disposal of treated municipal sewage sludge. The disposal of municipal sludge, produced annually in millions of dry metric tons, is a growing problem. Pathogenic organisms may become concentrated in sludges during treatment processes, posing a potential human health risk when receptors are exposed.

The purpose of the SLDGFILL model is to determine the probability of infection of a human receptor from pathogens in a sludge-only landfill (monofill) or in surface disposal sites. The ultimate objective is to assist the U.S. Environmental Protection Agency (EPA) in its technical criteria development and regulatory activities, but the immediate uses include (1) using the model as a research and risk assessment tool to illustrate information gaps and research needs and (2) applying the model in performing actual pathogen risk assessments.

The exposure pathway addressed by the SLDGFILL model is infiltration from the sludge disposal site to groundwater and subsequent ingestion by a human receptor of groundwater from a drinking-water well. The definition of the human receptor does not include workers exposed in the production, treatment, handling or transportation of sludge. This model is geared toward the protection of the general public, but using infection rather than illness as the measure of risk results in a conservative approach designed to protect sensitive subpopulations. It is assumed that workers can be required to use special measures or equipment to minimize their exposure to sludge-borne contaminants.

In the SLDGFILL model, quantity of treated sludge and other parameters specific to the disposal site are entered by the user. Pathogen parameters required for SLDGFILL include (1) density of pathogens in treated municipal sewage sludge destined for landfilling or surface disposal; (2) infectivity; (3) inactivation rates in sludge, soil and groundwater; and (4) dispersion or transport in the environment. Because many factors affect the density of pathogens in sludge, a wide range of densities has been reported for each type of pathogen found in sludge. These

densities in sludge (liquid, dewatered or dry wt) may be summarized as follows:

Bacteria	$1.4 \times 10^2 - 10^7$ organisms/100 mL $5 \times 10^2 - 8.5 \times 10^4$ MPN (most probable number)/g $\sim 2 - 9 \times 10^6$ CFU (colony-forming units)/g
Viruses	0 - 260 particles/g 1.3 - 410 PFU (plaque-forming units)/100 mL 0.1 - < 2.3 PFU/g 0 - 19 MPNCU (MPN of cytopathogenic units)/100 mL 0.3 - 26 TCID ₅₀ (tissue culture infectious dose for 50% response)/g dry wt
Helminths	$< 10 - 11,000$ ova/kg dry wt
Protozoa	0 - 38,700 cysts/g dry wt 70 - 30,000 cysts/L

The range of reported minimum infective doses for pathogenic bacteria is $10 - 10^{11}$ organisms; for viruses, the range is $9 \times 10^{-1} - 9 \times 10^4$ virus particles, $2 \times 10^{-1} - 5.5 \times 10^6$ PFU, or $1 - 1 \times 10^{7.6}$ TCID₅₀; for protozoa, the range is 1 - 100 cysts; and for helminths, 1 egg has been known to cause infection.

For the model, survival of pathogens in sludge, soil and water is presented in terms of inactivation rate constants (\log_{10} day⁻¹). The most important of the factors that affect pathogen survival in sludge, soil and water are: temperature, survival increasing with lower temperatures; moisture, survival increasing with conditions that encourage moisture retention, such as clay soil or high rainfall; and pH, survival enhanced at median values (pH 5-8). Because of these factors affecting survival, inactivation rate constants based on experimental data may differ by several orders of magnitude, even for a specific pathogen. In general, survival rates for bacteria range from 1.6×10^{-10} /day to 0.96/day and those for viruses range from 2×10^{-4} /day to 0.996/day. Helminths have been reported to persist for up to 15 years in soil, and protozoan cysts have survived from < 1 day to over a year in soil. Thus, the ranking of pathogen persistence in the environment, from longest to shortest, is helminth eggs, viruses, bacteria and protozoan cysts.

The depth to the groundwater presents the greatest barrier to the transport of pathogens and, hence, to exposure and risk. Filtration and adsorption are the processes responsible for limiting pathogen transport through the unsaturated zone. The size of the organism, therefore,

determines which pathogen will be transported the greatest distance. In general, viruses, the smallest of the pathogens considered, have the potential to travel farther in the environment. Large particles like helminth eggs and protozoan cysts typically do not migrate into groundwater because of the physical barrier provided by the soil, unless there are vertical cracks or fissures. Due to their persistence, potential for transport and low infectious dose, viruses seem to represent the worst case when estimating human health risk from landfilling of sewage sludge.

The SLDGFILL model for pathogen risk assessment was run with many combinations of input parameters to simulate the transport of sewage sludge pathogens from a landfill and from a surface disposal site to a nearby drinking-water well. The subsequent risk of infection to humans who drink from the well was estimated for each run. The probability of infection is calculated using a beta-Poisson model. Conservative exposure assumptions include a drinking water consumption rate of 2 L/day and parameters describing highly infective pathogens. Projections by the model predict that the risk of infection from ingestion of bacteria in groundwater is not significant even at 50 m from the sludge source. In contrast, viruses in well water downgradient from a surface disposal site present a potentially significant health hazard to consumers.

The parameters to which the SLDGFILL model are most sensitive are resuspension coefficients, which describe the adsorption of pathogens to sludge and soil particles. Other parameters to which the model is sensitive are infective dose, pathogen density in sludge and inactivation rate in water. Data on infective doses are scarce, making further research necessary for reliable use of the model to predict health risks. It is likely that viruses present a greater health risk because they are expected to have a lower minimum infective dose and are more readily transported through soil.

Future research should be oriented toward satisfying the following information needs to allow more realistic modeling of human health risk from pathogens in landfilled and surface-disposed municipal sludge:

- field data on subsurface transport, in both the saturated and unsaturated zones, of bacteria and viruses;
- inactivation rates of pathogens under field conditions in sludge, soil and water;
- solids-to-water suspension factors applicable to sludge- and soil-bound pathogens;

- leaching characteristics of sludge-bound pathogens;
- interaction of factors affecting pathogen resuspension from sludge and soil; and
- parameters needed to describe infective doses of selected indicator species and strains of pathogens in sludge.

2. INTRODUCTION AND DESCRIPTION OF GENERAL METHODOLOGIC APPROACH

2.1. PURPOSE AND SCOPE

Pathogenic organisms present in municipal sewage sludge pose potential health risks that must be addressed in the evaluation of sludge management (disposal/reuse) options. As a part of its regulatory function, the U.S. EPA develops nationally applicable technical criteria for sludge disposal and reuse based on the potential for adverse health impacts from the sludge. These criteria may regulate concentrations of pathogens in the sludges, as well as regulating other factors within the management practices, such as rates of disposal and process controls. Current sludge disposal and reuse practices include land application, landfilling, incineration and surface disposal. To derive regulatory criteria for pathogens in sludge, the U.S. EPA's Environmental Criteria and Assessment Office is developing a series of methodologies for assessing health risks resulting from land application, landfilling (monofilling) and surface disposal of sludge. This document, which is one in that series, describes a methodology and computer model for evaluating the potential risk to humans from pathogenic microorganisms following landfilling or surface disposal of municipal sewage sludge.

With increasing concern about the importance of uncontaminated groundwater, evidenced by aquifer and well-head protection zones and the proposed Draft Groundwater Disinfection Rule (U.S. EPA, 1992), modeling risk of contamination by pathogens from sludge becomes economically valuable. Better predictive ability concerning pathogen risk allows disposal of sludge by methods that protect human health without requiring levels of treatment beyond what is needed. For example, a knowledge of the relative significance of pathogen densities in sludge, pathogen viability during transport in the subsurface environment and what constitutes a sufficient distance to groundwater wells (setback distance) can be used to design a sludge landfill or surface disposal site whose operation is not likely to adversely affect human health.

The model, SLDGFILL, described in this document calculates the probability of human infection from pathogens in drinking water from a well near a municipal sewage sludge landfill or surface disposal site. This methodology and model, based on the "Sandia Model" (U.S. EPA, 1980), were modified and their development has been continued by Science Applications

International Corporation, Oak Ridge, TN. Previous volumes in this series address pathogen risk from land application of sludge (U.S. EPA, 1989c,d; 1990b; 1991a,b).

This report is not concerned with chemical contaminants in municipal sludge since the U.S. EPA is examining that issue separately (U.S. EPA, 1989b). Also, risks associated with the treatment, transport, handling and accidental release of sludge are not addressed in this document. Codisposal of sludge with solid refuse is regulated under the Resource Conservation and Recovery Act (U.S. EPA, 1989b); no codisposal practices will be considered in this methodology and model development.

The SLDGFILL model is complex enough to represent the major factors determining transport and inactivation of pathogens migrating from a sludge landfill. Yet the model is simple enough to avoid the impractical complexity that requires numerous and often unavailable input parameters. The model runs on a personal computer, and data can be added and modified with no knowledge of programming languages. Although all the information required for an accurate risk assessment is not yet available, additional data can be easily incorporated into the current model, thus improving the model's value and predictive ability.

Use of the model to predict acceptable distances to groundwater wells or outcrops (by running the model iteratively) and implementation of regulatory controls to achieve an acceptable risk level are possible uses of the model to protect human health. When the groundwater disinfection rule is implemented, local utilities may use a pathogen risk model such as SLDGFILL to indicate adequate separation between a pathogen source and a groundwater well, thereby eliminating or limiting the need for groundwater disinfection.

2.2. DEFINITION AND COMPONENTS OF RISK ASSESSMENT

According to the National Academy of Science (NRC, 1983), *risk assessment* is "the characterization of the potential adverse health effects of human exposures to environmental hazards." *Risk management*, by contrast, is "the process of evaluating alternative regulatory actions and selecting among them" by considering available technology, costs and other nonrisk factors.

The process of risk assessment was subdivided into four working components by the National Academy of Science (NRC, 1983). (1) *Hazard identification* is "the process of

determining whether exposure to an agent can cause an increase in the incidence of a health condition...." (2) "The process of characterizing the relation between the dose of an agent...and the incidence of an adverse health effect in exposed populations and estimating the incidence of the effect as a function of human exposure to the agent" is *dose response assessment*. (3) *Exposure assessment* is "the process of measuring or estimating the intensity, frequency, and duration of human exposures to an agent...or of estimating hypothetical exposures that might arise...." (4) "The process of estimating the incidence of a health effect...by combining the exposure and dose-response assessments" is *risk characterization*. The definitions of hazard identification and dose-response assessment have been expanded by the U.S. EPA to include the nature and severity of the toxic effect as well as the incidence (U.S. EPA, 1989a).

2.3. RISK ASSESSMENT IN THE METHODOLOGY DEVELOPMENT PROCESS

The definition of the management practice is the first step in the development of a risk assessment methodology. This methodology and model deal with the landfilling and surface disposal of municipal sewage sludges, the products of typical wastewater treatment processes. Surface disposal refers to disposal of municipal sewage sludge or biosolids on dedicated sites in waste "piles." A surface disposal site is an area of dedicated land on which the sewage sludge remains for at least 1 year or longer (U.S. EPA, 1989e). Surface disposal may also include surface impoundments, or sludge lagoons. Landfilling has been defined as the burial of sludge with a soil cover that exceeds the depth of the plow zone (Walsh, 1978). The management practices addressed include trench landfills, area fills, diked containment landfills, dedicated-site surface disposal and sludge lagoons. These practices are described more fully in Chapter 3.

The following information is required by the SLDGFILL model for risk assessment for pathogens in municipal sewage sludges:

- the sludge reuse or disposal option and the conditions of the application (frequency, quantity, etc.), i.e., specific sludge management practices;
- the types of pathogens present in the sludge, their numbers (level or concentrations), their survival capabilities and parameters describing their virulence; and

- the fate of the pathogens in the environment, including the route of exposure to human receptors, and the magnitude and duration of the exposure.

All of these information requirements and the data available to satisfy them are addressed in this report.

2.3.1. Hazard Identification and Dose-Response Assessment. Infection and disease, the adverse effects on human health resulting from exposure to pathogens, have been identified as hazards in the risk assessment process. For purposes of discussion, sewage-borne pathogens are generally divided into four or five major groups: bacteria, viruses, protozoa, helminths and, sometimes, fungi. The World Health Organization (WHO, 1981), Kowal (1982, 1985) and U.S. EPA (1988a) document the presence of bacterial, viral and parasitic (protozoan and helminthic) pathogens in municipal sludges. Fungi are generally not significant pathogens in sewage except in relation to composting of sludge. Most pathogenic microorganisms found in sewage cause gastroenteric disease of some form, although secondary effects of the organisms may also be important (U.S. EPA, 1989c). The pathogens commonly found in municipal sludges are listed in Table 2-1 and described in Chapter 4.

The pathogenic composition of sludges varies both in type and concentration, depending on many factors including the degree of urbanization of a community, the rate of disease in it, population sanitary habits, population density and season of the year (Fradkin et al., 1985). For this reason, achieving a quantitative hazard assessment for microbial pathogens in municipal sludge is difficult. The U.S. EPA (1988a; 1990b; 1991a,b), Reimers et al. (1981; 1986), Pederson (1981) and Yanko (1988) present extensive surveys of reported levels of pathogenic bacteria, viruses and parasites in treated municipal sludge. Table 2-2 presents a condensed summary of those data.

Because of the lack of available data to support quantitative assessments for all pathogens identified in sludges, representative organisms were selected by the U.S. EPA to act as surrogates in the risk assessment process. In addition to their known presence in sludge and their ability to cause human disease, selection criteria for these surrogates included adequacy of

Table 2-1. Pathogens of Concern in Sewage Sludges

Type	Organism
Bacteria	<p><i>Campylobacter jejuni</i> <i>Escherichia coli</i> (pathogenic strains) <i>Leptospira</i> spp. <i>Salmonella</i> spp. <i>Shigella</i> spp. <i>Vibrio cholerae</i> <i>Yersinia enterocolitica</i> <i>Yersinia pseudotuberculosis</i></p>
Viruses	<p>Adenovirus Astrovirus Calicivirus Coronavirus</p> <p>Enteroviruses Coxsackievirus A Coxsackievirus B Echovirus New enteroviruses Poliovirus</p> <p>Hepatitis A virus Hepatitis E virus Norwalk virus and other small round structured viruses Parvovirus and parvovirus-like agents Reovirus Rotavirus</p>

Table 2-1. (continued)

Type	Organism
Protozoans	<i>Balantidium coli</i> <i>Cryptosporidium</i> spp. <i>Dientamoeba fragilis</i> <i>Entamoeba histolytica</i> <i>Giardia lamblia</i> <i>Isospora</i> spp. <i>Toxoplasma gondii</i>
Helminths	<i>Ancylostoma duodenale</i> <i>Ascaris lumbricoides</i> <i>Echinococcus</i> spp. <i>Hymenolepis nana</i> <i>Taenia</i> sp. <i>Toxocara</i> spp. <i>Trichuris</i> sp.
Fungi	<i>Aspergillus fumigatus</i> <i>Candida albicans</i> <i>Cryptococcus neoformans</i> <i>Epidermophyton</i> spp. and <i>Trichophyton</i> spp. <i>Trichosporon</i> spp. <i>Phialophora</i> spp.
Source: U.S. EPA, 1988b; Gerba, 1983a; Thurn, 1988; Hurst, 1989.	

Table 2-2. Densities of Pathogens in Treated Sludge

Organism	Range of Reported Densities
Bacteria	
<i>Escherichia coli</i>	0.014-10 ⁷ number/100 mL 0.05-10,000 MPN/g 2x10 ⁵ -8.8x10 ⁶ CFU/g
<i>Salmonella</i>	<0.6-1x10 ⁷ number/100 mL >0.1-4.9x10 ³ MPN/g dry wt <0.1-85,000 MPN/g ≥2-<24 CFU/g
<i>Shigella</i>	≥20 CFU/g
<i>Yersinia</i>	10 ⁶ -10 ⁹ number/g wet wt <0.1-2.5x10 ⁶ MPN/g 2x10 ⁵ CFU/g
Viruses	
Enteric viruses	0-260 units/g 0.007-0.04 PFU/mg TSS 0-19 MPNCU/100 mL
Picornavirus	<2.3 PFU/g
Enteroviruses	1.3-410 PFU/100 mL 0.3-260 TCID ₅₀ /g dry wt
Echovirus type 7	0.1 PFU/g
Reoviruses	6-17 PFU/100 mL
Helminths	
<i>Ascaris</i>	565-9600 ova/kg dry wt
Nematodes	100-11,000 ova/kg dry wt
<i>Toxocara</i>	280-1730 ova/kg dry wt
<i>Trichuris</i>	<10-7700 ova/kg dry wt
Protozoa	
<i>Cryptosporidium</i>	1250-38,700 oocysts/g dry wt 140-4000 oocysts/L

Table 2-2. (continued)

Organism	Range of Reported Densities
<i>Giardia</i>	70-30,000 cysts/L
Protozoa	0 cysts/kg dry wt (in D&M sludge)
<p>CFU = colony forming units (number of viable bacteria capable of forming colonies on a particular medium)</p> <p>D&M = distribution and marketing</p> <p>MPN = most probable number (not an actual enumeration but an index of bacteria that more probably than any other number would give the laboratory result)</p> <p>MPNCU = most probable number of cytopathogenic units (most probable number of particles capable of causing cytopathic effects as measured by areas of clearing in a cell culture)</p> <p>PFU = plaque forming units (number of particles capable of causing cytopathic effects as measured by areas of clearing on a cell culture sheet)</p> <p>TCID₅₀ = median tissue culture infective dose (that quantity of a cytopathogenic agent (virus) that will produce a cytopathic effect in 50% of the cultures inoculated)</p> <p>TSS = total suspended solids</p>	
<p>Sources: U.S. EPA, 1988b; Pedersen, 1981; Yanko, 1988; Rao et al., 1986b; Kowal, 1985; Jakubowski, 1990; Sorber and Moore, 1987.</p>	

available data, known minimum infective dose, hardiness outside the human host, survivability typical of other group members and known routes of infection. Representative pathogens selected by the U.S. EPA were *Salmonella* spp. for enteric bacteria, enteroviruses for enteric viruses, *Entamoeba histolytica* and *Giardia lamblia* for parasitic protozoans, *Ascaris lumbricoides* and *Ascaris lumbricoides* var. *suum* for helminths and *Aspergillus fumigatus* for fungi. The three pathogens dealt with in the current version of the model are *Salmonella* spp., representing bacteria; enteroviruses, representing the enteric viruses; and *Ascaris lumbricoides*, representing both helminths and protozoa. As more data become available on other pathogens, pertinent parameters such as infectious dose and inactivation rate may be modified to represent other pathogens such as *Giardia* or rotaviruses.

Dose-response assessment examines the relationship between the occurrence of infections and disease and the exposure to pathogens. The "dose" of pathogens is the number of viable organisms to which a host is exposed, and dose response is (1) no infection, (2) subclinical infection (without apparent illness) or (3) infection with illness. The incidence of disease in a population is likely to increase with an increase in the concentration of pathogens to which the population is exposed.

Risk assessment involves understanding the dose-response relationship for each pathogen identified in sludge. Dose response for a specific pathogen is dependent on the number of organisms required to produce infection or disease in the host. Thus, because of variability among hosts, there are no clearly defined exposure levels that always result in infection, even for a given species or strain. Many factors affect the host response, including the virulence or pathogenicity of the organism, the length of exposure and host characteristics such as site of exposure, degree of immunity, age and general health and prior treatment with antibiotics. The virulence of a pathogen depends to some extent on the susceptibility of the host population and also on the ability of the pathogen to overcome such host defenses as inflammatory and immune responses. Blaser and Newman (1982) observe that organisms that are host-adapted to humans (humans are the only host) may have lower infective doses than nonadapted strains.

The U.S. EPA (1992) recognizes that while one infectious unit, such as a single virus particle or *Giardia* cyst, can cause infection, much larger doses, even orders of magnitude

larger, may be needed to cause disease. Although a much higher number of organisms or infectious particles may be required to produce illness instead of infection, using infection as a detection endpoint would protect more susceptible subpopulations. In other words, avoiding infection is a conservative means for avoiding disease (Regli et al., 1991).

Dose-response data that are available for some bacteria, viruses and parasites are summarized in Table 2-3. The minimum infective dose, the lowest dose that will infect any exposed individual, has been estimated for a few microbial pathogens. Estimated frequency of infection and disease related to probable exposure levels are drawn from epidemiological data, or, in some cases, are based on the exposure of volunteers to known doses. Virus concentrations can also be determined by measuring cytopathic effect by infecting tissue cultures (U.S. EPA, 1990b; 1991a,b).

2.3.2. Exposure Assessment. The exposure assessment step begins with the identification of pathways of potential exposure, that is, migration routes of pathogens from or within the disposal/reuse site to a target organism or receptor. In this pathogen risk assessment model, humans drinking groundwater are the receptors of concern. The potential exposure pathways, described more fully in Chapter 5, include suspended particulates (aerosols), surface water runoff and groundwater. Of the possible routes for pathogens to reach the human receptor, surface water runoff and particulate suspension can be controlled by the use of good management practices, which are defined in Chapter 3 (U.S. EPA, 1989b). Therefore, only groundwater remains a pathway of concern for pathogens in this model.

Human exposure to sludge or contaminated groundwater can be highly variable. Ideally, quantifying exposures of individuals would best assess human risk for any given pathway. However, difficulties of estimating the distributions of each of the parameters involved in the exposure calculations and modeling population distributions and behaviors in the vicinity of the disposal site preclude quantifying the distribution profile for each exposure pathway in this model. By varying the parameters describing exposure, the model user may gain an appreciation for the range of risks that would potentially be encountered by exposed individuals.

Default values, describing reasonable, worst-case assumptions, are provided for testing the model. The compounding of worst-case assumptions, however, can lead to improbable

Table 2-3. Dose-Response Data^a

Organism	Infective Dose	Range
Bacteria		
<i>Escherichia coli</i>	10 ⁴	10 ⁴ -10 ¹⁰
<i>Salmonella</i> (various strains)	10 ² ^b	10 ⁴ -10 ¹⁰ 10 ² -10 ⁵ ^b
<i>Shigella</i>	10-10 ²	10-10 ⁹
<i>Vibrio cholerae</i>	10 ³	10 ³ -10 ¹¹
Viruses		
Echovirus 12	HID ₅₀ 919 PFU HID ₁ 17 PFU predicted	17-919 PFU
Poliovirus	1 TCID ₅₀ < 1 PFU	4×10 ⁷ TCID ₅₀ (infants) 0.2-5.5×10 ⁶ PFU (infants)
Rotavirus	HID ₅₀ ~ 10 ffu ^c HID ₂₅ 1 ffu estimated	9×10 ⁻¹ -9×10 ⁴ ffu ^c
Parasites		
<i>Entamoeba coli</i>	1-10 cysts	1-10 cysts
<i>Cryptosporidium</i>	10 cysts ^d	10-100 cysts ^d
<i>Giardia lamblia</i>	1 cyst (estimated)	NR
Helminths	1 egg	NR
^a Source: Kowal, 1985. ^b Seattle Metro, 1983. ^c Ward et al., 1986. ^d Casemore, 1991. HID = Human infective dose. TCID ₅₀ = Tissue culture infectious dose for 50% response. PFU = Plaque forming units. ffu = Focus-forming units. NR = Not reported.		

results. Therefore, the key to effective use of this model is a careful and systematic examination of the effects of varying each of the input parameters, using estimates of central tendency and upper-limit values to gain an appreciation for the variability of the result.

2.3.3. Risk characterization. Risk characterization consists of combining the results of the exposure and dose-response assessments to estimate the probability of a health effect. Risk assessments ordinarily proceed from source to receptor. That is, the source, or sludge disposal/reuse practice, is first characterized, and contaminant movement away from the source is then modeled to estimate the degree of exposure to the human receptor. Human health effects are then predicted based on the estimated exposure and dose-response relationships. This computer model sums the exposures of a human receptor to pathogens daily and computes the probability of the human receptor receiving an exposure exceeding an infective dose.

2.4. POTENTIAL USES OF THE MODEL IN DETERMINING RESEARCH NEEDS

One of the values of the pathogen risk assessment computer model described herein is its ability to identify areas in which additional research is needed. For example, a major hurdle in any risk assessment is estimating exposure by a variety of routes or pathways to a population that varies according to activity patterns. The use of a conservatively defined human receptor is based, at least in part, on the difficulty in estimating exposure of a population to a changing level, or dose, of pathogens. Information on infectious dose for most pathogens is limited, and distribution of pathogens in soil or groundwater is often unknown. This model assumes random distribution of pathogens in environmental media, but data are not available to verify this assumption. Further research on pathogen exposure pathways and infectious dose levels would facilitate the predictive accuracy of this model and its successors.

Another obvious data gap, illustrated by this methodology and model development, is the degree of survival and transport of pathogens in the environment. Information on the fate of pathogens in sludge, subsurface soil and groundwater is extremely limited. The concentration and survival rates of pathogens leaching through soil into groundwater are unavailable for viruses, protozoa and helminths, while bacterial concentration data are few (U.S. EPA, 1988a). More data are needed concerning the transport of pathogens through sludge and through the unsaturated and saturated zones.

Conducting a sensitivity analysis of the model can reveal areas in which additional research is crucial, as well as areas of low priority. Identifying (1) particular features of sludge disposal practices, (2) properties of the pathogens or (3) characteristics of the model that have a large impact on risk projections can highlight areas in which more research would significantly improve the predictive capability of the model. In contrast, identifying input parameters to which the model is not sensitive shows that research into more precise values for those parameters has a low priority.

Results of risk projections may be unexpected, counterintuitive or contrary to practical experience or good scientific judgement. Unrealistic model outcomes or unexpected sensitivity or insensitivity to input parameters indicates the need for field validation of those results and perhaps additional research on development and refinement of the model. If suitable field data on pathogen survival and transport become available, the many different models for groundwater transport, including SLDGFILL, can be compared to determine which model features are the most important, which ones provide sufficient accuracy and which ones need further refinement.

2.5. POTENTIAL USES OF THE MODEL IN RISK MANAGEMENT

Risk assessment provides the starting point for risk management considerations and the foundation for regulatory decision-making. While the risk assessment is not the sole determinant for regulatory decisions, it provides important information to be evaluated along with societal concerns (costs, benefits, acceptability).

The computer model described in this document can be used to provide information for making and justifying regulatory decisions regarding sludge landfill management practices. Risks associated with different regulatory strategies--establishing acceptable distances to groundwater wells or outcrops, requiring certain thickness of the unsaturated zone or depth to groundwater, specifying conditions of the sludge applications (frequency, duration), and limiting initial pathogen concentrations in sludge--could be compared using the SLDGFILL model. The model would help to identify conditions or actions with the greatest impact on reducing the human health risk. Risk management efforts could then be focused on these actions and conditions to make decisions and establish regulations that will have the greatest influence on protecting human health.

In addition to potential use by risk managers making regulatory decisions, the computer model may be useful for regulators and permit reviewers evaluating proposed sludge landfill sites. Where hydrogeologic conditions of the proposed site are well known, the computer model can be used to estimate the transport of pathogens to a groundwater well and the concentration of pathogens in the drinking water source.

The model could also be used to evaluate proposed regulations and treatment technologies. Risk-based regulations proposed by U.S. EPA (1992) for groundwater disinfection can be evaluated to determine what technology might be used to achieve the proposed exposure limits for groundwater potentially contaminated by leachate from the sludge landfill. For example, alternative disinfection methods may be more effective against different pathogens. By comparing the risks from the different pathogens after their numbers have been reduced by projected treatments, the treatments providing the greatest reduction in risk should be identified. Utilities could also use the model to illustrate sufficient separation between a pathogen source and a groundwater source or wellhead (and thus sufficient health protection), thereby avoiding the need for unnecessary groundwater disinfection.

Over time, as the model is refined by a better understanding of the fate of pathogens in the environment, pathogen inactivation rates and the minimum infective dose in humans, the importance of the model as a management tool will continue to increase.

2.6. LIMITATIONS OF THE MODEL

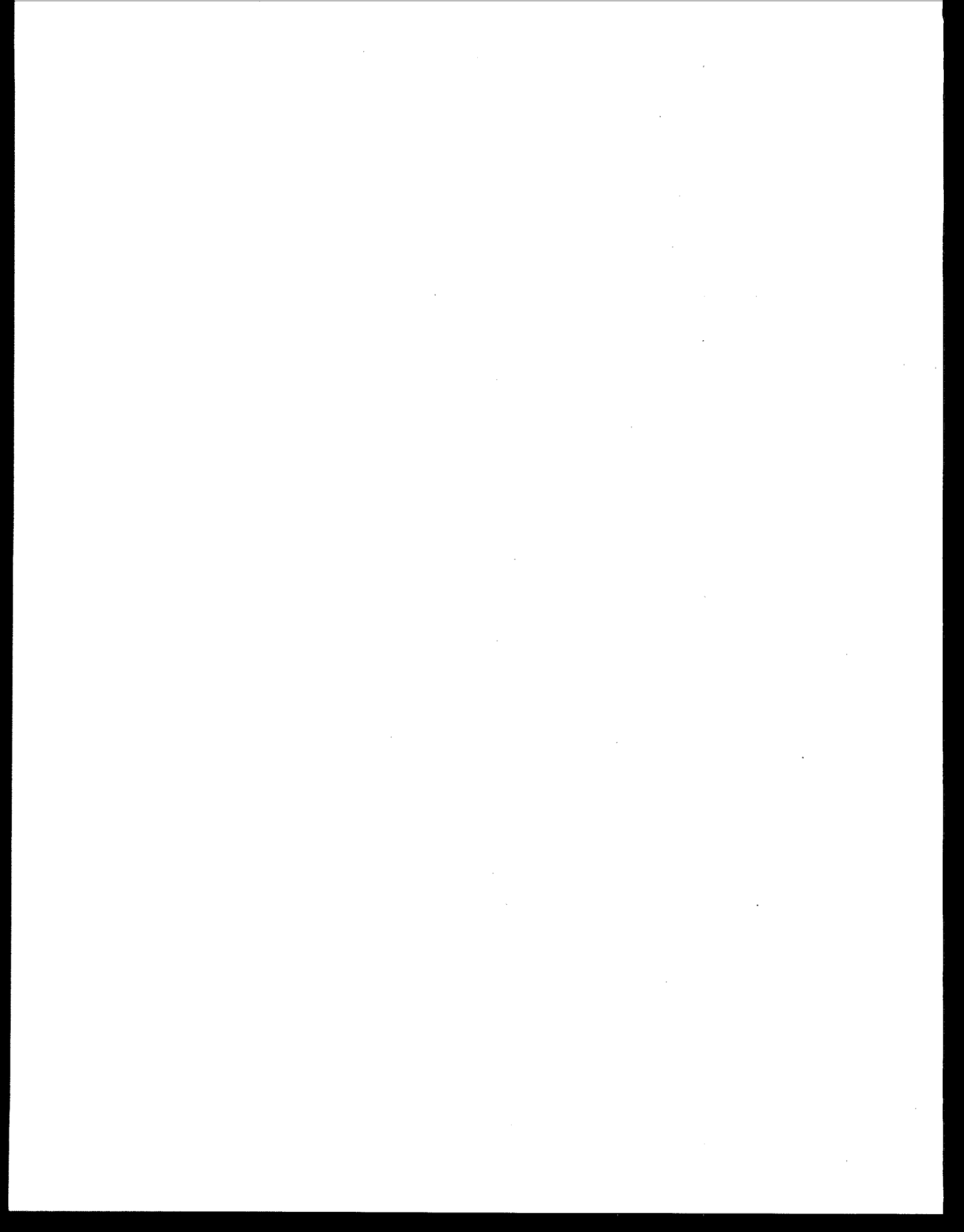
In several cases, assumptions have been simplified to prevent the model from becoming too cumbersome for practical application. If the user were required to input all possible variables, the time required to collect the information and enter it before a model run would be prohibitive. As a result, the flexibility of the model has been restricted to some extent.

The predictive value of the model depends on reliable input parameters and on the accuracy with which initial pathogen concentrations are determined. Municipal sludges are highly variable mixtures of residuals and by-products of the wastewater treatment process, and the distribution of microbial types in sludge will depend in part on the competition among microbes. The model does not address competition among microorganisms. Also, variations

in sewage may result in varying efficacy of treatment, so that the concentration of a particular pathogen cannot be precisely predicted.

Variability in weather or disposal practices is likely to result in differing rates of growth or die-off in sludge, soil, air and water. Although the model does not allow for growth as currently configured, the die-off term (inactivation parameter) could be used to model growth; however, growth of gastroenteric bacteria, such as some *Salmonella* strains, is unlikely to occur in soil beneath a sludge landfill or surface disposal site. Exponential die-off rates are assumed to apply until the end of the practice, even though under certain circumstances linear die-off rates may be more appropriate; consequently, the modeled rates may not be completely realistic. The model does not allow for changes in inactivation rate or variability in groundwater flow.

The model, as currently configured, is limited to one-dimensional, advection/dispersion transport with retardation and die-off, and it only accommodates one pathogen type per model run. As a result, the infection algorithm is simplified.



3. DESCRIPTION OF DISPOSAL PRACTICES

Municipal sewage sludge, a mixture of organic and inorganic semisolids from human activities, is a by-product of the physical, biological and chemical treatment of municipal wastewater in sewage treatment plants. The physical removal of settleable solids from raw wastewater (i.e., primary treatment) produces sludges containing 3-7% solids; further removal of additional solids may be accomplished by the biological and chemical methods of secondary wastewater treatment. The resultant primary and secondary sludges may be subjected to treatment processes designed to reduce sludge volume, improve its workability and lower its potential environmental and health risks. Such treatments include stabilization processes (for example, aerobic and anaerobic digestion, composting and lime treatment), conditioning, disinfecting, dewatering, thickening and drying (Lu et al., 1982).

The disposal of municipal sludges, produced annually in millions of dry metric tons, is a growing problem. Pathogenic organisms become concentrated in sludges during treatment processes, posing a human health risk that affects disposal options and practices (Lu et al., 1982). Decisions on the final disposal of sludge are based on the characteristics of the sludge (e.g., solids content, stability, quantity, toxic compound and pathogen content), local conditions (e.g., site hydrogeology, soil characteristics, climate) and governmental regulations. Because ocean dumping of sludge is restricted, disposal usually means some form of sludge application to land, including application to agricultural and reclaimed land, distribution and marketing programs, surface disposal (e.g., lagooning) and landfilling (Corbitt, 1990).

The scope of sludge disposal practices addressed by the SLDGFILL model includes sludge-only landfilling and surface disposal. The model does not address co-disposal of sludge with refuse, nor does it include incinerated or composted sludges.

3.1. SURFACE DISPOSAL

As explained in the U.S. EPA's final rules for use or disposal of sewage sludge, surface disposal is a term that is used to describe "what are essentially piles of sludge left on the land surface. . ." (U.S. EPA, 1993). A surface disposal site is an area of land on which the sewage sludge has remained for at least 1 year or longer and, typically, on which no daily or final cover

is established on the sewage sludge (U.S. EPA, 1989e). Surface disposal is also used to characterize sludge lagoons. According to U.S. EPA (1990a), surface impoundments or lagoons may be created by waste treatment plants for the long-term storage or treatment of sludges. Storage and treatment lagoons, such as those used for drying, may accumulate bottom layers of sludge that remain in place for years before being removed. A lagoon, an earth basin created for untreated or digested sludge, may be any shape but is usually rectangular. According to Corbitt (1990), drying lagoons are typically dikes with an interior slope of 1:3 vertical to horizontal, a capacity of 35-38 kg/m³/yr, a depth of 0.15-1.2 m and a depth to groundwater of > 1.2 m. Lagooning is not usually considered a method for permanent disposal because lagoons have both odor and insect vector disadvantages. When removal of sludges from impoundments becomes impractical or satisfactory disposal methods are not identified by the waste treatment facility, these impoundments may become permanent disposal sites. Although not technically disposal facilities, lagoons for long-term storage of sludge are included in this surface disposal model because (1) sludge stored in lagoons may pose health or environmental risks similar to permanent disposal facilities, and (2) future plans for sludge removal from storage lagoons may change, resulting in permanent disposal (U.S. EPA, 1990a).

3.2. LANDFILLING

Landfilling, the method most widely used for disposal of sludge (Corbitt, 1990), has been defined as the burial of sludge with a soil cover that exceeds the depth of the plow zone. Sludge may be deposited in sanitary landfills with other refuse (co-disposal) or placed in sludge-only (monofill) trench landfills, area fills or diked containment landfills. Although sludge stabilization prior to landfilling is not required in all states, some degree of stabilization is recommended for sludges before disposal by any landfilling method (Walsh, 1978). Stabilization processes are important in reducing pathogen levels and controlling putrescence; some also dewater and reduce sludge mass. To prevent the contamination of ground and surface waters, landfill leachate and runoff are typically controlled by providing adequate surface drainage, natural attenuation, the use of liners and by collection and treatment. Monitoring wells are typically used to establish baseline groundwater quality, for surveillance during operation and to monitor after closure (Corbitt, 1990).

In trench filling, sludge is confined to excavated trenches, trench depth depending on side wall stability, distance to groundwater and the limitations of the equipment used. *Trench landfills* are recommended only for areas with sufficient depth to groundwater and bedrock and with ground slopes of $<10\text{-}20\%$. Soil is not used for a bulking agent, and hauling vehicles usually dump sludge directly into the trench (Walsh, 1978). Narrow trenches <3 m (10 ft) are used for a single layer of sludge, typically covered by 0.6-1.2 m (2-4 ft) of soil. Sludges with solids content $<15\%$ will not support cover without the addition of a soil bulking agent, which is generally not cost effective (Walsh, 1978). Thus, the recommended minimum solids content for narrow trenches is 15-28%, depending on the width. Trenches wider than 3 m (10 ft) require a sludge solids content $\geq 20\text{-}28\%$ (Corbitt, 1990). Sludge layers may alternate with layers of soil; soil cover is normally 0.9-1.5 m (3-5 ft) depending on the type equipment to be used. The installation of liners is impractical for narrow trenches, but liners are useful to protect groundwater under wide trenches (Lu et al., 1982).

The term *area fill* applies to disposal on the surface of the ground in layers or in a mound. Liners are useful with these methods to protect groundwater, and surface drainage must be controlled. Area fill layer methods can handle sludges with solids content as low as 15%, but mound methods require sludge solids of at least 20%. In these methods, soil is mixed with sludge as a bulking agent to absorb moisture, improve workability and stabilize and improve bearing capacity for operation of equipment. Typically, sludge layers of $\sim 0.3\text{-}0.9$ m (1-3 ft) for layer fills and ~ 1.8 m (6 ft) for mound fills alternate with layers of sludge-soil mixture. Recommended final soil cover thickness is 0.3 m (1 ft) for mound fills and 0.6-1.2 m (2-4 ft) for area fills (Corbitt, 1990). Area fill and diked containment type landfills may be used in areas of shallow groundwater and bedrock and medium to steep terrain (Walsh, 1978).

At *diked containment landfills*, earthen dikes have been constructed creating enclosures above the original soil surface, thereby controlling surface drainage. Often used under conditions such as a high water table or shallow bedrock, this type of disposal is useful for sludges of at least 20% solids. Soil bulking is generally not used. Sludge lifts, or layers, of 1.2-3 m (4-10 ft) may alternate with soil layers, with final soil cover of 0.9-1.5 m (3-5 ft) (Corbitt, 1990). The weight of the applied sludge and soil at the typical depths of these

containments (3-9 m or 10-30 ft) may force moisture from the sludge into the surrounding walls and the soil below the containment, necessitating liners or other leachate controls (Walsh, 1978).

4. IDENTIFICATION OF PATHOGENS

Parameters for microbial pathogens required for the SLDGFILL model include (1) density of pathogens in treated municipal sewage sludge destined for landfilling; (2) parameters describing minimum infective dose; (3) inactivation rates in sludge, soil and groundwater; and (4) dispersion or transport in the environment. Summary data on pathogen density in treated municipal sewage sludge and a compilation of minimum infective doses were presented as Tables 2-2 and 2-3, respectively, in Section 2.3.1 as part of the discussion of hazard identification and dose response. Inactivation rates are discussed in the following sections within the description of each pathogen type. Generally not available for pathogens in sludge, die-off or inactivation rates for bacteria and viruses in soil and water are summarized in Table 4-1. Information on dispersion of pathogens in the environment is limited in its applicability to generating a rate of transport in environmental media. Although data supporting the model parameter requirements are summarized, a more complete review of current knowledge on pathogens is presented in U.S. EPA (1990b; 1991a,b).

4.1. PATHOGENIC BACTERIA

Pathogenic bacteria are found in municipal wastewater and sewage sludge (WHO, 1981; Kowal, 1982, 1985; U.S. EPA, 1988a,b; Pedersen, 1981; Feachem et al., 1983). Most of the bacterial pathogens in sludge are enteric. Their natural habitat is the intestinal tract of animals and humans, and they are members of the families Enterobacteriaceae and Vibrionaceae. Exposure commonly occurs by the fecal/oral route; disease outbreaks are often associated with contaminated food or water. Infection may result in either an asymptomatic carrier state or disease, usually some form of gastroenteritis. Some enteric bacteria may also invade the body from the gut, causing generalized or localized infections.

The U.S. EPA has classified pathogenic bacteria into two categories: those of major concern and those of minor concern. Bacteria of major concern are those commonly found in wastewater and sludges and resulting in disease to the general population. Bacteria of minor concern are opportunistic pathogens that cause disease only in debilitated or immunologically compromised individuals. Bacteria of major concern are listed in Table 4-2. Some bacteria of

Table 4-1. Pathogen Inactivation Rates in Soil and Water

Organism	Inactivation Rate Constant (log ₁₀ day ⁻¹)
Bacteria Soil	
<i>Escherichia coli</i>	0.015-6.39
<i>Salmonella</i>	0.0155-2.99
<i>Shigella</i>	0.268-0.320
Viruses Soil	
Poliovirus type 1	0.0017-0.7077
Viruses	0.057-3.69
Bacteria Water	
<i>Campylobacter fetus</i>	0.156-0.890
<i>E. coli</i>	0.0328-9.8
<i>Salmonella</i>	0.0255-3.01
<i>Shigella</i>	0.0814-0.422
<i>Vibrio cholera</i>	1.00
<i>Yersinia enterocolitica</i>	0.0228-0.0382
Viruses Water	
Coxsackievirus	0.0039-0.2455
Echovirus	0.0039-0.628
Enteric viruses and coliphage	0.174-0.374
Poliovirus	0.0075-2.383
Rotavirus	0.36
Sources: Sorber and Moore, 1987; Moore et al., 1988; Bitton et al., 1983; Hurst, 1988; Hurst et al., 1978; Reddy et al., 1981; Hurst et al., 1989; Yates et al., 1985; Kutz and Gerba, 1988; Cubbage et al., 1979; Keswick et al., 1982.	

Table 4-2. Pathogenic Bacteria of Major Concern in Sewage Sludges

Organism	Disease	Nonhuman Reservoir
<i>Campylobacter jejuni</i>	Gastroenteritis	Cattle, dogs, cats, poultry
<i>Escherichia coli</i> (pathogenic strains)	Gastroenteritis	Cattle (<i>E. coli</i> ϕ 157.H7)
<i>Leptospira</i> spp.	Leptospirosis	Domestic and wild mammals, rats
<i>Salmonella paratyphi</i> A,B,C	Paratyphoid fever	---
<i>S. typhi</i>	Typhoid fever	---
<i>Salmonella</i> spp.	Salmonellosis	Domestic and wild mammals, birds, turtles
<i>Shigella sonnei</i> , <i>S. flexneri</i> , <i>S. boydii</i> , <i>S. dysenteriae</i>	Shigellosis (bacillary dysentery)	---
<i>Vibrio cholerae</i>	Cholera	---
<i>Yersinia enterocolitica</i> , <i>Y. pseudotuberculosis</i>	Yersiniosis	Domestic and wild birds and mammals
Source: Kowal, 1985; U.S. EPA, 1988b; Domingue, 1983.		

minor concern include: *Bacillus cereus*; *Clostridium perfringens*, *Enterobacter* spp., *Francisella tularensis*, *Klebsiella* spp., *Legionella pneumophila*, *Listeria monocytogenes*, *Mycobacterium tuberculosis*, *M. avium* complex, *Pseudomonas aeruginosa*, *Staphylococcus aureus* and *Streptococcus* spp.

Salmonella spp. and *Shigella* spp. are the most common bacterial pathogens in municipal wastewater (Kowal, 1985). Although many *Salmonella* infections are symptomless, most of the serotypes affecting humans produce acute but transient gastroenteritis (Feachem et al., 1983). Direct human transmission of these serotypes is rare. Although the Centers for Disease Control reported only ~49,000 cases of salmonellosis during 1988, most cases go unreported, and there has been a steady increase in incidence over the past 35 years (CDC, 1989). Ingestion of contaminated food or drink is the main cause of an estimated two million cases in the United States per year (Domingue, 1983). Animals, including poultry, farm animals, pets and rats and mice, are an important reservoir of these organisms. Gastroenteritis from salmonellosis is serious only for infants or the elderly with underlying health problems.

Other *Salmonella* serotypes, including *S. choleraesuis*, *S. typhi* and *S. paratyphi*, invade the tissues producing septicemia, typhoid fever (enteric fever) or paratyphoid fevers (Domingue, 1983). Food or water contaminated directly or indirectly from human excreta is the usual source of infection of *S. typhi*; the primary source of infection of *S. paratyphi* is also humans, although animals are a reservoir for the organisms. In areas with high standards for sanitation and public health, these diseases are not prevalent. The mortality rate for paratyphoid is lower than that for typhoid; with the use of appropriate drugs, the mortality rate for typhoid can be as low as 1-2%.

Although the infective dose of *Salmonella* is reportedly relatively high, 10^5 - 10^8 organisms (Kowal, 1985), such numbers may be easily achieved because some *Salmonellae* are capable of significant regrowth in sludges or foodstuffs (Ward et al., 1984). There is also some evidence that infection with *Salmonellae* may occur at lower levels (Blaser and Newman, 1982; D'Aoust and Pivnick, 1976). A wide range of densities of *Salmonella* in treated sludge products has been reported (U.S. EPA, 1991a).

Members of the genus *Shigella* are a major cause of dysentery, producing diarrhea, fever, vomiting and cramps (Feachem et al., 1983). Although foodborne and waterborne outbreaks have been reported, the usual mode of infection is the direct fecal/oral route under conditions of poor hygiene and sanitation (Feachem et al., 1983). The infection may be mild or severe, depending on the health and age of the patient, the serotype of the organism and the infecting dose. Mortality for untreated cases of the severe form may be as high as 25% but usually is much lower.

The infective dose for *Shigella* has been reported to be as few as 10-100 organisms (Kowal, 1985). Difficulties in enumerating the organism partially explain the fact that organisms are seldom found in sewage even when shigellosis is known to be present in the community. Conventional treatments for sewage remove 90-99% of the organisms. Although data on the survival of *Shigella* in sludge treatment processes are few, Feachem et al. (1983) conclude that the conditions of most processes will result in high rates of destruction.

Feachem et al. (1983) suggest that *Campylobacter* is the most common bacterial cause of diarrhea in many countries, including the United States. Stelzer and Jacob (1991) found that raw sewage and river water contained 1.05×10^3 campylobacters/100 mL and typically < 10 campylobacters/100 mL, respectively, but none was found in digested, conditioned sewage sludge. Incidence of campylobacteriosis is similar to that of other enteric pathogens, with contaminated poultry products, unpasteurized milk and nonchlorinated drinking water serving as the main vehicles. Several large outbreaks of waterborne campylobacteriosis involving hundreds of people have been reported (Stelzer and Jacob, 1991). Infection has resulted in septicemia, meningitis, spontaneous abortion and septic arthritis.

The infective dose of *Campylobacter* has not been determined. A recent U.S. EPA study (Yanko, 1988) found no published reports of *Campylobacter* in sludge and did not find *Campylobacter* in the final sludge products of 24 sludge treatment facilities examined. Although the methodology for detecting the organism may have been inadequate, Yanko (1988) concludes that the sensitivity of the organism to oxygen and its susceptibility to drying make it unlikely to persist through composting and sludge drying processes.

The genus *Vibrio* includes several species of enteric human pathogens; the most significant, *Vibrio cholerae*, results in the diarrheal disease, cholera (Feachem et al., 1983). Water and foodborne transmission have been clearly demonstrated, but direct person-to-person transmission may occur. The role of animal reservoirs has not been determined. Untreated cases may result in death from dehydration and loss of electrolytes. Endemic to several areas of Africa and Asia, cholera is difficult to control because asymptomatic and mild infections may be widespread and have major significance to transmission. The ingestion of 10^3 - 10^4 organisms has been shown to cause mild or subclinical infection (Kowal, 1985). Normal gastric acidity is an important defense against infection. There are no reports of reduction of *V. cholerae* numbers during sludge treatment, but studies on the survival of the organism in feces suggest that a process with a retention time >5 days and a warm environment decrease survival. Feachem et al. (1983) conclude that any sludge digestion, composting or storage process will eliminate the organism.

Yersinia enterocolitica causes an acute enterocolitis and septicemia, which are normally self-limiting (Feachem et al., 1983). Animals are reservoirs, but their relationship to transmission is not clear. Transmission by food and water has been reported. Kendall and Gilbert (1980) report that the organism multiplies readily in foods. High levels of *Yersinia* organisms have been reported in some treated wastewater sludges (Metro, 1983; Yanko, 1988), and Langeland (1983) suggests that *Yersinia* may grow in sewage sludge.

Members of the genus *Leptospira* are parasites of rodents. They are transmitted to humans by contact with the urine of animals or water contaminated with animal urine. Infection can result in severe illness that may be fatal, but usually a milder form of the disease results. Although not present in the feces of infected animals and humans, *Leptospira* in urine may result in their presence in sludges. Feachem et al. (1983) conclude that sludge treatments involving anaerobic processes and heat will rapidly inactivate these organisms.

4.1.1. Bacteria Persistence and Inactivation in Sludge. Bacteria become concentrated in sludges during primary (screening and settling) and secondary (biological) sewage treatment processes. Concentrations of pathogenic bacteria in these sludges are lowered by conventional sludge treatment processes. Ward et al. (1984) and U.S. EPA (1988b) report that mesophilic

anaerobic digestion, aerobic digestion, composting, air drying and lime stabilization achieve bacterial reductions of 0.5- > 4 orders of magnitude (an order of magnitude is equivalent to one \log_{10} reduction). According to these reports, composting and lime stabilization processes produce the best reductions of bacterial densities in sludge. The success of a given treatment process in lowering pathogen levels depends, in general, on its retention time and its ability to produce a hostile environment (e.g., thermophilic processes) for pathogens (Feachem et al., 1983). In examining the final sludge products from 24 sludge treatment facilities, including composting, air-drying and heat-treatment facilities, Yanko (1988) reported that densities of bacterial pathogens in sludges varied greatly between facilities and between samples of products from the same facility.

Inadequate or incomplete sludge treatment or recontamination may result in the regrowth of some pathogenic bacteria under the proper environmental conditions. According to Ward et al. (1984), bacteria associated with gastroenteritis are the only bacterial pathogens likely to regrow, and the bacteria that cause salmonellosis are capable of significant regrowth in sludges. Organic content and microbial antagonism are among the environmental factors affecting regrowth. There are fewer competing microorganisms in compost and concomitantly greater nutrient availability for the bacteria; microbial competition or antagonism and less nutrient availability are more probable in a sludge monofill. Hence, bacterial regrowth is less likely in a sludge monofill than in composted sludge.

4.1.2. Bacteria Persistence and Inactivation in Soil. Rates of inactivation of bacteria in soil vary with the strain, the condition of the organism, the method of sludge application, the degree of predation and competition by other microorganisms, atmospheric conditions and the physical and chemical composition of the soil (Moore et al., 1988). Temperature and humidity are the most significant environmental parameters affecting inactivation in soil. In their review of survival and transport of pathogens, Sorber and Moore (1987) found that temperature was the only physical or meteorological parameter related to microorganism survival in sludge-amended soil, survival increasing as temperature decreased. Yates and Yates (1988) suggest that soil moisture is the dominant factor for survival of enteric bacteria in soil. The presence of organic matter in the soil is conducive to pathogen survival not only because of the nutrient value but also because organic matter increases the water-holding capacity of the soil, which allows

enhanced survival of the bacteria. Zibilske and Weaver (1978) observed an interaction between soil moisture and soil temperature in the survival of *S. typhimurium* in two soil types; dry soil conditions and higher temperatures were most detrimental to survival in both soil types.

Hagedorn (1984) summarized information on bacterial transport through the soil: (1) when carried by percolating water in unsaturated soil, bacterial movement is limited to a few dozen cm, but much longer distances are possible under saturated flow conditions; (2) retention of bacteria by soil is inversely proportional to soil particle size; (3) the major limiting factor to bacterial transport through soils is filtration by soil particles; (4) soils containing a greater percentage of clay are more effective in adsorbing bacteria; and (5) the importance of inactivation increases under conditions of unsaturated flow or extended retention of bacteria. Other physical soil characteristics affecting bacterial transport include: organic matter type and content, pH, cation exchange capacity (CEC) and pore size distribution (Moore et al., 1988). Other soil environmental and chemical factors affecting transport include: temperature, chemical makeup of ions in the soil solution and their concentrations, bacteria density and dimensions and nature of the organic matter in the waste effluent solution (concentration and size). However, because rate of transport in soil is strain-specific and there is no consistent pattern in mobility (Alexander et al., 1991), the influence of these factors is difficult to quantify.

4.1.3. Bacteria Persistence and Inactivation in Groundwater. Enteric bacteria show very little growth in groundwater (Matthess and Pekdeger, 1985); their survival in groundwater depends primarily on the biological, physical and chemical conditions of the groundwater and on the processes that control the transport of bacteria. According to Matthess and Pekdeger (1985), enteric bacteria do not flourish in groundwater in the presence of active indigenous microorganisms. Burton et al. (1987) report that survival rates for several species were greater in sediments than in the overlying surface waters, with particle size related to survival. Resuspension of the upper-layer sediments could create a potential health hazard. Gerba (1985) reports that shallow wells are more frequently positive for indicator organisms and average higher densities than deep wells.

Adsorption and filtration limit the movement of bacteria in groundwater. Rate of movement is usually slow unless coarse soils or channels exist. The biologically active layer of sorptive small particles and microbial slimes at the sediment-water boundary is very effective

in filtering out bacteria and preventing their migration to the aquifer (Matthess and Pekdeger, 1985). Continuous adsorption-desorption reactions delay movement of bacteria relative to the groundwater, giving inactivation processes more time to affect bacteria in the saturated subsurface. However, survival time in groundwater may be longer because conditions such as increased moisture, lower temperature and the absence of sunlight and other microorganisms may be favorable.

4.2. VIRUSES

Viruses are more resistant to inactivation than bacteria, and they are smaller and thus more mobile (U.S. EPA, 1992). The presence and pathogenicity of viruses in sewage and sludge are documented in U.S. EPA (1988a,b), Kowal (1985), Feachem et al. (1983) and WHO (1981). The major viruses found in wastewater are listed in Table 4-3. These viruses adsorb to suspended particles and become concentrated in the sludges during wastewater treatment. Although they will not grow in sludges as bacteria will, viruses may persist for many weeks if temperatures are cool (Feachem et al., 1983). Animal reservoirs have not been shown to be significant for the pathogenic viruses likely to be found in sludge, but a few cases have been reported that suggest the transmission of hepatitis A from animals.

Viruses are not normally present in the feces of persons who are not infected, but concentrations of $>10^6$ - 10^9 infectious particles may be in 1 g of feces from an infected person, even if the individual does not exhibit disease (Feachem et al., 1983). Enteric viruses are transmitted primarily from person to person by the fecal/oral route. Transmission occurs by direct personal contact or contact with contaminated surfaces, by contact with recreational water, by ingestion of contaminated food or water and possibly by the airborne route. Inhalation results in infection following the mucociliary translocation and ingestion of viral particles.

The enteroviruses, including polioviruses, coxsackieviruses and echoviruses, comprise a large group of pathogens causing a wide variety of diseases. They normally infect the digestive tract or the respiratory system, causing gastroenteritis or influenza-like disease. Spread to other organs, such as the liver or central nervous system, results in more severe, but generally short-lived, disease. Poliomyelitis, caused by infection of the central nervous system by polio-

Table 4-3. Human Viruses in Sludge and Wastewater

Virus	Disease or Symptoms
Adenovirus	respiratory and eye infection
Astrovirus	may be associated with gastroenteritis, diarrhea
Calicivirus	gastroenteritis
Coronavirus-like Particles	respiratory tract infections, gastroenteritis
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
Hepatitis E	hepatitis
Norwalk virus and other small round structured viruses	epidemic gastroenteritis with diarrhea, vomiting, abdominal pain, headache, myalgia
Papovavirus	may be associated with progressive multifocal leukoencephalopathy
Parvovirus and Parvovirus-like Agents	gastroenteritis, aplastic anemia, fever, rash, fetal death or damage including hydrops fetalis
Reovirus	possibly fever, diarrhea and upper respiratory disease, but relationship to clinical disease in humans is not clear
Rotavirus	acute gastroenteritis with severe diarrhea, vomiting
Sources: Kowal, 1985; Feachem et al., 1983; Kucera, 1983; Akin et al., 1978; U.S. EPA, 1988a; Rao et al., 1986a; Levy and Read, 1990.	

virus, may result in permanent disability or recurring complications later in life. Although most people recover, 4-10% of the paralytic polio cases result in death from respiratory failure (Feachem et al., 1983).

Hepatitis A virus (HAV) occurs endemically in all parts of the world, and it represents the greatest waterborne health threat from viruses because of the severity of hepatitis A illness and because HAV is more resistant to disinfection than many other pathogens (Sobsey et al., 1991). The severity of the disease increases with the age of the victim, but, in general, recovery is complete. Person-to-person contact by the fecal/oral route is the most common method of transmission, but food and waterborne transmission have been reported.

A number of viruses have been found to be associated with diarrheal disease, including the caliciviruses, the coronaviruses, Norwalk agent and other small round viruses; the most significant of these are the rotaviruses, a major cause of childhood gastroenteritis. Transmission is fecal/oral, usually person-to-person, but sometimes by water or food.

Because of the many difficulties in estimation and measurement, reported infective doses for enteroviruses vary widely (Table 2-3). Kowal (1985) reviewed the oral infective dose for poliovirus and found ranges of $1-1 \times 10^{7.6}$ TCID₅₀ and $0.2-5.5 \times 10^6$ PFU. Schiff et al. (1984) reported the human oral 50% infective dose (HID₅₀) for echovirus was 919 PFU in volunteers and predicted an HID₁ (1% infective dose) of 17 PFU. When rotavirus was administered orally to volunteers, Ward et al. (1986) found an HID₅₀ of ~ 10 focus-forming units (ffu) and estimated that $\sim 25\%$ of susceptible adults would be infected by 1 ffu. Regli et al. (1991) present data suggesting a minimum infective dose for rotavirus of ~ 3 . These and other data suggest that the infective dose of enteroviruses to humans is 10 or fewer infectious virus particles. A review of the health significance of viruses in water suggested that the minimum infective dose of enteric viruses in healthy adults may be larger than 1 PFU, but a single PFU may be infective to susceptible individuals (IAWPRC, 1983).

The U.S. EPA (1992) has developed a proposed Ground-Water Disinfection Rule in which they propose using a "synthetic virus" for risk calculations. On the basis of model calculations, a limit of 2×10^{-7} viruses/L has been proposed as a target limit in groundwater (Regli et al., 1991). The synthetic virus would combine properties of several viruses--enteroviruses representing worst-case waterborne occurrence, rotaviruses for dose-response and

HAV for estimating disinfection efficiency. This combination of properties would create a reasonable worst-case situation. Because this proposed rule is currently a draft and will not be promulgated until 1995, SLDGFILL uses a representative enterovirus. However, the model could be configured to use the synthetic virus, which would result in a more conservative risk assessment.

4.2.1. Virus Persistence and Inactivation in Sludge. A range of 2-215 enteric virus units/g of raw sludge has been reported in the United States (Gerba, 1983a); Pedersen (1981) reports average geometric mean values for enteric viruses of 390 PFU/g dry wt in primary sludge, 320 PFU/g dry wt in secondary sludge and 360 TCID₅₀ in mixed sludge. Viruses are less easily removed by treatment processes than bacteria; viruses may be protected in sludge by adsorption and association with solids. The U.S. EPA (1991b) records mean densities of enteroviruses in digested sludges of 3.3-138 PFU/100 mL of sludge and 0.3-53 TCID₅₀/g dry wt of sludge. Mean densities of 0-260 enteric virus units/g sludge and 0.007-0.04 PFU/mg of total suspended solids are reported for enteric viruses. Determining the fate of hepatitis A, rotaviruses and other viral agents of gastroenteritis in sewage and sludge treatment processes has been hampered by inadequate isolation techniques for the viruses (Feachem et al., 1983). 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 periods to ensure that all parts of the mass are heated." Rao et al. (1986a) suggest that higher temperatures may be necessary because HAV was infective at 80°C in the presence of high concentrations of some salts. Ionic detergents have been shown to protect poliovirus from heat in raw sludge (Ward et al., 1976). Although aqueous ammonia speeds inactivation of enteroviruses at moderate temperatures, adsorption of the virus particles to sludge solids may protect them from heat inactivation and from inactivation by ammonia (Ward and Ashley, 1978).

4.2.2. Virus Persistence and Inactivation in Soil and Water. The most important factor influencing persistence and inactivation of viruses in both soil and water is temperature, with lower temperatures enhancing survival and infectivity (Yates and Yates, 1988). Temperature and desiccation may act synergistically to influence the fate of viruses in soil (Gerba and Bitton, 1984). Yeager and O'Brien (1979a,b) suggest that the mechanisms for viral

inactivation in moist soils differ from those in dry soils and that inactivation occurs during the drying process.

Microbial activity may play a role in the inactivation of viruses in soil and water; investigations have yielded inconsistent results. Hurst (1988) suggests that microbial antagonism in soil results from the metabolic products of bacteria or bacterial interference with viral adsorption onto soil particles. Yates et al. (1990) examined filtered and unfiltered groundwater and concluded that bacteria may produce a substance that inactivates viruses. Viruses survive well at the pH levels of natural waters (pH 5-9) (Bitton et al., 1987), and Bitton (1978) asserts that enteric viruses will not be affected by the pH values of the natural environment. According to Salo and Cliver (1976), virus persistence relative to pH in the aqueous environment varies with the type of virus. Gerba and Bitton (1984) suggest that pH may have indirect effects on inactivation by affecting adsorption. Although conflicting reports in the literature indicate that the relationship is not clear-cut, adsorption may be minimal at alkaline pH values. Pancorbo et al. (1987) report that inactivation of human rotavirus type 1 was significantly correlated with water pH, with inactivation increasing as pH increases.

The association of viruses with organic and inorganic particles in water appears to enhance survival, possibly by protecting viruses from light, heat and biologic degradation. Yates and Yates (1988) conclude that the survival of viruses in soil may be enhanced or reduced by adsorption to soils or other materials, depending on the sorbent, and that organic matter competes with virus particles for sites on soil particles. Gerba (1984) reports that organic material acts as an eluting agent, desorbing viruses from the soil. Gerba (1985) and Hurst et al. (1980) found that viral survival increased with greater adsorption by soil.

Yates and Yates (1988) conclude that many soil properties influence viral persistence in soil by affecting the degree of adsorption to soil particles. Hurst et al. (1980) suggest that viral adsorption increases with high aluminum levels and with lower levels of resin-extractable phosphorus. Clay minerals may increase viral adsorption to soil, and clay can be protective to the viral genome (Gerba and Bitton, 1984). According to Sobsey (1983), increasing the soil CEC increases virus adsorption. Minerals have been shown to be better adsorbents than soils in some studies (Sobsey and Shields, 1987). According to Bitton (1980), virus persistence in

natural water is affected by the formation of aggregates, possibly by protecting the aggregated virus particles from environmental factors.

Inactivation of enteroviruses in the environment can be influenced by salt species and their concentrations. Yates and Yates (1988) report that the type and concentration of salts in the soil affect virus adsorption to soil and that the presence of some cations in the media is protective against heat for some viruses.

In their study of groundwater in the United States, Yates et al. (1985) found that water characteristics such as hardness, turbidity, total dissolved solids, nitrate, ammonia, sulfate and iron were not significantly correlated with the inactivation of three viruses at in situ temperatures. However, Jansons et al. (1989) found that the viral inactivation rate increased with an increase in the dissolved oxygen in groundwater.

Adsorption to particles and susceptibility to inactivation in soil and water vary with the type of virus and the particular strain. HAV has been shown to persist longer than polio and echovirus at 25°C in groundwater, wastewater and soil suspensions.

In summary, virus inactivation varies by species and as a result of environmental, physical and chemical factors. Table 4-1 gives an indication of the ranges of inactivation rates for several viruses in soil and in water. This range of values has been used in SLDGFILL to model virus risk from a sludge monofill to a nearby human receptor.

4.2.3. Virus Transport in Soil, the Subsurface and Groundwater. The potential for virus removal is greater in the unsaturated zone than in the saturated zone because of adsorption, filtration and other retardation processes, which increase the likelihood of inactivation (Yates and Ouyang, 1992). Viruses applied to soil in sludge will be less mobile than those in sewage water because they will be adsorbed to sludge solids (Lance and Gerba, 1982). The application rate of sludges will affect the number of viruses passing through the soil to the groundwater (Rao et al., 1986a).

Adsorption is the primary mode of removal of free virions in soil, desorption allowing further transport through the soil (Yates and Yates, 1988). Initially, most viruses applied to soil are retained in the upper soil layers (Rao et al., 1986a). Lance et al. (1976) suggest that viruses near the surface desorb and migrate vertically through the soil with periodic rainfall; Lance and Gerba (1984) found that poliovirus moved farther in columns of loamy sand under saturated

conditions than under unsaturated flow conditions. Lance and Gerba (1980) suggest that virus adsorption is not affected by increases in water 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, allowing little contact between viruses and soil particles. Column and field studies suggest that water flow velocity is possibly the most important soil characteristic affecting virus movement in soil (Lance and Gerba, 1982).

Coarse-textured soils that do not adsorb well promote migration of viruses. In field studies, Lance and Gerba (1982) found that movement of viruses to groundwater was a problem primarily in coarse sandy or gravelly soils. Soils with high CEC and large surface area, such as clay, are active in virus adsorption (Yates and Yates, 1988), and some minerals have been shown to increase virus adsorption and retention in soil (Sobsey and Shields, 1987). However, soluble organic matter reduces virus adsorption, thereby enhancing virus persistence and mobility in soils (Gerba and Bitton, 1984).

Sobsey and Shields (1987) report a number of studies indicating greater viral adsorption in neutral and acidic materials. Gerba et al. (1981) found that for poorly adsorbed viruses (including coxsackie B4 viruses, echo 1 viruses and phage MS-2) viral adsorption to soil was greatly affected by pH, as well as by CEC and organic matter, but for 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. Although high pH values will promote virus desorption and migration in the soil, these pH values are expected only under experimental conditions (Rao et al., 1986a).

Increasing the concentration of ionic salts increases virus adsorption to soil particles, retarding virus transport (Sobsey, 1983). During rainfall, the ionic strength of the soil water decreases, and desorption and redistribution of viruses creates the potential for groundwater contamination (Gerba, 1983b). Gerba and Bitton (1984) report that remobilization varies with the nature of the soil and with virus type and strain. Adsorptive capacity is both type- and strain-dependent (Gerba et al., 1980). The electronegativity of a virus type is determined by the physicochemical composition of the capsid surface, and desorption and migration in the soil are affected by this charge on the particle (Rao et al., 1986a).

Because viruses are small in diameter, porous aquifers are not effective in filtering out virus particles; the large flow paths permit rapid passage of suspended viruses (Matthess and Pekdeger, 1985). Passage through loamy aquifers with high cation concentrations effectively removes viruses that adsorb well, retarding transport and providing time for inactivation to occur. Also, the microbial slime and sorptive small particles at the water/sediment boundary impede transport. Heavy rainfall may result in decreased cation concentration and further virus transport.

Viruses, because of their small size, mobility, resistance to inactivation and low infective dose, are the most likely of the pathogens to result in a potential health risk as a result of sludge landfilling. Although virus transport in the subsurface has been studied and modeled, there is little quantitative laboratory or field data to determine the relative impacts of climatic, biologic, chemical or physical factors on subsurface transport, particularly in the unsaturated zone.

4.3. PROTOZOAN PARASITES

WHO (1981), Kowal (1982, 1985) and U.S. EPA (1988a) document the presence of protozoan parasites in sludge at different stages of treatment. Table 4-4 lists the protozoa, commonly found in sewage sludge, that are significant human pathogens. Because animals are often reservoirs of protozoan parasites, sludges containing animal wastes may have high levels of protozoa. Protozoa are present in sewage and sludge as cysts and oocysts, dormant structures resistant to adverse environmental conditions (U.S. EPA, 1990b). Epidemiological evidence suggests little risk to human health from parasites in municipal sludge, but their persistence in the environment and their low infective doses mean that protozoa in sludge cannot be dismissed from health risk considerations (Kowal, 1985). However, the large size of protozoan cysts, relative to viruses and bacteria, makes transfer of the cysts to groundwater after landfilling or surface disposal of sludge unlikely (Kowal, 1985).

The protozoan pathogens cause a variety of symptoms, including enteritis, diarrhea and dysentery, by colonizing the gastrointestinal tract of humans and other mammals. Kowal (1985) identifies the protozoan parasites of greatest human health significance in sludge as *Balantidium coli* (balantidiasis), *Entamoeba histolytica* (amebiasis) and *Giardia lamblia* (giardiasis). All are

Table 4-4. Protozoa of Concern in Sewage Sludge

Pathogen	Effect/Disease	Nonhuman Reservoir	Human Infective Stage
<i>Balantidium coli</i>	Balantidiasis	pigs, other mammals	cyst
<i>Cryptosporidium parvum</i>	Cryptosporidiosis	cattle, sheep and other domestic and wild animals	oocyst
<i>Dientamoeba fragilis</i>	Amebiasis		unknown
<i>Entamoeba histolytica</i>	Amebiasis (amebic dysentery)		cyst
<i>Giardia lamblia</i>	Giardiasis	mammals	cyst
<i>Isospora belli</i> , <i>I. hominis</i>		dog dog	oocyst oocyst
<i>Toxoplasma gondii</i>	Toxoplasmosis	cat	oocyst
Source: Kowal, 1985; U.S. EPA, 1988b; Sorber and Moore, 1987.			

transmitted by water contaminated with cysts, and *Entamoeba* and *Giardia* are also transmitted by contaminated food. Amebiasis, giardiasis and balantidiasis are often asymptomatic infections (Kowal, 1985); the diseases may be debilitating, but they are rarely fatal in developed countries. However, severe cases of amebiasis may produce liver, lung or brain abscesses and death. *Cryptosporidium parvum*, a coccidian protozoan, has recently been recognized as a widespread pathogen of humans and animals with a potential for waterborne transmission equal to or greater than *Giardia* (Rose, 1988; Current, 1987); it has been the cause of several outbreaks of waterborne disease. Cryptosporidiosis ordinarily produces mild to severe diarrhea, but in immunologically compromised individuals, it may result in a life-threatening cholera-like illness and may not be confined to the gastrointestinal tract.

The infectious dose of protozoans has been determined to be small, as few as one cyst of *Giardia* or 10-100 oocysts of *Cryptosporidium* (Casemore, 1991).

4.3.1. Persistence in Sludge. Trophozoites, the active stage of flagellate protozoans, and sporozoites; the active stage for coccidian protozoans, can become precysts following a period of reproduction. These precysts can secrete a tough membrane to protect the parasite (Kowal, 1985). It is these thick-walled, environmentally resistant, dormant structures that are excreted in the feces and are found in sewage and sludge. These forms are capable of causing human infection and are, therefore, the source of concern, if any, in landfilled sewage sludge (U.S. EPA, 1990b).

There is little information on the survivability of *Giardia* and *Cryptosporidium* during sludge treatment processes. According to Reimers et al. (1981) and Leftwich et al. (1981), 99% of primary municipal sludges and 89% of final municipal sludges from southern states contained large numbers of viable parasite cysts and ova. Direct counts (centrifugation) of *Giardia* cysts in 11 municipal secondary sludges ranged from 70-30,000 cysts/L (Sykora et al., 1991). The highest average concentration (1723 cysts/L) was in dewatered but not digested sludges. Yanko (1988) examined the final sludge products from 24 treatment facilities, including composts, air-dried and heat-treated sludges, and found no protozoan cysts. Kaye and Rose (1987) reported concentrations of *Cryptosporidium* ranging from 1250-38,700 oocysts/g dry wt in anaerobically digested sludges. Filtration at water treatment plants removes *Cryptosporidium* oocysts, which

are resistant to routinely used disinfectants (Fayer and Ungar, 1986; Current, 1987; Rose, 1988). Table 2-2 illustrates the wide range of densities of protozoan cysts and oocysts in treated sludge, from as few as 70 cysts/L to as many as 38,700 oocysts/g dry wt; some researchers found none at all in multiple sludge samples.

~~4.3.2. Inactivation of protozoan cysts in soil~~
the survival of protozoan cysts in soil. The U.S. EPA (1990b) documents the sensitivity of the cysts of *Entamoeba histolytica* to drying, with longer survival in moist soils. Yanko (1988) reports protozoan cysts survive a maximum of 10 days on soil; Frenkel et al. (1975) found that cysts of *Toxoplasma* species survived up to 410 days in soil.

The persistence of *Cryptosporidium* oocysts and *Giardia* cysts in water is well documented (Madore et al., 1987; Hayes et al., 1989). Jakubowski (1990) reports that *Giardia* cysts survive best in water at temperatures of 4-8°C and that temperatures below 20°C allow the cysts to survive for long periods. Survival time of *Entamoeba histolytica* in water is also temperature dependent, survival decreasing with a rise in temperature. *Cryptosporidium* oocysts were not infective after exposure to temperatures below freezing or above 65°C (Tzipori, 1983). Kaye and Rose (1987) report survival of protozoan cysts in water in the laboratory for > 140 days.

Unless there are vertical cracks or fissures, protozoan cysts are large enough (~5-25 µm dia) that they do not migrate vertically through soil into the groundwater (U.S. EPA, 1988b). For example, Seattle Metro (1983) found that cysts of *Entamoeba histolytica* were unable to pass through 61 cm (24 in.) of sand. So, despite their relatively long survival time in soil and water, protozoan cysts are unlikely to be a health risk in groundwater.

4.4. HELMINTH PARASITES

WHO (1981), Kowal (1982, 1985) and U.S. EPA (1988a) survey helminth parasites present in sewage sludges and discuss the associated diseases. The pathogenic helminths, some of which are only incidental parasites of humans, include both the nematodes (or pinworms, roundworms and whipworms) and the cestodes (or tapeworms). Most have animal reservoirs. Table 4-5 shows the significant human pathogenic helminths that are most commonly found in

The dwarf tapeworm, *Hymenolepis nana*, requires no intermediate host and inhabits the human intestinal tract. Light infections may be asymptomatic, but anorexia and digestive disturbances may occur. Humans may serve as the intermediate host for the pork tapeworm, *Taenia saginata*, by ingestion of feces contaminated with eggs. Cysticercosis results as the larvae migrate into tissues and encyst. Localization of the migrating larvae in the ear, eye, central nervous system or heart may be quite serious. When humans are the intermediate host for the dog tapeworms, *Echinococcus granulosus* and *E. multilocularis*, hydatid cysts formed by larvae in body organs can cause serious problems as the cysts grow or rupture.

A single helminth egg may produce human infection; however, because infection is dose-responsive, many infections are asymptomatic (Kowal, 1985).

4.4.1. Persistence in Sludge. Helminths typically reproduce in the gut and generally require more than one host to complete their life cycle. The adult worm typically lives in the gut of the definitive, or final, host and sheds fertilized ova, either free or in proglottids, in the feces. Helminth ova are the resistant stage found in sludge (U.S. EPA, 1990b).

Schwartzbrod et al. (1989) report that ordinary wastewater treatments, such as activated sludge, lagoon treatment and sand filtration, concentrate parasite eggs in sludge and lower the density in wastewater effluent. Temperatures $>55^{\circ}\text{C}$ during aerobic and anaerobic digestion are lethal to parasite eggs (Leftwich et al., 1981); drying beds are effective for inactivating eggs when moisture is $\leq 5\%$. Black et al. (1982) report that survival and viability of helminth eggs during mesothermic digestion varies with the type of digestion, anaerobic or aerobic, and the species of helminth. Fewer viable *Ascaris* and *Toxocara* eggs were found in anaerobic sludges than in aerobic sludges, possibly because of the higher temperatures in anaerobic digestion (Reimers et al., 1986). Mbela et al. (1990) conclude that high temperature is a significant factor in *Ascaris* inactivation during aerobic and anaerobic digestion, and that liming and caustic stabilization increase inactivation. High temperatures have also been reported to be more effective for inactivating parasite eggs during lagooning (O'Donnell et al., 1984) and for decreasing survival of *Taenia* eggs during other sewage treatment processes (Storey, 1987). At temperatures $<51^{\circ}\text{C}$, heating alone for 1 hour was not effective in destroying viable ova, but heating to 55°C for 15 minutes was lethal (Pike et al., 1988).

Although Yanko (1988) reported that helminth ova were detected regularly in the sludge products from the 24 sludge treatment facilities studied, no indications of viability were observed. One or more of *Ascaris* spp., *Toxocara* spp., *Trichuris trichiura* or *Trichuris vulpis* were detected in 89% of the municipal sludge samples from plants in four northern states; densities of eggs were 565, 265, 270 and 370 eggs/kg dry wt, respectively (Reimers et al., 1986).

4.4.2. Inactivation and Transport in Soil and Water. The eggs and larvae of helminths are sensitive to desiccation and sunlight, but under cool, moist conditions they may survive and remain infective for years (Kowal, 1985). *Ascaris* eggs may survive in soil up to 15 years (U.S. EPA, 1988b). Although some field studies indicate that subsurface conditions are more conducive to survival of helminth eggs than conditions at the soil surface, other studies have failed to find a correlation between viability and solar radiation, relative humidity or soil temperatures. Leftwich et al. (1988a) reported that repeated freeze-thaw conditions reduced viability of *Ascaris* eggs, with greater soil-moisture promoting viability despite temperature.

Sorber and Moore (1987) conclude that the size of helminth ova prevents their vertical migration through soil. Based on their measurement of the rate of transport of *T. saginata* and *A. lumbricoides* ova in soil columns, Storey and Phillips (1985) calculated that the ova would move 100 cm in 65 years assuming an average annual rainfall of 152 cm (60 in.).

No data were found on survival or transport of helminths in water.

4.5. FUNGI

Pathogenic fungi can be divided into the yeasts (*Candida* spp., *Cryptococcus neoformans* and *Trichosporon* spp.) and filamentous molds (*Aspergillus* spp., *Epidermophyton* spp., *Phialophora* spp. and *Trichophyton* spp.). Because these fungi are ubiquitous in nature, even pasteurized sludges may become recontaminated (WHO, 1981). Therefore, it is difficult to evaluate their public health significance. *Aspergillus fumigatus* is prevalent in municipal compost, but composted sludge is not buried in landfills (U.S. EPA, 1988a) because of its capacity for beneficial reuse.

4.6. PATHOGEN SUMMARY

The parameters required for the SLDGFILL model--density of pathogens in sludge, minimum infective dose, inactivation rates and data on transport in the environment--have been described. Summary data on pathogen density in treated sludge and on minimum infective doses were presented in Tables 2-2 and 2-3, and inactivation rates are summarized in Table 4-1. The ranking of pathogen persistence in the environment, from longest to shortest, is helminth eggs, viruses, bacteria and protozoan cysts (U.S. EPA, 1988a). The most important of the factors that affect pathogen survival in sludge, soil and water are temperature, survival increasing with lower temperatures; moisture, survival increasing with conditions that encourage moisture retention, such as clay soil or high rainfall; and pH, survival enhanced at median values (pH 5-8). The groundwater pathway is the most significant route of human exposure following surface disposal or landfilling (see Chapter 5). Depth to groundwater presents the greatest barrier to the transport of pathogens and hence to exposure and potential risk. Filtration and adsorption, which delay transport and allow more time for inactivation, are the processes responsible for limiting pathogen movement through the unsaturated zone. The size and surface charge of the organism, therefore, determine which pathogen will be transported the greatest distance. Viruses, the smallest of the pathogens considered, have the potential to travel farther in the environment. Large particles like helminth eggs and protozoan cysts do not migrate into groundwater because of the physical barrier provided by the soil, unless there are vertical cracks or fissures. Therefore, viruses seem to present the greatest potential for producing human health effects from landfilling or surface disposal of sewage sludge.

5. IDENTIFICATION OF EXPOSURE PATHWAYS

Considering current sludge surface disposal facility or landfill design and operating practices, the potential pathways for offsite migration of pathogens can be summarized (U.S. EPA, 1989b,e) as illustrated in Figure 5-1:

- groundwater infiltration--infiltration of water and drainage of leachate from sludge, transporting pathogens to the underlying aquifer;
- surface runoff--suspension of pathogens in surface runoff from the sludge working face and transport to nearby surface waters (this pathway is not relevant to trench monofills, because the sludge is placed below the surface in an enclosed trench); and
- particulate suspension--suspension in air of particles and pathogens from the working face with subsequent transport downwind.

5.1. GROUNDWATER INFILTRATION

Infiltration of water from and through the sludge into groundwater and subsequent uptake of pathogens in drinking water is considered the most significant of the potential exposure pathways (U.S. EPA, 1989b,e). Sludge lagoons, surface disposal sites or landfills receiving recharge will allow formation of leachate and downward migration of that leachate to groundwater. Groundwater may be used as drinking water or to irrigate crops, water livestock, provide drinking water for wildlife or serve as recharge for surface water habitats for edible aquatic organisms. Because the greatest threat and most likely occurrence of health effects would result from drinking groundwater contaminated by this leachate, risk evaluations will be based on drinking water concerns. Each of the other pathways is considered as a supplementary, or less serious, groundwater pathway. Therefore, the most conservative assumption for evaluating risk from pathogens in sludge is direct ingestion of groundwater from a drinking-water well near the landfill site.

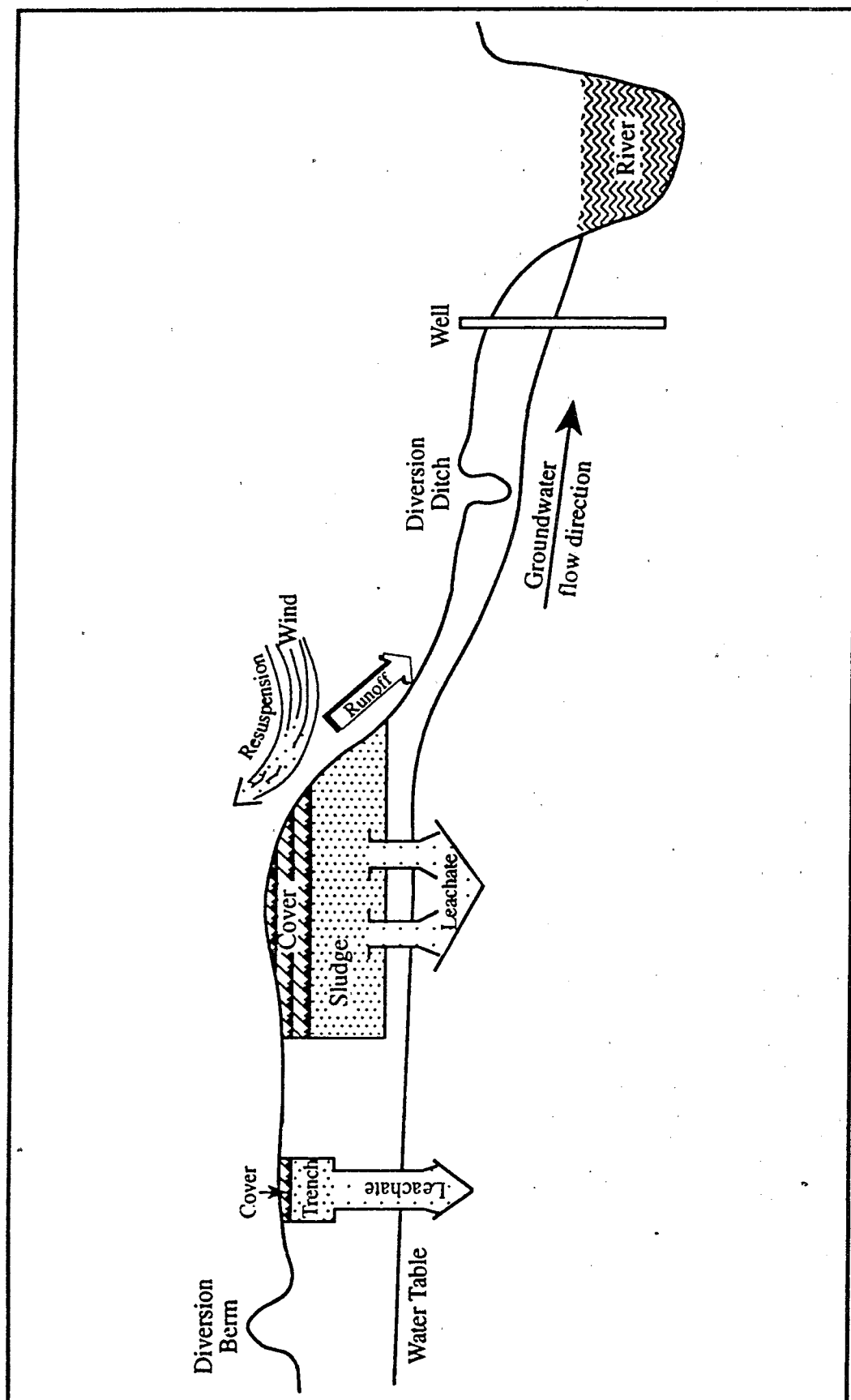


Figure 5-1. Offsite Migration Pathways for Pathogens from Sewage Sludge Landfills or Surface Disposal Sites

5.2. SURFACE RUNOFF

Pathogen migration in surface runoff may result from suspension of pathogens in surface water following contact between sludge and runoff. Therefore, pathogens must be present at the surface of the sludge or soil for pathogen migration by this route. Because good management practices require the use of clean soil for cover, the working face is the only significant source for pathogen-contaminated runoff. As explained in the description of disposal practices, operating procedures require drainage ditches or dikes to control runoff and runoff from the working face. All trench fills and sludge lagoons will contain runoff by design, since the working face is below grade relative to the surrounding areas. Area fills must include design provisions to prevent runoff from upgradient and to contain drainage downgradient. Assuming good operating practices, runoff becomes a part of the groundwater pathway or is eliminated. Precipitation that runs off the working face will collect below the face in a drainage control ditch where it will percolate into the soil, be used for dust control or be routed to treatment. In the first two instances, the runoff becomes part of the groundwater pathway. In the third instance, the pathway is terminated. Therefore, the surface runoff pathway is not considered in this model.

Because good sludge management practices will prevent the surface runoff pathway from being a significant route for exposure from pathogens in sludge, regulations to control risk from this pathway are best focused on requiring these practices: diversion dikes/berms or ditches to redirect runoff from adjacent areas away from the disposal area, and berms or ditches at the foot of the disposal area to collect runoff from that area and from the working face. These berms and ditches should be designed to contain the estimated runoff from a 24-hr, 100-yr storm event (U.S. EPA, 1989b, 1990a).

5.3. PARTICULATE SUSPENSION

Like the surface runoff pathway, the particulate suspension pathway requires pathogen-bearing sludge to be at the surface where it can be disturbed by wind or human activity. The working face is the only location where this occurs to any appreciable extent. With application of daily cover, the working face will only be exposed for a period of 8-12 hours in a 24-hour period. Furthermore, particulate suspension will occur only above a given wind scour threshold

velocity or with mechanical disturbance. Most sludges would not be susceptible to particulate suspension because of their high moisture content and their tendency to mat as they dry, making them unlikely to be dispersed by wind. Since particulate suspension would occur only under a limited set of conditions, it is preferable to regulate operating procedures such as requiring placement of daily cover over landfilled sludges. Cover would typically consist of clean soils or a mixture of sludge and soil at least 15 cm (6 in.) deep (U.S. EPA, 1989b).

In summary, good management practices should control any health risks from pathogen transport in runoff and resuspended particulates in the atmosphere. However, similar regulatory controls would not eliminate potential pathogen exposure through the groundwater pathway. Therefore, only exposure to pathogens via the groundwater pathway is included in the SLDGFILL model.

6. MODEL DESCRIPTION AND RESULTS

6.1. OVERVIEW OF THE METHOD

The SLDGFILL model was adapted from the pathogen risk assessment model for land application of sewage sludge (U.S. EPA, 1989c,d). SLDGFILL addresses the disposal of dewatered sludges ($\geq 15\%$ solids) in sludge-only landfills (monofills) that use trench, area fill or diked containment methods (U.S. EPA, 1988a) and long-term storage or disposal of sludge in surface containment lagoons (U.S. 1990a). SLDGFILL is considerably simpler than the parent model because it deals only with the groundwater pathway and only with disposal sites from which pathogens may be leached into groundwater (Chapter 5, "Identification of Exposure Pathways"). The other exposure routes are less significant or better regulated through good sludge management practices (U.S. EPA, 1988a; 1989b). The receptor is an individual ingesting water directly from a drinking-water well near the site. Appendix A (User's Manual) describes how to run the model.

In SLDGFILL, the saturated zone groundwater transport and infection algorithms from the parent model have been modified. A groundwater transport subroutine for unsaturated soil was added. Based on the van Genuchten model (van Genuchten and Alves, 1982), it combines features of the saturated zone transport subroutine from the parent model with U.S. EPA's unsaturated zone transport methodology for chemicals in sludge (U.S. EPA, 1989b).

This sludge pathogen risk assessment model is a compartment-vector model with four compartments: bulk sludge, unsaturated soil, saturated soil and groundwater (drinking-water) well. The model begins with a trench filled with dewatered sludge or a lagoon filled with liquid sludge as the worst case for the source term in each application. The number of organisms in each compartment is calculated for a column with square cross section, 1 m (all units are metric) on each side and the entire depth of the compartment. The number of organisms may increase or decrease by transfer from one compartment to another or may decrease by inactivation or death. Growth equations are not included because viruses and human parasites do not reproduce without a suitable host and the growth of pathogenic enteric bacteria is not favored by conditions in a sludge monofill or surface disposal site. Under certain conditions (e.g., addition of compost

or other nutrient mixtures), bacteria may increase by regrowth; however, such substrates are not considered in this application, which is specific for monofills or sludge lagoons.

Transfers are assumed to be unidirectional, from sludge through soil to the well from which the exposed individual drinks. The numbers of pathogens to be transferred are calculated on a daily basis. Inactivation or die-off is included in each transfer step, as well as retardation and dispersion, which are calculated by the groundwater transport subroutines.

6.2. ASSUMPTIONS

With the exception of *Cryptosporidium* and *Giardia*, it is assumed that helminth ova and protozoan cysts are too large to move through the soil. Table 6-1 lists conservative values for resuspension coefficients (SSPNDB and SSPNDS) to address these pathogenic parasites. Bacteria and viruses are considered to move freely, with limitations described by parameters of the groundwater transport subroutine.

The model is intended to be used for distances from a few meters to several hundred meters and for times from several days to a few years, and a description of short-term events is not necessary. In particular, a daily step is adequate for groundwater, because the model does not need to consider rapid events such as surface runoff (assumed to be prevented by site management).

Two key features of microbial fate-and-transfer models are inactivation and transport in groundwater. It is assumed in this model that inactivation or death of pathogens in sludge, soil and groundwater follows exponential kinetics with a constant inactivation rate. Therefore, inactivation is described by equations of the form

$$N = N_0 * e^{-kt}$$

where

- N = number of surviving pathogens,
- N₀ = initial number of pathogens,
- k = exponential inactivation coefficient,
- t = time.

Table 6-1. Parameters for Pathogen Risk Assessment Methodology: Sludge Landfilling and Surface Disposal

Parameter	Definition	Value ^a	Description of Values
DSATZN P(1)	Depth to saturated zone (m)	Range 0-42.7; Median 21.3 ^b ; Default 3.5	Site-specific.
AQUIFR P(2)	Aquifer thickness (m)	1 10 40	Site-specific. Shallow, confined aquifer. Default value. Deep, extensive aquifer.
PORWTR P(3)	Volumetric moisture content, or pore water (θ , standard groundwater model term)	0.06 0.32 0.30	Site-specific. Clay. Default value; medium sand aquifer. Coarse sand.
ANRAIN P(4)	Annual rainfall (cm)	150	Site-specific.
EVAP P(5)	Fraction of rainfall lost by evaporation and runoff	0.5	Site-specific.
WCSAT P(6)	Saturated water content of subsurface soil (fraction) ^c	Range: 0.332-0.582 0.437 0.464 0.475	Site-specific. Default value; sand. Clay loam. Clay.
USATCND P(7)	Unsaturated conductivity rate (m/sec)	6.4E-7	Site-specific.
GSATCND P(8)	Saturated conductivity rate (m/sec) of subsurface soil ^c	1.7E-7 5.8E-5 6.4E-7	Site-specific. Clay. Default value; sand. Clay loam.

Table 6-1. (continued)

Parameter	Definition	Value ^a	Description of Values
DEPTH P(9)	Depth of sludge (m)	Range 0.9-6.1; Median 3.5 ^b	Application-specific. Default value.
SOLIDS P(10)	Fractional solids of sludge	Range .03-.30 Median .17 ^b 0.05	Application-specific. Default value. Surface disposal in lagoons.
BLKDEN P(11)	Bulk density of sludge (g/cm ³)	1.3	
SMRSLP P(12)	Slope of soil moisture retention curve (dimensionless) ^c	8.52 10.40 11.40	Site-specific. Default value; clay loam. Silt clay. Clay.

Table 6-1. (continued)

Parameter	Definition	Value ^a	Description of Values
PATHDN P(13)	Pathogen density in the sludge (pathogens/kg dry weight)	2×10^2 5×10^3 1.1×10^4 1×10^2 5×10^4 5×10^6 2×10^3 1×10^5 1×10^6	Parasites: Low reported value; lower value expected to be insignificant. Default value. Highest reported value. Bacteria: Lower value expected to be insignificant. Default value. Above highest reported values. Virus: Lower value expected to be insignificant. Default value. Above highest reported values.
INACTB P(14)	Inactivation rate in sludge (\log_{10}/day)	0	Assumes protective effect of bulk sludge; to be changed as data allow.
INACTS P(15)	Inactivation rate in moist soil (\log_{10}/day)	0.0037-0.033 0.016-6.39 0.0017-3.69	Parasites (Default 0.0037). Bacteria (Default 0.016). Viruses (Default 0.0017).

Table 6-1. (continued)

Parameter	Definition	Value*	Description of Values
INACTW P(16)	Inactivation rate in water (\log_{10}/day)	0.0128	Parasites (Default 0.0128).
		0.0228-3.01	Bacteria (Default 0.0228).
		0.0039-2.383	Viruses (Default 0.0075).
SSPNDB P(17)	Sludge/water resuspension coefficient (cm^3/g)	Range 20-2000	Pathogen-specific.
		200	Parasites (conservative).
		2000	Bacteria.
		20	Viruses.
SSPNDS P(18)	Soil/water resuspension coefficient (cm^3/g)	Range 10-1000	Pathogen-specific.
		100	Parasites (conservative).
		1000	Bacteria.
		100	Viruses.
DSTAR P(19)	Diffusivity (cm^2/sec)	1.000E-6	
INFALF P(20)	beta-Poisson alpha	0.17	Parasites.
		0.33	Bacteria.
		15	Viruses.
INFBET P(21)	beta-Poisson beta	1.32	Parasites.
		139.9	Bacteria.
		1000	Viruses.
GRADI P(22)	Hydraulic gradient (unitless)	0.01	Site-specific.

Table 6-1. (continued)

Parameter	Definition	Value ^a	Description of Values
XWELL P(23)	Distance of well from sludge area source (m)	20	Arbitrary lower value.
		50	Default value.
		150	Arbitrary upper value.
^a Values from literature were used where possible to establish ranges and default values. ^b Source: Walsh, 1978. ^c Source: U.S. EPA, 1988c.			

Microbial inactivation rates are known to be sensitive to temperature, and algorithms accounting for annual fluctuations in temperature were incorporated in the models designed to evaluate soil amendment with sludge (U.S. EPA 1989c,d; 1990b; 1991a,b). However, Yates and Ouyang (1992) showed that daily temperature fluctuations in soil were minimal below 20 cm, so effects on virus inactivation rates were not significant. Therefore, because it is assumed that a thick pile of sludge will insulate the soil compartments from rapid surface temperature fluctuations, temperature-dependent inactivation rates were not included in this model.

The exponential die-off or inactivation rate coefficient may change as the number of surviving organisms decreases, or inactivation may become linear with time. In the absence of sufficient data describing inactivation of bacteria and viruses in soil, exponential inactivation at a constant rate is assumed in this model.

Composted or incinerated sludges are not considered in this model. Although liners may be used in lagoons, wide-trench or area fill methods, this model assumes no liners for a more conservative modeling approach. It is assumed that the soil compartments are homogeneous. Unsaturated soil and the aquifer have different properties, but the model's calculations are based on homogeneity throughout each compartment. In actuality, pathogen transport is accelerated through cracks and solution channels and impeded by layers of less permeable material. Groundwater velocity, hydrodynamic dispersion coefficient and retardation coefficient are calculated from parameter values input by the user and represent the average properties of the soil through which the pathogens must pass.

The groundwater transport algorithm used in this model is based on advection and dispersion (U.S. EPA, 1989c,d; van Genuchten and Alves, 1982). Advection is bulk flow through the soil, and dispersion describes the diversion of particles from direct flow lines by the presence of a network of pores and channels. Groundwater transport models for pathogens may also include consideration of filtration/clogging in soil pores or adsorption/desorption reactions between pathogens and soil particles. Few field data are available to validate the various models, so it is difficult to determine which are best. A comparison of the SLDGFILL model's predictions for transport in the unsaturated zone with experimental data obtained in the laboratory is presented in Section 6.5.2. Because water typically percolates more slowly through sludge than through soil, the soil layer beneath the sludge trench should remain unsaturated. If

not, as in the case of a sludge lagoon, the depth of the unsaturated zone must be modeled as zero.

The calculated concentration of pathogens reaching the aquifer is used as the starting concentration of pathogens migrating to the well. This is highly conservative because vertical mixing with uncontaminated water is not included. However, regulations for the protection of groundwater require that water released to Class I aquifers (in current use or of sufficient quality for use as a drinking water source) must meet the requirements for drinking water before it is diluted, and it seems reasonable to base evaluations of pathogen transport on similar restrictions. In addition, it was shown by U.S. EPA (1989e) that mixing was not significant over distances < 150 m when a three-dimensional solution was used to model pathogen transport.

The probability of infection is calculated in this model by a beta-Poisson relationship, in which it is assumed that the dose-response curve is adequately represented by the empirically derived equation

$$P^* = 1 - (1 + n/\beta)^{-\alpha}$$

where P^* is the probability of infection and α and β are empirically derived parameters (see Section 6.4.5). The effect of each day's exposure is independent of the outcome for any other day. This assumption ignores additive effects of exposure or the induction of immunity by subinfective exposures. However, it is conservative, because low-level exposure typically reduces susceptibility to infection. U.S. EPA (1992) has suggested that groundwater protection should be based on that assumption and has proposed to use the beta-Poisson model for groundwater protection.

On the basis of model calculations, a limit of 2×10^7 viruses/L has been proposed as a target limit in groundwater (Regli et al., 1991). Results in Section 7.2 are interpreted in light of this proposed limit.

6.3 INPUT PARAMETER REQUIREMENTS

Parameters are identified by name and by parameter number [parameter 1, DSATZN, is denoted P(1)]. Parameters describe physical characteristics of the site, nature and amount of sludge or properties of the pathogens. Default values and descriptions of the parameters are shown in Table 6-1. The ranges of these values are taken from literature discussed in Chapter 4

and from U.S. EPA (1988b, 1990b, 1991a,b). In some cases, intermediate values have been added to provide additional detail.

Before computations begin, the parameters are converted to common units of days, meters and kilograms, as shown in Table 6-2.

6.3.1. Pathway Data. These parameters describe bulk sludge in a full trench or lagoon. A full trench or lagoon as a starting point simplifies calculations and provides a maximum source term of sludge pathogen number in the disposal site. The loading parameters are:

DEPTH	= P(9)	Depth of sludge (m)
SOLIDS	= P(10)	Fractional solids of sludge
BLKDEN	= P(11)	Bulk density of sludge (g/cm ³)
PATHDN	= P(13)	Density of pathogens in sludge (number/kg dry wt)

Site-specific parameters: Site-specific parameters describe physical properties of the site's underlying soil and the relevant weather conditions. They are:

DSATZN	= P(1)	Depth to saturated zone under sludge (m)
AQUIFR	= P(2)	Thickness of aquifer (m)
PORWTR	= P(3)	Fractional water content of aquifer
ANRAIN	= P(4)	Annual rainfall (cm)
EVAP	= P(5)	Fraction of rainfall lost by evaporation and surface runoff
WCSAT	= P(6)	Saturated water content of subsurface soil (fraction)
USATCND	= P(7)	Saturated conductivity rate of vadose-zone soil (m/s)
GSATCND	= P(8)	Saturated conductivity rate of aquifer soil (m/s)
SMRSLP	= P(12)	Slope of the soil moisture retention curve (unitless).

Groundwater transport parameters: Groundwater transport parameters are used by the transport subroutine and describe the soil and groundwater through which the pathogens pass to the well. They are:

DSTAR	= P(19)	Diffusivity (cm ² /sec)
GRADI	= P(22)	Hydraulic gradient (unitless)
XWELL	= P(23)	Distance from the sludge area source to the groundwater well (m)

Table 6-2. Parameter Conversion Factors*

Parameter Name	Final Number	Final Units	Conversion Factors
DSATZN	P(1)	m	
AQUIFR	P(2)	m	
PORWTR	P(3)	fraction	
ANRAIN	P(4)/(365*100)	m/day	(365 days/yr)*(100 cm/m)
EVAP	P(5)	fraction	
WCSAT	P(6)	fraction	
USATCND	P(7)*86400	m/day	86400 sec/day
GSATCND	P(8)*86400	m/day	86400 sec/day
DEPTH	P(9)	m	
SOLIDS	P(10)	fraction	
BLKDEN	P(11)*1000	kg/m ³	(10 ⁶ cm ³ /m ³)/(1000 g/kg)
SMRSLP	P(12)	number	
PATHDN	P(13)	number/kg	
INACTB	P(14)	log ₁₀ /day	
INACTS	P(15)	log ₁₀ /day	
INACTW	P(16)	log ₁₀ /day	
SSPNDB	P(17)/1000	kg/m ³	(1000 g/kg)/(10 ⁶ cm ³ /m ³)
SSPNDS	P(18)/1000	kg/m ³	(1000 g/kg)/(10 ⁶ cm ³ /m ³)
DSTAR	P(19)	cm ² /sec	
INFALF	P(20)	unitless	
INFBET	P(21)	unitless	
GRADI	P(22)	unitless	
XWELL	P(23)	m	

*P(x) is the value entered by the model user. Mathematical conversion is performed within the model, as indicated in the second column, to produce the Final Value in the Final Units shown.

6.3.2. Pathogen-Specific Data. Organism-specific properties characterize survival of pathogens, their interaction with sludge and soil particles and their infective dose. They are:

INACTB	= P(14)	Inactivation rate in bulk sludge (\log_{10}/day)
INACTS	= P(15)	Inactivation rate in soil (\log_{10}/day)
INACTW	= P(16)	Inactivation rate in water (\log_{10}/day)
SSPNDB	= P(17)	Sludge-to-water resuspension factor [(number/g sludge)/(number/cm ³ water)=cm ³ /g]
SSPNDS	= P(18)	Soil-to-water resuspension factor [(number/g sludge)/(number/cm ³ water)=cm ³ /g]
INFALF	= P(20)	beta-Poisson alpha (required to calculate risk of infection)
INFBET	= P(21)	beta-Poisson beta (required to calculate risk of infection)

6.3.3. Processes and Transfers. Transfers and inactivation are calculated at daily intervals in a do-loop structure. The concentration of organisms in water suspension in each compartment is calculated using the assumed solids/water distribution coefficients. Subsurface transport subroutines are called daily from the unsaturated soil and saturated soil compartments, and the number of pathogens in each compartment is calculated from the transfers in and out and the inactivation rate appropriate for each compartment. It is assumed that inactivation of pathogens occurs at a constant exponential rate in each compartment. The only human exposure considered in the model is consumption of drinking water from an offsite well.

6.4. CALCULATIONS

6.4.1. Source Term. Bulk Sludge in Trench or Lagoon--A full trench or lagoon as a starting point simplifies calculations while providing a maximum source term of sludge pathogen number in the disposal unit. Sludge-specific loading parameters describe only the sludge. It is known that the survival of sludge pathogens is enhanced by moisture, organic matter and moderate pH values (Kowal, 1985; U.S. EPA, 1988a). In this study it was assumed that the moisture and nutrient content of sludge protect pathogens from inactivation; this assumption is probably overly conservative, but it provides a reasonable worst-case assessment of potential risks from pathogen transport. Depth of monofill application should allow the protective effects of bulk sludge to remain constant; the variable INACTB [P(13)] assumes the default exponential

die-off rate of $0 \log_{10}/\text{day}$, but can be changed when data on die-off rates in bulk sludge become available.

The initial number of sludge pathogens/ m^2 of sludge is calculated from the concentration of pathogens (number/kg solids), the fraction of sludge solids, the bulk density (kg/m^3) and the depth of sludge (m):

$$N(1) = \text{PATHDN} * \text{SOLIDS} * \text{BLKDEN} * \text{DEPTH} \quad (\text{number}/\text{m}^2).$$

SLUDGE is the mass of sludge solids. The mass of sludge particles is calculated from the fraction of sludge solids, the bulk density and the depth of sludge:

$$\text{SLUDGE} = \text{SOLIDS} * \text{BLKDEN} * \text{DEPTH} \quad (\text{kg}/\text{m}^2).$$

WATER is the depth of water (m) in the sludge. The water content is expressed as meters of water/ m^2 of sludge, using the bulk density, solids content and depth of the sludge and the density of water ($1000 \text{ kg}/\text{m}^3$):

$$\text{WATER} = (1 - \text{SOLIDS}) * \text{BLKDEN} * \text{DEPTH} / 1000 \quad (\text{m}).$$

Not all of the soil water is free to migrate. Some of it is associated with particles or colloids or trapped by capillarity in soil pores. In this discussion, the water that is free to move through the soil will be termed free water. Particulate-associated pathogens can be leached into free water. Although the actual distribution between water and particles is likely to depend on chemical composition of the sludge or soil and of the water solution, it is assumed that the distribution is constant through the compartment and that suspended and particulate-bound pathogens are in equilibrium (i.e., the ratio of pathogens adsorbed to solids to pathogens suspended in free water in the same volume of soil remains constant). The ratio of particulate-bound to suspended organisms is given by the parameter SSPNDB for bulk sludge.

SSPNDB [P(16)] is the sludge/water resuspension coefficient for a particular sludge and pathogen. The concentration of pathogens suspended in free water in the sludge column is calculated by using the distribution of pathogens between water and solids (SSPNDB for sludge, SSPNDS for soil). The equation is derived as follows:

$$\begin{aligned} \text{Total Number } N(1) &= N_{\text{water}} + N_{\text{solid}} \\ N_{\text{water}} &= \text{Conc}_{\text{water}} * \text{Amt. of water [in m because } N(1) \text{ is calculated} \\ &\quad \text{per } \text{m}^2 \text{ of cross section]} \\ &= C(1) * \text{WATER} \end{aligned}$$

$$\begin{aligned}
 N_{\text{solid}} &= \text{Conc}_{\text{solid}} * \text{Amt. of sludge} \\
 &= \text{Conc}_{\text{sludge}} * \text{SLUDGE}.
 \end{aligned}$$

For any distribution coefficient SSPND_x ,

$$\begin{aligned}
 \text{SSPND}_x &= (\text{Pathogens/kg solid}_x) / (\text{Pathogens/m}^3 \text{ water}) \\
 &= \text{Conc}_{\text{solid}} / \text{Conc}_{\text{water}}.
 \end{aligned}$$

Therefore,

$$\begin{aligned}
 \text{Conc}_{\text{solid}} &= \text{Conc}_{\text{water}} * \text{SSPND}_x \\
 \text{Conc}_{\text{sludge}} &= C(1) * \text{SSPNDB}
 \end{aligned}$$

and

$$N_{\text{sludge}} = C(1) * \text{SSPNDB} * \text{SLUDGE}.$$

Combining equations,

$$\begin{aligned}
 N(1) &= (C(1) * \text{WATER}) + (C(1) * \text{SSPNDB} * \text{SLUDGE}) \\
 &= (C(1) * \text{WATER}) + (C(1) * \text{SLUDGE} * \text{SSPNDB}) \\
 &= C(1) * (\text{WATER} + \text{SLUDGE} * \text{SSPNDB})
 \end{aligned}$$

and

$$C(1) = N(1) / (\text{WATER} + \text{SLUDGE} * \text{SSPNDB}) \quad (\text{pathogens/m}^3).$$

Transfer from compartment 1 to compartment 2 (TR12) is assumed to occur by leaching of water percolating through the sludge layer. The amount of rain (m/yr) passing through and leaching pathogens from the sludge is calculated by using the annual amount of rainfall and the fraction that evaporates or runs off:

$$\text{WFLUX} = \text{ANRAIN} * (1 - \text{EVAP}) \quad (\text{m/day}).$$

It is assumed that the sludge remains saturated because percolation is slow and the soil cap placed on top of each day's application retards evaporation. Based on this assumption, leaching is characterized by a constant rate rather than pulses associated with rainfall. Assuming a constant leaching rate may overestimate the length of time for which concentrations of pathogens are elevated and may underestimate what would be peak concentrations of pathogens in individual pulses (U.S. EPA, 1989d).

The concentration $[C(1)]$ of pathogens in pore water in compartment 1 (sludge) is used to calculate the transfer TR12:

$$C(1) = N(1) / (\text{WATER} + \text{SLUDGE} * \text{SSPNDB})$$

$$TR12 = C(1)*WFLUX$$

where N(1) is the number of pathogens in compartment 1 (pathogens/m²). To determine the number of pathogens transferred to compartment 2, the concentration [C(1)] is multiplied by the volume of water/day percolating through a 1-m² column of sludge at the average recharge rate. The number of pathogens remaining in compartment 1 is the current number times the die-off rate minus the flux to the unsaturated zone:

$$N(1) = N(1)*(10^{**(-INACTB))}-TR12.$$

6.4.2. Unsaturated Zone Transport. Unsaturated transport, modeled according to methods described for chemicals in sludge (U.S. EPA, 1989b), uses the solution of a one-dimensional advective-dispersive transport model (van Genuchten and Alves, 1982). This is the same method used to model unsaturated zone transport in MULTIMED (Multimedia Exposure Assessment Model for Evaluating the Land Disposal of Wastes) developed by the U.S. EPA (Salhotra et al., 1990). This is also the same model used for transport in the saturated zone. Each daily loading of pathogens is treated as an independent pulse through the unsaturated zone. The resulting concentration at the saturated zone boundary is calculated for every day in the model run, and all values for the same day of the model run are added together. Thus, the concentration reaching the saturated zone [C(2)] is the sum of all of the portions of each day's input that arrives at the saturated zone on that day.

6.4.2.1. Initial Populations and Transfers--The initial concentration of pathogens in unsaturated soil may be specified as POPL(2) during the parameter input phase of the model run. The initial number of pathogens/m² in this compartment is calculated from the initial concentration (number/kg), the bulk density of soil (kg/m³) and the depth of unsaturated soil (m):

$$N(2)=POPL(2)*USDEN*DSATZN \quad (\text{number/m}^2).$$

Unless compartment populations are entered at the beginning of the model run, it is assumed that the initial pathogen number in the soil compartment is zero.

The number of organisms entering the compartment daily from sludge is given by TR12, the product of pathogen concentration in the sludge water phase and the flux of water through the sludge. This number is decreased by TR23, the product of water flux and concentration of pathogens in water in the unsaturated zone:

$$TR23 = C(2)*WFLUX.$$

The latter is calculated by SUBROUTINE UNSATZN, which uses the concentration of pathogens in the sludge water phase as the concentration entering unsaturated soil. Die-off in this compartment is assumed to be exponential, described by INACTS [P(17)] (logs/day) for soil-bound organisms and INACTW [P(15)] (logs/day) for suspended organisms.

The bulk density of soil in the unsaturated (USDEN) zone is used to calculate the distribution of pathogens between solids and water. This calculation assumes a particle density of 2650 kg/m³ (U.S. EPA, 1989b):

$$\text{USDEN} = (1 - \text{WCSAT}) * 2650 \text{ (kg/m}^3\text{)}.$$

Particulate-associated pathogens are leached into free water. Although the actual distribution between water and particles is likely to depend on chemical composition of the sludge or soil and of the water solution, it is assumed that the distribution is constant through the compartment and that suspended and particulate-bound pathogens are in equilibrium. The ratio of particulate-bound to suspended organisms is given by the parameter SSPNDS for soil.

6.4.2.2. Derivation of Particle/Water Distribution Equations--The concentration of pathogens suspended in free water in the soil column is calculated by using the distribution of pathogens between water and solids (SSPNDS for soil). The equation is derived as follows:

$$\text{Total Number } N(1) = N_{\text{water}} + N_{\text{solid}}.$$

For the unsaturated zone, the amount of water is the product of the depth to the saturated zone (DSATZN) and the water content of soil in the unsaturated zone (FUNSAT, defined below). The amount of soil is the depth of unsaturated soil (DSATZN) times its bulk density (USDEN):

$$N_{\text{water}} = C(2) * \text{DSATZN} * \text{FUNSAT}$$

$$N_{\text{unsat. soil}} = \text{Conc}_{\text{unsat. soil}} * \text{USDEN} * \text{DSATZN}.$$

For any distribution coefficient SSPND_x,

$$\begin{aligned} \text{SSPND}_x &= (\text{Pathogens/kg solid}_x) / (\text{Pathogens/m}^3 \text{ water}) \\ &= \text{Conc}_{\text{solid}} / \text{Conc}_{\text{water}}. \end{aligned}$$

Therefore,

$$\text{Conc}_{\text{solid}} = \text{Conc}_{\text{water}} * \text{SSPND}_x$$

$$\text{Conc}_{\text{unsat. soil}} = C(2) * \text{SSPNDS}$$

and

$$N_{\text{unsat. soil}} = C(2) * \text{SSPNDS} * \text{USDEN} * \text{DSATZN}.$$

Combining equations,

$$\begin{aligned} N(2) &= (C(2)*DSATZN*FUNSAT) + (C(2)*SSPNDS*USDEN*DSATZN) \\ &= (C(2)*DSATZN)*FUNSAT + (C(2)*DSATZN)*(USDEN*SSPNDS) \\ &= (C(2)*DSATZN)*(FUNSAT + USDEN*SSPNDS) \\ &= C(2)*(DSATZN*(USDEN*SSPNDS + FUNSAT)) \end{aligned}$$

and

$$C(2) = N(2)/(DSATZN*(USDEN*SSPNDS + FUNSAT)) \quad (\text{number/m}^3).$$

6.4.2.3. Pore Water Velocity--Moisture content in the unsaturated zone is used to calculate the pore water velocity in the unsaturated zone (U.S. EPA, 1989b). The fractional water content, FUNSAT, is the product of the saturated water content of the soil and a nonlinear function of the ratio of the flux through unsaturated soil to the flux through saturated soil. The latter term is approximated by using the slope of the soil retention curve, which is related to soil type (U.S. EPA, 1989b). For typical values of water content the slope may range from 7 to 11, but this parameter appears in an exponent with other terms. The corresponding range of exponents is 0.04 - 0.059:

$$FUNSAT = (WCSAT*(WFLUX/USATCND))^{(1/(2*SMRSLP+3))}.$$

Pore water velocity is used by the subsurface transport subroutine to calculate transport in the unsaturated zone. Pore water velocity in the unsaturated zone is calculated from the site-specific annual recharge rate and the moisture capacity and the matric potential (a measure of the pressure required to remove water from the soil) of the unsaturated soil (U.S. EPA 1989b). It is calculated by dividing the flux through the unsaturated zone (WFLUX) by the fractional moisture content (FUNSAT):

$$VUNSAT = WFLUX/FUNSAT \quad (\text{m/day}).$$

6.4.2.4. Retardation and Dispersion Coefficients--The retardation coefficient for unsaturated soil (RUS) is calculated from the soil-water suspension coefficient (SSPNDS), the soil bulk density (USDEN) and the moisture content in the unsaturated zone (FUNSAT). SSPNDS is input as P(18). USDEN is calculated as described in Section 6.4.2.1, and FUNSAT is calculated as described in Section 6.4.2.3. These parameters are then used to calculate the retardation coefficient:

$$RUS = 1.0 + ((SSPNDS*USDEN)/FUNSAT).$$

In this context, SSPNDS is analogous to K_D , the soil-water partition coefficient for solutes being transported in groundwater. RUS is passed to the groundwater transport subroutine for unsaturated soil.

The hydrodynamic dispersion coefficient for unsaturated soil (DUNSAT) is calculated from the flux of water through the unsaturated zone, which is equal to the annual rainfall [ANRAIN, P(4)] minus the fraction that evaporates [EVAP, P(5)]:

$$WFLUX = ANRAIN * (1 - EVAP)$$

This value is used in an equation that has been empirically derived to approximate the hydrodynamic dispersion of solutes in groundwater:

$$DUNSAT = (0.6 + 2.93 * (WFLUX * 100) ** 1.11) / 10,000$$

DUNSAT is also passed to the groundwater transport subroutine for unsaturated soil.

6.4.2.5. Daily Number of Pathogens--The number of pathogens in compartment 2 [N(2)] is calculated as:

$$N(2) = N(2) + TR12 - TR23 - (N(2) - CX(2) * DSATZN * FUNSAT) * (1 - 10 ** (-INACTS))$$

where INACTS [P(14)] is the pathogen inactivation rate on the soil particles (logs/day) and CX(2) is the average concentration of suspended pathogens in the unsaturated zone.

6.4.3. Saturated Zone Transport. Transport is modeled by the solution of a one-dimensional advective-dispersive transport model (van Genuchten and Alves, 1982) as in the unsaturated zone. The concentration of pathogens from the unsaturated zone, C(2), is used along with the transport parameters and inactivation rate, INACTS [P(14)], to determine the concentration in the groundwater at the well, C(3).

6.4.3.1 Initial Populations and Transfers--The initial concentration of pathogens in saturated soil may be specified as POPL3 during the parameter input phase of the model run. The initial number and concentration of pathogens/m² are analogous to the number in the unsaturated zone:

$$N(3) = POPL(3) * GW DEN * AQUIFR \quad (\text{number/m}^2).$$

Die-off rate is exponential, described by the inactivation rate INACTW [P(15)] (logs/day). The concentration is calculated from the number of pathogens present in the 1-m² column whose volume is (1 m²) * AQUIFR * PORWTR m³; each day the number is incremented by TR23 and adjusted for die-off. Concentration is calculated using the suspension factor and calculated water

volume. The resulting concentration is then passed to SUBROUTINE GRDWTR to calculate transfer to the offsite well.

The bulk density of soil in the saturated (GWDEN) zone is used to calculate the distribution of pathogens between solids and water. This calculation assumes a particle density of 2650 kg/m³ (Brady, 1984):

$$GWDEN = (1-PORWTR)*2650 \text{ (kg/m}^3\text{)}.$$

Particulate-associated pathogens are leached into free water. Studies have shown that the fraction of microorganisms suspended in water is dependent on properties of the soil as well as on the type of microorganism (Burge and Enkiri, 1978; Drewey and Eliassen, 1968; Gerba et al., 1975; Marshall, 1971; Reddy et al., 1981). Although the actual distribution between water and particles is likely to depend on chemical composition of the sludge or soil and of the water solution, we assume that the distribution is constant through the compartment and that suspended and particulate-bound pathogens are in equilibrium or steady-state. The ratio of particulate-bound to suspended organisms is given by the parameter SSPNDS for soil.

6.4.3.2 Particle/Water Distribution--The concentration of pathogens suspended in free water in the soil column is calculated by using the distribution of pathogens between water and solids (SSPNDS for soil). For the saturated zone,

$$\begin{aligned} N_{\text{water}} &= C(3)*AQUIFR*PORWTR \\ N_{\text{sat. soil}} &= \text{Conc}_{\text{sat. soil}}*AQUIFR*GWDEN \\ N(3) &= (C(3)*AQUIFR*PORWTR) + (C(3)*SSPNDS*AQUIFR*GWDEN) \\ &= C(3)*AQUIFR*(PORWTR + GWDEN*SSPNDS) \end{aligned}$$

and

$$C(3) = N(3)/(AQUIFR*(GWDEN*SSPNDS + PORWTR)) \text{ (number/m}^3\text{)}.$$

6.4.3.3 Pore Water Velocity--The velocity of pore water in the saturated zone (VGW) depends on the hydraulic gradient [GRADI, P(22)], the saturated conductivity rate [GSATCND, P(8)] and the fractional water content of the aquifer [PORWTR, P(3)]:

$$VGW = (GSATCND*GRADI)/PORWTR$$

VGW is calculated after data input and passed to the groundwater transport subroutine for saturated soil.

6.4.3.4 Retardation and Dispersion Coefficients--The retardation coefficient for saturated soil (RGW) is calculated from the soil-water suspension coefficient (SSPNDS), the soil bulk density (GWDEN) and the fractional moisture content in the aquifer (PORWTR). SSPNDS is input as P(18). Like USDEN, GWDEN is calculated by assuming a soil particle density of 2650 kg/cubic meter, reduced by the fraction of saturated soil that is water [PORWTR, P(3)]:

$$GWDEN = (1 - PORWTR) * 2650$$

The moisture content for the aquifer is not reduced from PORWTR because the aquifer is assumed to be saturated with water. These parameters are then used to calculate the retardation coefficient:

$$RGW = 1.0 + ((SSPNDS * GWDEN) / PORWTR)$$

RGW is passed to the groundwater transport subroutine for unsaturated soil.

The hydrodynamic dispersion coefficient for saturated soil (DGW) is calculated by a standard empirically fitted equation for groundwater transport:

$$DGW = (VGW * 0.1 * XWELL) + DSTAR,$$

where VGW is calculated as described in Section 6.4.3.3, XWELL (distance from the sludge source to the groundwater well) is input as P(23) and DSTAR (the dispersivity of pathogens in water) is input as P(19). For solutes, dispersivity decreases with increasing molecular weight; because pathogens are very large relative to solute molecules, DSTAR is assumed to be very small (1×10^{-6}). Along with VGW and RGW, DGW is passed to the groundwater transport subroutine for the saturated zone.

6.4.4. Offsite Well. If the concentration of pathogens in the groundwater is greater than zero, SUBROUTINE GRDWTR is called. The number of pathogens/m² in the well compartment is equal to the concentration in groundwater multiplied by the thickness of the aquifer. The concentration of pathogens in the well is assumed to be the same as their concentration in groundwater at distance XWELL from the source:

$$N(4) = C(3) * AQUIFR \quad (\text{number/m}^2)$$

$$C(4) = C(3) \quad (\text{number/m}^3).$$

The output from SUBROUTINE GRDWTR is passed to SUBROUTINE RISK, which calculates the probability of ingesting more than an infective dose of pathogen in 2 L of well water.

6.4.5 Risk of Infection. For infection to occur, a susceptible host must be exposed, by a suitable exposure route, to a sufficient number of pathogens that are virulent enough to cause infection. This model assumes that individuals ingesting contaminated drinking water from a groundwater well are susceptible to the pathogens in the water. The quantity of water ingested daily is not assumed to vary among exposed individuals. The number of pathogens ingested is calculated from the modeled concentration in the well water. This number is used in the dose-response model to calculate the probability of infection.

A number of models have been proposed to describe the dose responses of infectivity of pathogens. Most are based on mathematical theories of infection, with parameters that are empirically fitted to clinical data. This SLDGFILL model uses the beta-Poisson model (Haas, 1983), which assumes that both individual susceptibility to pathogens and the infectivity of the pathogens themselves vary within the populations being modeled. The probability of infection is the probability of receiving more than an infective dose, or 1 minus the sum of the probabilities of receiving all multiples less than the infective dose. In the beta-Poisson model, the effectiveness of the pathogen-host interaction is described by a distribution rather than a single value (Haas, 1983). The resulting equation is:

$$P^* = 1 - (1 + (N/\beta))^{-\alpha}$$

which appears in the SLDGFILL code as

$$XPROB = 1 - (1 + (SUMX/INFBET))^{*(-INFALF)},$$

where

- XPROB = the daily probability of infection,
- SUMX = the calculated number of pathogens ingested daily,
- INFBET = empirically derived value of β for the pathogen in question
- INFALF = empirically derived value of α for the pathogen in question.

Currently available values of β and α are presented in Table 6-3. Values for additional pathogens are being developed and are expected to be available within several months (Haas, personal communication, 1994), which will extend the flexibility of the SLDGFILL model. For the model determinations presented in this document, values of α and β for *Salmonella* were used. Other bacterial pathogens (e.g., *Shigellae*) are generally much more infective, but they

Table 6-3. Values of Alpha and Beta for Selected Pathogens

Pathogen	Alpha	Beta	Reference
<i>Salmonella</i> spp.	0.33	139.9	Rose (1993)
<i>Shigella dysenteriae</i>	0.5	100	Haas (1983)
<i>Shigella flexneri</i> 2A##	0.2	2000	Haas (1983)
<i>Entamoeba coli</i>	0.17	1.32	Haas (1983)
Echovirus 12	1.3	75	Haas (1983)
Echovirus 12	0.374	186.7	Regli et al. (1991)
Poliovirus 1	15	1000	Haas (1983)
Poliovirus 1	0.119	200	Haas (1983)
Poliovirus 1	0.11	1524	Regli et al. (1991)
Poliovirus 3	0.5	1.14	Haas (1983)
Poliovirus 3	0.409	0.788	Regli et al. (1991)
Rotavirus	0.26	0.42	Regli et al. (1991)

are found in sludge less frequently and at lower concentrations than Salmonellae. Whenever possible, pathogen-specific values should be used to estimate risk for other pathogens of interest.

6.5. RESULTS

The results of several model simulations are presented in the following sections. The default values for bacteria and viruses shown in Table 6-1 were used as input in the initial model runs (Section 6.5.1). Section 6.5.2 compares the predictions of the SLDGFILL model with results obtained in a laboratory soil column study and with the predictions of another unsaturated-zone transport model for pathogens. Section 6.5.3 presents the results of modeling transport of bacteria and viruses when the parameter values were chosen to characterize six geographically and climatologically different sites described in the pathogen risk assessments for land application of sludge (U.S. EPA, 1990b; 1991a,b). The results show that pathogen concentrations are attenuated with each step from the source so that no pathogens reach saturated soil 3.5 m below the sludge within 2 years; that under default conditions, pathogen concentrations in soil water reach an early maximum followed by a slight steady decrease; that viruses are transported at significantly higher concentrations than bacteria and parasites; and that site-specific differences in rainfall and soil parameters have relatively little effect on pathogen transport.

6.5.1. Kinetics of Pathogen Transport. Figure 6-1 shows the kinetics of transport of bacteria, viruses and parasites in soil water from sludge into unsaturated soil. Under default conditions, pathogens do not reach unsaturated soil in 2 years.

Pathogens are rapidly transported into the unsaturated soil, but under default conditions they do not reach the saturated soil in 2 years. Because transport rates are proportional to SSPNDS [P(18)] (see Chapter 7), it is possible to extrapolate the time required to traverse the unsaturated and saturated zones. To calculate the time required for viruses to reach the aquifer, the default values of PATHDN [P(13)] and SSPNDS [P(18)] (1×10^5 and 100, respectively) were used. SSPNDB [P(17)] was set at 0. Viruses were assumed to have reached the aquifer when their concentration became $2 \times 10^{-7}/L$, the proposed limit for groundwater (U.S. EPA, 1992). The extrapolated time required to reach an aquifer 3.5 m below the sludge layer was ~ 420 years. To calculate the time required to reach the well after entry into the aquifer, DSATZN

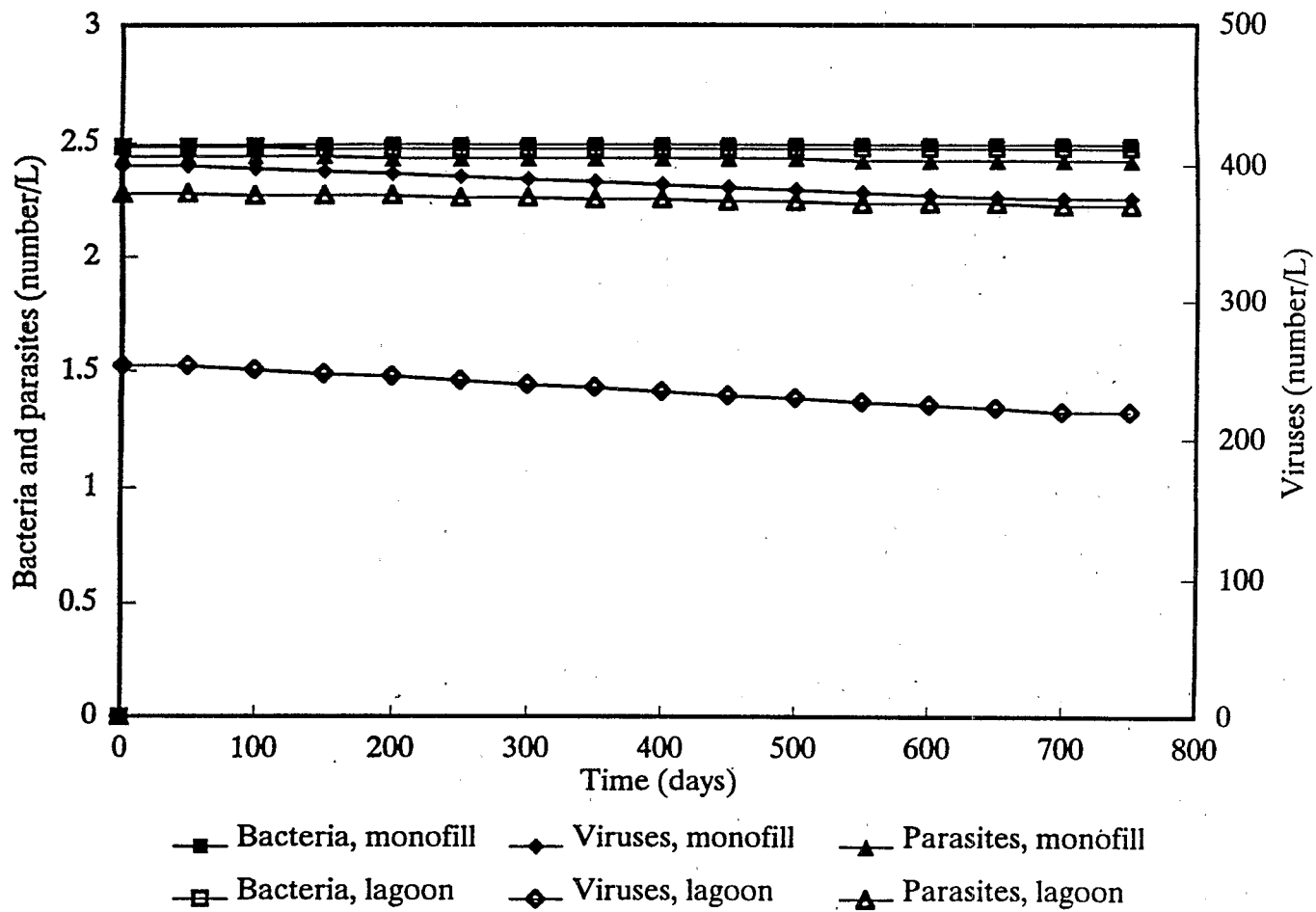


Figure 6-1. Pathogen Concentration in Unsaturated Soil

[P(1)] was set to 0, and XWELL [P(23)] was varied (see Section 7.2.2.5). The extrapolated time required for viruses to be transported 50 m in groundwater was ~128 years. Because SSPNDS is higher for bacteria than for viruses, the bacteria would be expected to appear in the aquifer and the well much later (>4000 years for bacteria and >1280 years for viruses). These times are much greater than the survival times in soil of any known pathogens.

Figure 6-1 shows that the concentrations of viruses in the unsaturated zone was ~200 times (from a monofill) or ~100 times (from a disposal lagoon) the maximum concentration of bacteria and parasites. These differences are the result of differences in particle/water resuspension coefficients and inactivation rates in the sludge and unsaturated zones. At all times, the calculated concentration of all pathogens in water from the groundwater well was $1 \times 10^{-10}/L$. Conditions required to show transport to the well are described in Chapter 7.

6.5.2. Comparison with Experimental Results. A report comparing the outcome of the VIRTUS model for unsaturated-zone transport to experimental data was published by Yates and Ouyang (1992). The predictions of the SLDGFILL model were compared with one of the data sets used to check the VIRTUS model. A large sludge layer was specified in these test runs so the input of viruses to the unsaturated zone would be constant as it was in the laboratory study (Powelson et al., 1990). The thickness of the unsaturated zone was varied to match the depths at which virus concentrations were measured in the study. As nearly as possible, groundwater transport parameters and soil properties were as specified by the authors of the study. Figure 6-2 shows that SLDGFILL predicts more rapid transport into the upper part of the column than the VIRTUS model and laboratory results indicated, whereas the SLDGFILL model predicted much less transport beyond 40 cm than the VIRTUS model and the laboratory results showed. However, the distance over which transport was measured in the laboratory study was very small compared to the distances for which the SLDGFILL model was designed, and the transport time was only 4 days. Field data over larger distances and longer times are needed to allow a better choice among transport models.

6.5.3. Site-specific Parameter Testing. In previous modeling studies of soil amendment with sewage sludge (U.S. EPA, 1990b; 1991a,b), six hypothetical sites have been described. Properties of the soil and climate at these sites were used to choose values for model

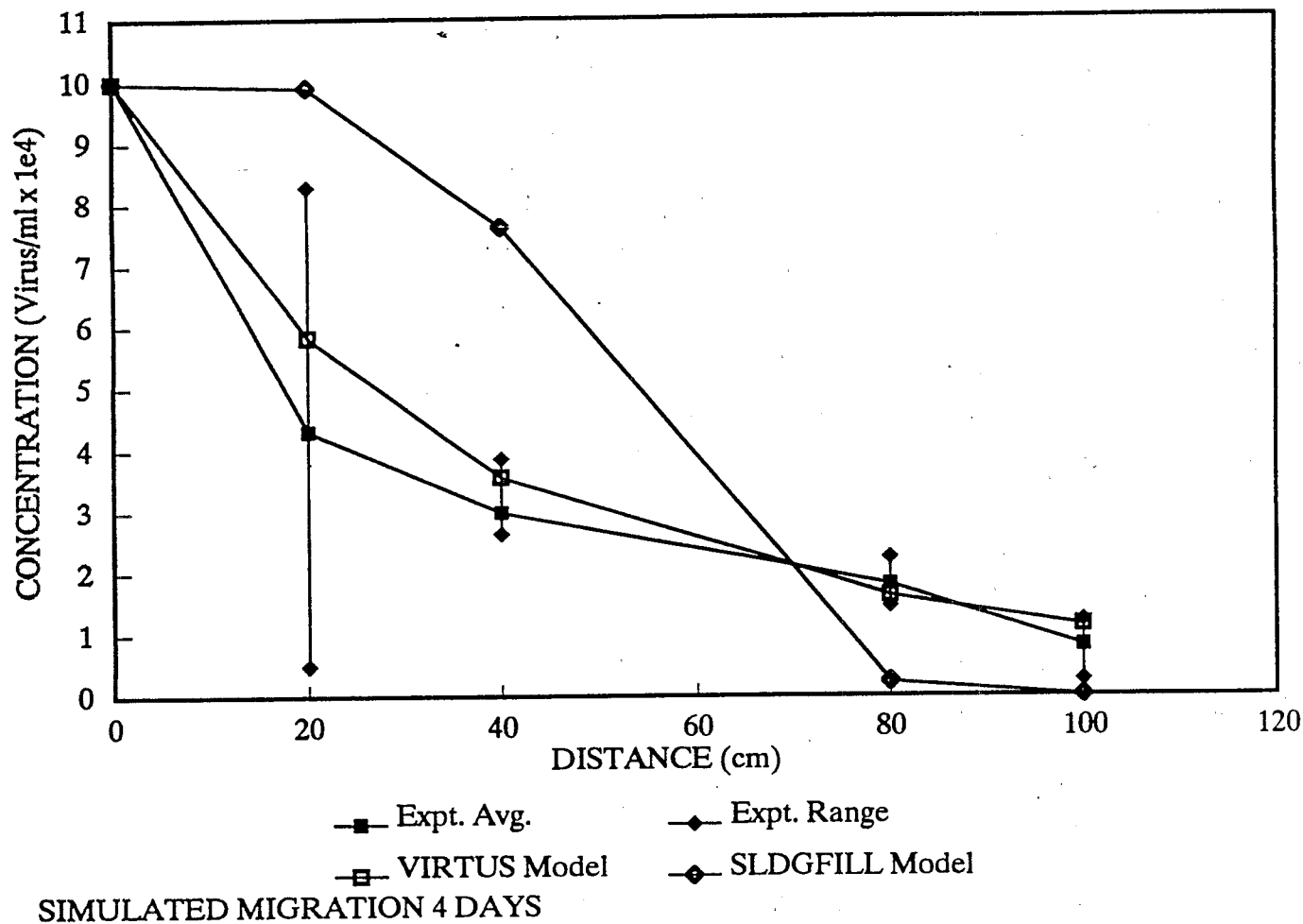


Figure 6-2. Comparison of Model Results with VIRTUS Model and Experimental Data

parameters, and the outcome was then determined. Variability in the results gave some indication of the importance of climate and soil type on applicability of the soil amendment technology. Selected values for the same sites were used to test the sensitivity of the SLDGFILL model to site-specific parameters. The parameters chosen were ANRAIN [P(4)], EVAP [P(5)], WCSAT [P(6)] and USATCND [P(7)]. Parameter values are listed in Table 6-4. Default values were used for groundwater transport parameters for the sites.

Annual rainfall amounts were calculated from average rainfall data given in soil surveys (USDA 1980; 1981a,b,c; 1985; 1989). In several cases, values were also included above and below which rainfall could be expected during 20% of the years. These values were taken to represent the 80% upper and lower confidence intervals in a normal distribution. From them, the assumed standard deviation was calculated, and the 95% upper and lower confidence intervals were calculated. When these values were not available, the same ratio of upper value to the mean was used. EVAP, the fraction of rainfall that evaporates or runs off, was the default value of 0.5 except in the case of Chaves Co., NM, which was described as being very dry (assigned a value of 0.7), and Highlands Co., FL (assigned a value of 0.6). WCSAT was calculated from soil bulk density, as described in Section 6.4.2.1. Soil bulk density was usually given in the soil surveys. If not, a value of 0.45 for WCSAT was used. USATCND was listed in tables of physical properties of the soils in the soil surveys. Artificial parameter values were used to allow a comparison of results: SSPNDB was set at 200, SSPNDS = 100, DSATZN = 0.1 and XWELL = 10. Model runs were done for 800 days.

Model runs for bacteria and viruses are compared in Table 6-5. The results show that site-specific parameters had little effect on the concentration in the unsaturated zone. The predicted virus concentrations in the unsaturated soil after 800 days ranged from 300/L to 980/L. However, the predicted maximum concentration of viruses in the saturated soil ranged from $1.3 \times 10^{-7}/L$ to $3.0 \times 10^{-2}/L$. These results show that site-specific parameters, most likely variation in saturated conductivity rate in the unsaturated soil, had a significant effect on transport into the aquifer. Even with these unrealistically low parameter values, pathogens did not reach the groundwater well in any model run.

Effects on soil-water suspension factors and inactivation rates are expected to be more important in determining site-specific impacts. However, values for these parameters were not

Table 6-4. Parameters for Site-Specific Model Evaluation

Site		ANRAIN P(4) (cm)	EVAP P(5) (fraction)	WCSAT P(6) (fraction)	USATCND P(7) (m/s)
No.	Location				
1	Anderson Co., TN	187	0.5	0.45	4.24×10^{-6}
2	Chaves Co., NM	42.7	0.7	0.46	4.24×10^{-7}
3	Clinton Co., IA	130	0.5	0.46	4.24×10^{-6}
4	Highlands Co., FL	200	0.6	0.42	2.5×10^{-2}
5	Kern Co., CA	37.9	0.5	0.46	2.82×10^{-5}
6	Yakima Co., WA	29.5	0.5	0.46	2.82×10^{-6}

Table 6-5. Results of Site-Specific Model Evaluation

Site		Organism	Concentration (Pathogens/L) at 800 days	
No.	Location		Saturated zone	Unsaturated zone
1	Anderson Co., TN	Bacteria	1.8×10^{-3}	6.2×10^1
		Virus	3.0×10^{-2}	9.8×10^2
2	Chaves Co., NM	Bacteria	6.6×10^{-8}	3.8×10^1
		Virus	1.1×10^{-6}	6.2×10^2
3	Clinton Co., IA	Bacteria	1.6×10^{-4}	5.3×10^1
		Virus	2.8×10^{-3}	8.5×10^2
4	Highlands Co., FL	Bacteria	4.6×10^{-7}	4.8×10^1
		Virus	7.7×10^{-6}	7.5×10^2
5	Kern Co., CA	Bacteria	7.5×10^{-9}	3.7×10^1
		Virus	1.3×10^{-7}	3.0×10^2
6	Yakima Co., WA	Bacteria	2.0×10^{-8}	3.7×10^1
		Virus	3.4×10^{-7}	6.1×10^2

known. For realistic modeling and for use of the model in a regulatory framework, more information must be gathered on inactivation rates of viruses and soil-water suspension of bacteria and (especially) viruses under natural conditions in soil.

6.6. SOURCES OF UNCERTAINTY

The site-specific values of many of the parameters used in the model may not be known with certainty. Soil properties [WCSAT, P(6), USATCND, P(7) and GSATCND, P(8)] may be available from site characterizations, but the inherent variability of soil structure means that a single soil sample may not be representative of the entire unsaturated zone. Depth to the saturated zone [DSATZN, P(1)] may vary seasonally and with the amount of water in the trenches. If the model is to be used to assess a potential site for its suitability as a sludge landfill, it will be necessary to gather site-specific information to reduce these uncertainties. If it is known that soil types vary between the soil surface and the aquifer or between the landfill site and the well in question, the model can be run with data describing each soil type and the most protective results can be used to establish concentration limits or set-back distances.

Properties of sludge [SOLIDS, P(10) and BLKDEN, P(11)] may vary from batch to batch and may not be determined for every batch of sludge submitted for disposal. Comparative model runs (Chapter 7) have shown that these factors do not have a major impact on model outcome, so it is not essential to know them precisely. PATHDN is specific to each batch of sludge and varies with different processes as well as different mixtures of raw sewage. PATHDN cannot be predicted accurately from process knowledge; therefore, direct determination of pathogen concentrations is necessary for use of the model. Similarly, the resuspension coefficient SSPNDB [P(17)] should vary with both the nature of the pathogen and the composition of the sludge-water mixture. There is not yet sufficient information about the relationships of the many interacting factors to allow reliable predictions of pathogen resuspension from sludge. More research should be done to find reliable ways to determine the leaching characteristics of sludge-bound pathogens.

Inactivation rates [P(14), P(15) and P(16)] under field conditions are not known with much accuracy, nor are solids-to-water suspension factors [P(17), P(18)]. Infective doses vary greatly among pathogens, among populations of pathogens with different histories and among

populations exposed to a given pathogen. Inactivation rates and infective doses are extremely important to model outcome and need to be predicted accurately.

Groundwater transport parameters may be not be known accurately. Retardation coefficients for pathogens are calculated from input parameters but are not known under field conditions, nor are hydrodynamic dispersion coefficients. These factors are of much greater importance than inactivation rates and infective doses. The groundwater transport subroutine used in this model is simple and may not be highly accurate. However, the imprecision of the input data may be less than the uncertainties caused by variability of soil properties.

The groundwater transport algorithm has not been validated. Many attempts have been made to model transport of pathogens in groundwater, but all are limited by lack of suitable field validation data. Most assume homogeneous conditions in the soil and constant flow rates and inactivation. Therefore, since the assumed conditions are not realistic, none of the models is likely to predict pathogen movement accurately.

A large contribution to the inherent uncertainty of the model is the number of pathogens and pattern of exposure required to produce an infection. Estimates of the minimum infective dose for bacteria and viruses vary considerably depending on several factors: route and vehicles of exposure (inhaled, ingested, in liquid, food or capsules); timing of the exposure (whether acute or chronic); resistance mechanisms of the host, including immune responses or barriers to infection such as stomach acidity and enzymes, circulating leucocytes, etc.; general health and age of the host; incidental treatment with antibiotics, which may reduce competition by normal bacterial flora and thus allow pathogens to colonize more readily; and virulence of the strain or preparation of pathogens used in the study. Especially with viruses, enumeration techniques may not be efficient, so the number of viruses in an administered dose may not be known accurately. The beta-Poisson model allows for variability of susceptibility in the test populations, so it is probably adequate to describe risks to sensitive individuals.

In the SLDGFILL model, each day's exposure is considered to provide a risk of infection independent of any other day's exposure. That is, the exposure is cumulative through that day, but there is no accumulation beyond that day. The host is assumed to become neither sensitized nor immune to the pathogen, and the host population is assumed to be homogeneous in its response to exposure. The endpoint of the assessment is infection, whether or not clinical

disease is observed. These principles are also followed in the U.S. EPA *Draft Ground-water Disinfection Rule* (U.S. EPA, 1992). Infection is assumed in this model to follow the beta-Poisson model presented in the draft rule (Regli et al., 1991; U.S. EPA, 1992).

Because there is little information that can be used to derive accurate estimates of infective dose (U.S. EPA, 1992), estimation of the risk of infection from a large number of sludge pathogens cannot be modeled with a known level of confidence. With more detailed information on infective doses, the model could more effectively be used to evaluate the relative benefits of treatment technologies that have differential effects on different pathogens.

In summary, uncertainties result from both the model structure and the input data. Structural uncertainty remains because the equations that represent the model structure may not include all of the important processes or may not represent them accurately. Uncertainties that result from the model structure are difficult to quantify until the model results are compared with actual field measurements. Structural uncertainty can be minimized by applying the model only to the situations for which it was designed. For example, SLDGFILL should be used to predict average concentrations of pathogens in groundwater over a year. It is not designed to represent short-term changes during storm events.

Parameter uncertainty results from parameters that cannot be easily measured in the field and parameters that vary spatially and temporally. The effects of parameter uncertainty are quantified in the sensitivity analysis in Chapter 7. The parameter uncertainty that will have the greatest effect on the results at a site depends on the potential parameter ranges for that site. A site-specific sensitivity analysis may be used to evaluate the benefit of making additional measurements to narrow parameter ranges. Defining those parameter values to which the model is most sensitive will have the greatest effect on minimizing uncertainty of the results.

7. SENSITIVITY ANALYSIS

The results presented in Section 6.5 showed that several site-specific parameters governing the amount and rate of leaching by rainwater had significant effects on the rates of simulated pathogen transport. To determine whether other parameters were of more importance, the model was tested by systematically varying each parameter over a realistic range of values. Preliminary model runs had shown that the rate of pathogen transport depends strongly on SSPNDS [P(18)]. SSPNDS is used to calculate the concentration of pathogens in soil water when the concentration in bulk soil is known, the retardation coefficients for unsaturated-zone (RUS) and saturated-zone (RGW) transport and the hydrodynamic dispersion coefficients (DGW and DUNSAT). It seemed that the effect on retardation was a likely cause for the dependence of transport on SSPNDS. Figure 7-1 shows the dependence of RGW on both SSPNDS and PORWTR [P(3)], two parameters used in the calculation of RGW. RGW is directly proportional to the value of SSPNDS and inversely proportional to PORWTR. Since RGW is the ratio of the rate of transport of pathogens in water to the rate of movement of the water, it is clear that high values of SSPNDS should make pathogens migrate very slowly in groundwater.

The rate of migration of pathogens in soil water in the unsaturated zone also depends on retardation, which is calculated from SSPNDS and parameters describing unsaturated soil moisture. Figure 7-2 shows the effect of SSPNDS on the rate of migration through the unsaturated zone. The value plotted as breakthrough time is the time required for the concentration of viruses in the aquifer to reach $2 \times 10^{-7}/L$, the proposed limit in drinking water (U.S. EPA, 1992), when DSATZN=5 cm and SSPNDS is varied. This figure confirms the marked sensitivity of the model to SSPNDS.

Because the times required to achieve maximum or steady-state concentrations under default conditions were much longer than the model can be run, the remainder of the sensitivity analysis was done using a version of the model in which SSPNDS is not used to calculate RGW and RUS. In this version of the model, SSPNDS=0 in the calculation of retardation and dispersion coefficients, but not in the calculation of particle/water resuspension. Unless otherwise noted, all parameters were held at default values. The parameter values were based on the information found in Chapter 4. Parameters for both bacteria and viruses were tested.

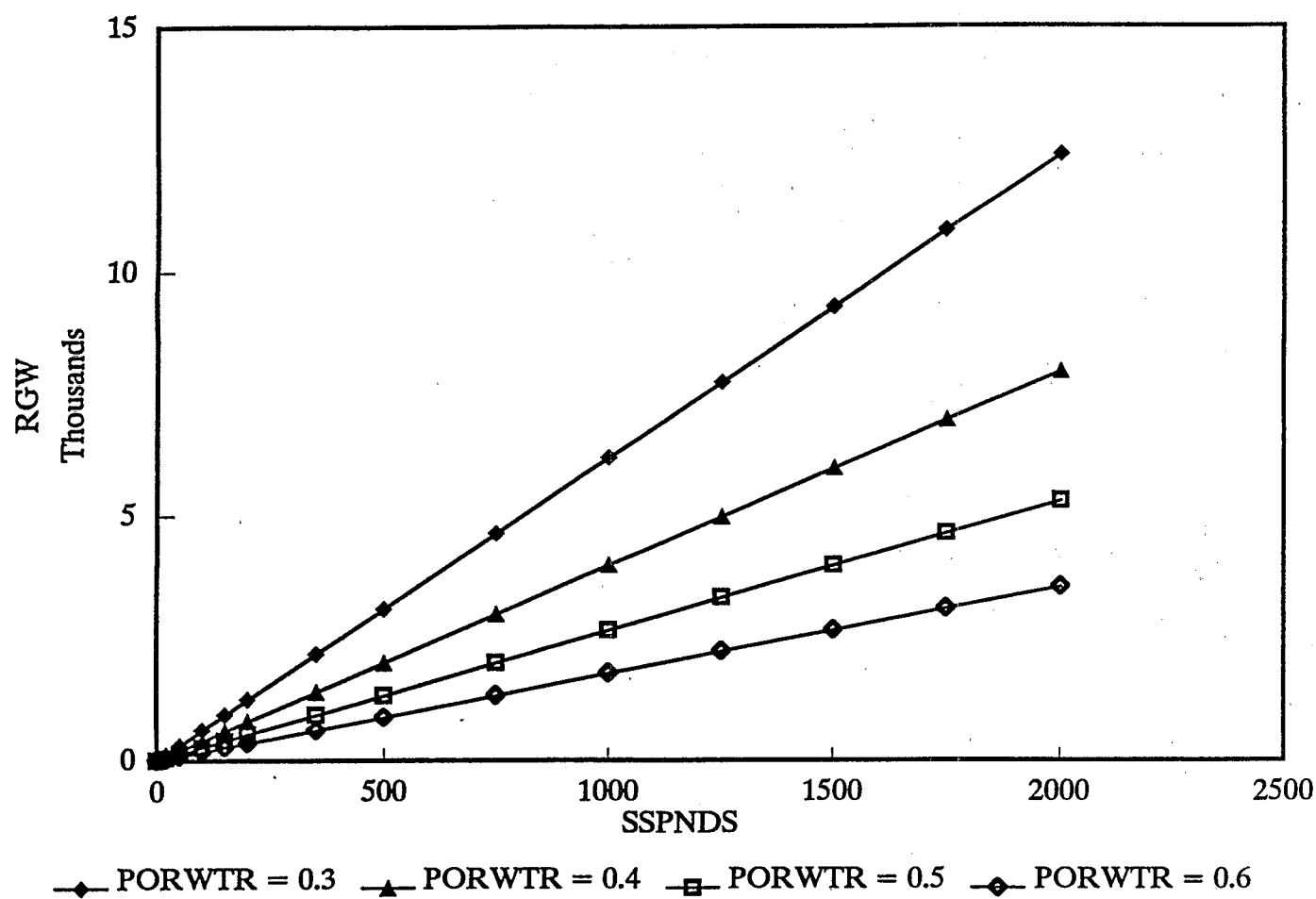
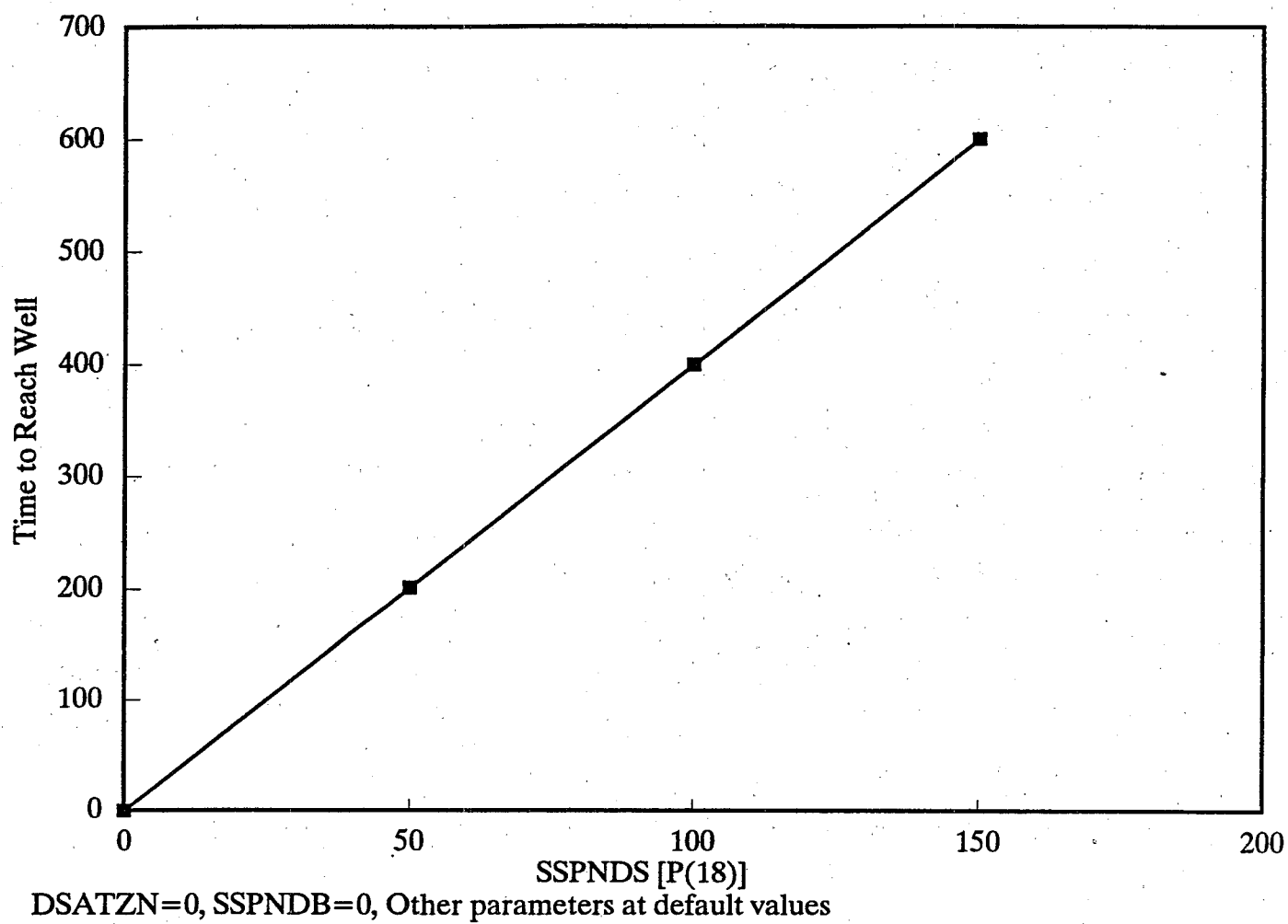


Figure 7-1. Dependence of Retardation Factor on SSPNDS and PORWTR



**Figure 7-2. Effect of SSPNDS on
Pathogen Transport Rate in Saturated Soil**

Results for bacteria are presented in Section 7.1, and results for viruses are shown in Section 7.2. The range of outcomes was examined to determine how sensitive the model is to each parameter.

Pathogen concentrations in each compartment reached steady-state levels representing the maximum observed concentrations for each combination of parameter values. In some cases, the time at which steady state was achieved was determined. This value was calculated arbitrarily as the day on which the concentration of pathogens in water in a given compartment was not more than 2% higher than the concentration 20 days earlier.

7.1. SENSITIVITY TESTING FOR BACTERIA

7.1.1. Parameter Values. The parameter values used for bacterial pathogens are listed in Table 7-1.

7.1.2. Results for Bacteria.

7.1.2.1. Dependence on Parameters--Individual parameters were varied over the ranges of values shown in Table 7-1 to determine the sensitivity of the model to each parameter for bacteria. Table 7-2 shows the calculated concentrations of bacteria in the groundwater well, saturated soil and unsaturated soil at 240 days. The maximum differences observed are plotted against parameter numbers in Figures 7-3 and 7-4. Figure 7-3 shows that two parameters, PATHDN [P(13)] and SSPNDB [(P18)], have the most effect on bacterial concentrations. PATHDN is the concentration of pathogens in sludge, and SSPNDB is the resuspension factor describing the ratio of the sludge pathogens associated with sludge particles to those in water suspension. Figure 7-4 shows the parameters that have a secondary effect on the outcome. Of these, DSATZN [P(1)] is the most important to concentration in unsaturated soil but not in the groundwater well. Inactivation coefficients in sludge and groundwater are important, as are some of the groundwater transport parameters. The effects of groups of parameters on concentration are discussed in more detail below.

7.1.2.2. Site-specific Parameters--Site-specific parameters describe the landfill site, including properties of the soil between the sludge trench and the groundwater aquifer. The parameters in this group are DSATZN [P(1)], AQUIER [P(2)], PORWTR [P(3)], ANRAIN [P(4)], EVAP [P(5)], WCSAT [P(6)], USATCND [P(7)], GSATCND [P(8)] and SMRSLP

**Table 7-1. Parameters Used to Test
the SLDGFILL Model for Bacteria**

Parameter			Run number					
No.	Name	Base	1	2	3	4	5	6
1	DSATZN	2.0	0.0001	2	4	7	10	20
2	AQUIFR ^a	10	5	5	10	10	10	20
3	PORWTR ^a	0.3	0.4	0.8	0.2	0.4	0.8	0.2
4	ANRAIN	150	50	100	150	200	500	-
5	EVAP	0.5	0.1	0.2	0.5	0.7	-	-
6	WCSAT	0.4	0.2	0.4	0.6	-	-	-
7	USATCND	10 ⁻⁷	10 ⁻⁸	10 ⁻⁷	10 ⁻⁶	-	-	-
8	GSATCND	10 ⁻⁷						
9	DEPTH	3.5	1	2	3.5	5	10	-
10	SOLIDS	0.17	0.1	0.2	0.25	0.3	-	-
11	BLKDEN	1.3	1.1	1.2	1.3	1.5	-	-
12	SMRSLP	9	7	8	9	10	11	-
13	PATHDN	5×10 ⁴	1×10 ²	5×10 ³	5×10 ⁴	5×10 ⁵	5×10 ⁶	-
14	INACTB	0	0.0	0.001	0.01	0.1	1	-
15	INACTS	0.016	0.005	0.01	0.02	0.1	1	-
16	INACTW	0.0228	0.005	0.02	0.1	0.5	1	-
17	SSPNDB	2000	2000	200	20	10	-	-
18	SSPNDS	1000	1000	100	20	10	-	-
	VGW ^b	3.6	0.36	1.08	3.6	10.8	36	-
	DGW ^b	60	20	40	60	80	100	-
23	XWELL	50	20	50	100	150	200	-

^a These parameters were varied together, as shown by the joined cells.

^b Subsurface transport parameter, usually calculated from SSPNDS and other parameters.

**Table 7-2. Bacterial Concentrations
at 240 Days in Model Test Runs**

Parameter	Run	Well	Pathogens/L Sat.	Unsat.
Base	0	3.14	6.53	17.5
DSATZN	1	6.7	14	24.9
P(1)	2	3.14	6.53	17.5
	3	1.47	3.06	12.9
	4	0.465	0.976	8.93
	5	0.145	0.309	6.73
	6	0.00208	0.00552	3.89
AQUIFR +	1	3.14	6.53	17.5
PORWTR	2	3.14	6.53	17.5
P(2) and	3	3.14	6.53	17.5
P(3)	4	3.14	6.53	17.5
	5	3.14	6.53	17.5
	6	3.14	6.53	17.5
ANRAIN	1	3.06	6.38	17.3
P(4)	2	3.1	6.46	17.4
	3	3.14	6.53	17.5
	4	3.17	6.61	17.6
	5	3.38	7.03	18.1
EVAP	1	3.22	6.71	17.7
P(5)	2	3.2	6.67	17.7
	3	3.14	6.53	17.5
	4	3.09	6.44	17.4
WCSAT	1	3.25	6.77	17.8
P(6)	2	3.14	6.53	17.5
	3	3.1	6.46	17.4
USATCND,	1	3.13	6.51	17.5
GSATCND	2	3.14	6.53	17.5
P(7) and P(8)	3	3.15	6.56	17.5

Table 7-2. (continued)

Parameter	Run	Well	Pathogens/L Sat.	Unsat.
DEPTH P(9)	1	3.14	6.53	17.5
	2	3.14	6.53	17.5
	3	3.14	6.53	17.5
	4	3.14	6.54	17.5
	5	3.14	6.54	17.5
SOLIDS P(10)	1	3.13	6.52	17.5
	2	3.14	6.54	17.5
	3	3.14	6.54	17.5
	4	3.14	6.54	17.5
BLKDEN P(11)	1	3.14	6.53	17.5
	2	3.14	6.53	17.5
	3	3.14	6.53	17.5
	4	3.14	6.53	17.5
SMRSLP P(12)	1	3.14	6.54	17.5
	2	3.14	6.54	17.5
	3	3.14	6.53	17.5
	4	3.14	6.53	17.5
	5	3.14	6.53	17.5
PATHDN P(13)	1	0.00627	0.0131	0.035
	2	0.314	0.653	1.75
	3	3.14	6.53	17.5
	4	31.4	65.3	17.5
	5	31.4	65.3	17.50
INACTB P(14)	1	3.14	6.53	17.5
	2	2.16	4.14	10.3
	3	0.086	0.087	0.0949
	4	0.000371	0.000299	0.00012
	5	0	0	0
INACTS P(15)	1	3.14	6.53	17.5
	2	3.14	6.53	17.5
	3	3.14	6.53	17.5
	4	3.14	6.53	17.5
	5	3.14	6.53	17.5

Table 7-2. (continued)

Parameter	Run	Well	Pathogens/L Sat.	Unsat.
INACTW P(16)	1	12.9	15.1	21
	2	3.87	7.32	17.9
	3	0.0167	0.948	12.4
	4	1.8E-12	0.0629	6.89
	5	3.6E-23	0.0133	5.16
SSPNDB P(17)	1	3.14	6.53	17.5
	2	30.6	63.8171	
	3	248	516	1380
	4	411	850	2260
SSPNDS P(18)	1	3.14	6.53	17.5
	2	3.14	6.53	17.5
	3	3.14	6.53	17.5
	4	3.14	6.53	17.5
DUNSAT	1	1.88	3.92	14.2
	2	2.67	5.56	16.4
	3	3.14	6.53	17.5
	4	3.46	7.2	18.3
	5	3.7	7.7	18.8
VGW	1	0.000011	1.53	17.5
	2	0.16	2.89	17.5
	3	3.14	6.53	17.5
	4	7.52	9.45	17.5
	5	10.2	10.9	17.5
DGW	1	3.13	6.53	17.5
	2	3.13	6.53	17.5
	3	3.14	6.53	17.5
	4	3.14	6.54	17.5
	5	3.15	6.54	17.5
XWELL P(23)	1	6.9	9.09	17.5
	2	3.14	6.53	17.5
	3	0.84	4.22	17.5
	4	0.219	3.05	17.5
	5	0.0235	2.4	17.5

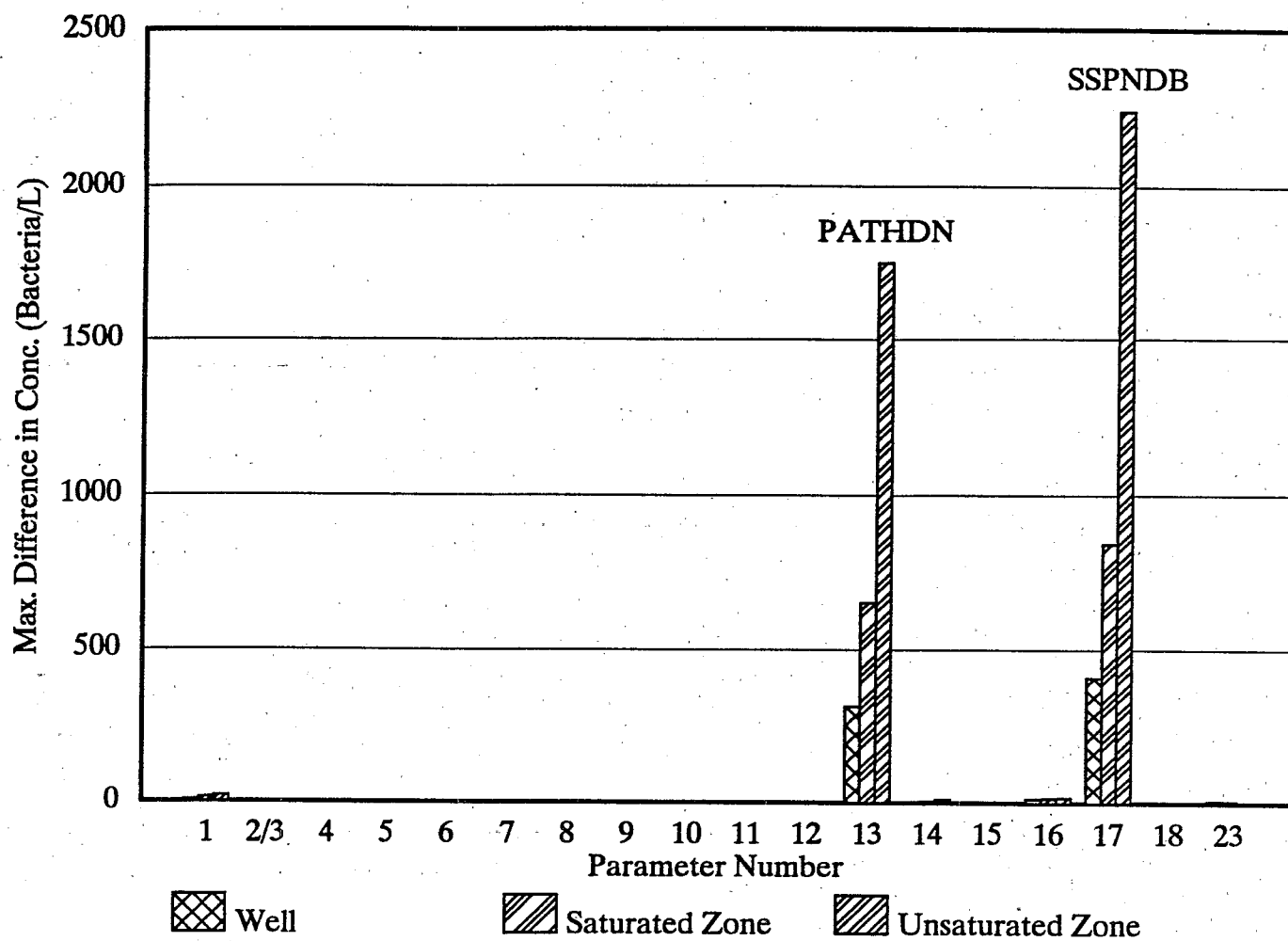


Figure 7-3. Parameters with Primary Impact on Model Output

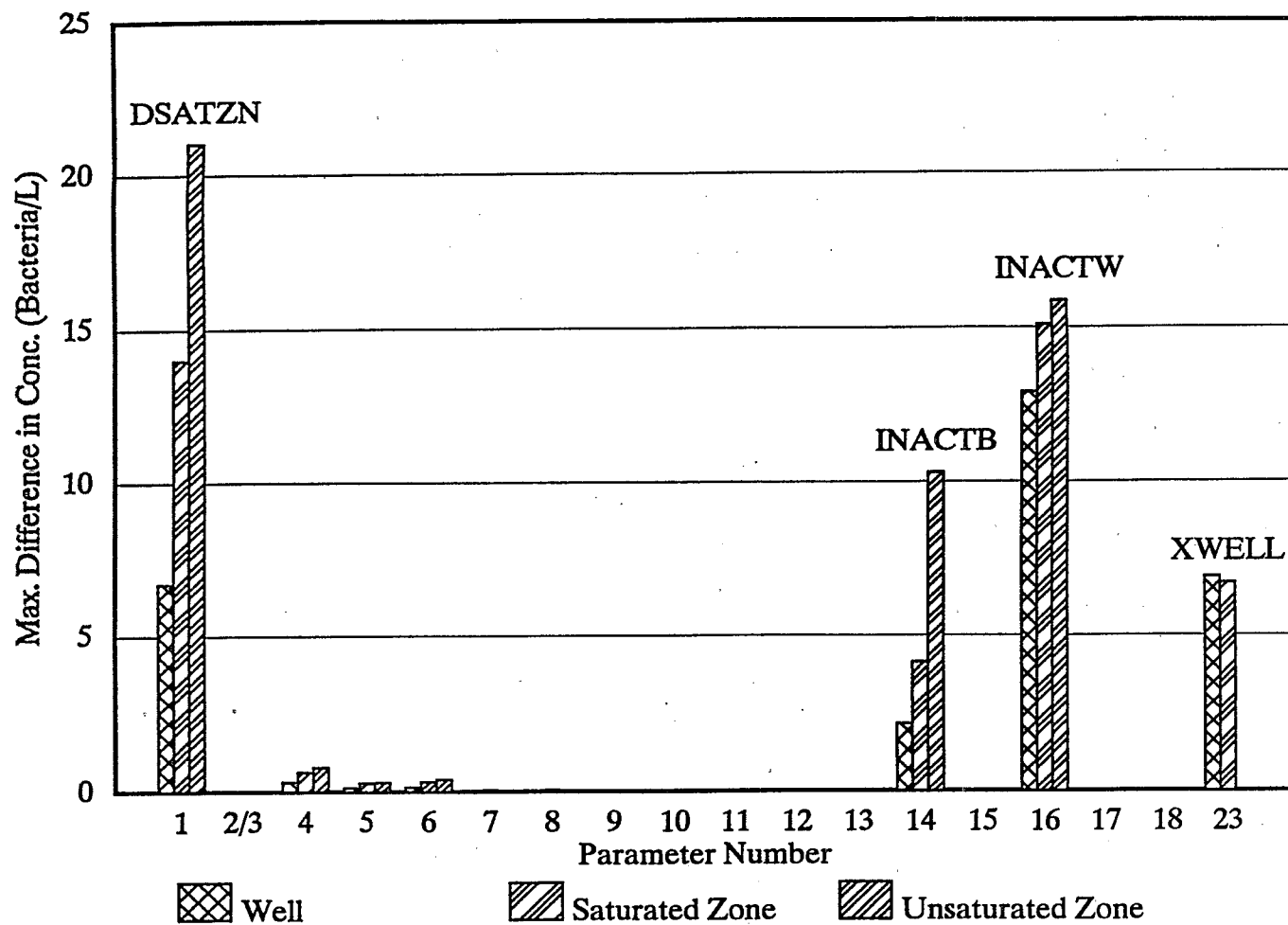


Figure 7-4. Parameters with Secondary Impact on Model Output

[P(12)]. Of these parameters, only DSATZN [P(1)] had a significant impact on bacterial pathogen concentrations and time to steady state. Figure 7-5 shows that both the maximum concentration of bacteria in the groundwater well and the time required to reach that concentration depend strongly on DSATZN. When all of the soil beneath the sludge layer is saturated (DSATZN=0) and retardation factors equal 1, the pathogen concentration at the well rises rapidly and remains high. With thicker layers of unsaturated soil, the well water pathogen concentrations rise later and reach lower steady-state levels. Figure 7-6 shows that even a shallow layer of unsaturated soil beneath the sludge trench greatly reduces the concentration of bacterial pathogens reaching the well. The time at which the pathogen concentration reaches steady state is shown in Figure 7-7. It is clear from this figure that unsaturated soil significantly retards the transport of pathogens even when the retardation factors are artificially low. Although the model does not allow for a transient rise of the water table to the sludge layer, the foregoing results show that that eventuality could increase the concentration of pathogens in groundwater and should be prevented by design parameters for a sludge monofill.

The size and volumetric moisture content of the aquifer do not have any effect on the concentration of pathogens in water in either the saturated soil or in the well. These parameters would be important if the pathogens were mixed throughout the vertical extent of the aquifer. Over short distances, however, vertical mixing of pathogens with the water in the saturated soil compartment is negligible (U.S. EPA, 1989e). In addition, groundwater protection regulations require that releases to Class I groundwater, which is defined as groundwater in use as a source of drinking water, meet requirements for quality without prior dilution. Therefore, it is most appropriate for the size of the aquifer not to be taken into consideration by the model.

The amount of rainfall to which the sludge is subjected has a minor effect on bacterial concentration in the well (Figure 7-4). Apparently, permeability of the sludge layer is so low that excessive rainfall does not greatly increase the rate of leaching from the sludge. The saturated water content of subsurface soil [WCSAT, P(6)] also has a minor effect on pathogen concentration in the well.

7.1.2.3. Bulk Sludge Parameters--These parameters describe bulk sludge in a full trench (or mounded area). They are DEPTH [P(9)], SOLIDS [P(10)], BLKDEN [P(11)] and PATHDN [P(13)]. The most important of the sludge-specific parameters is the concentration

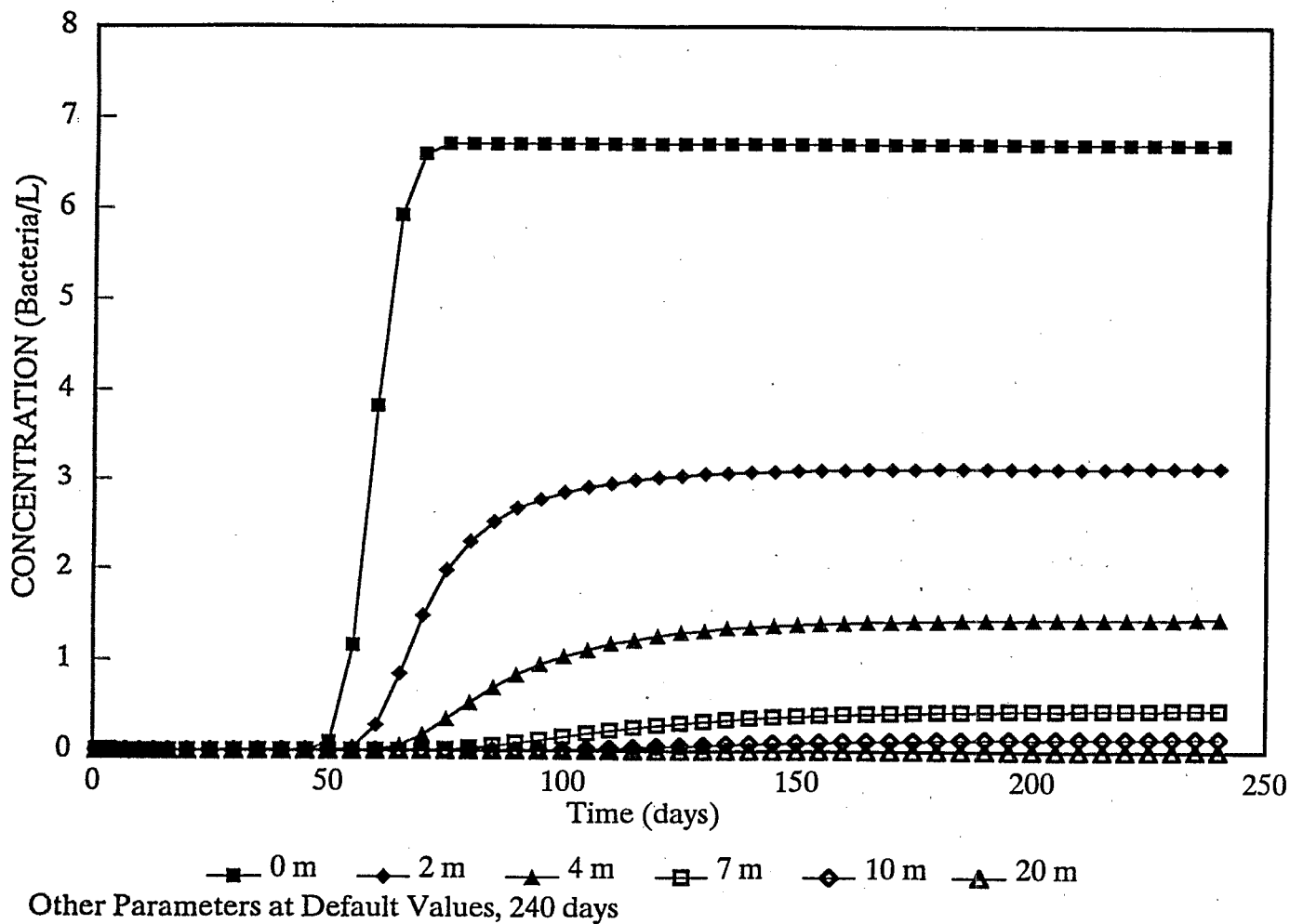
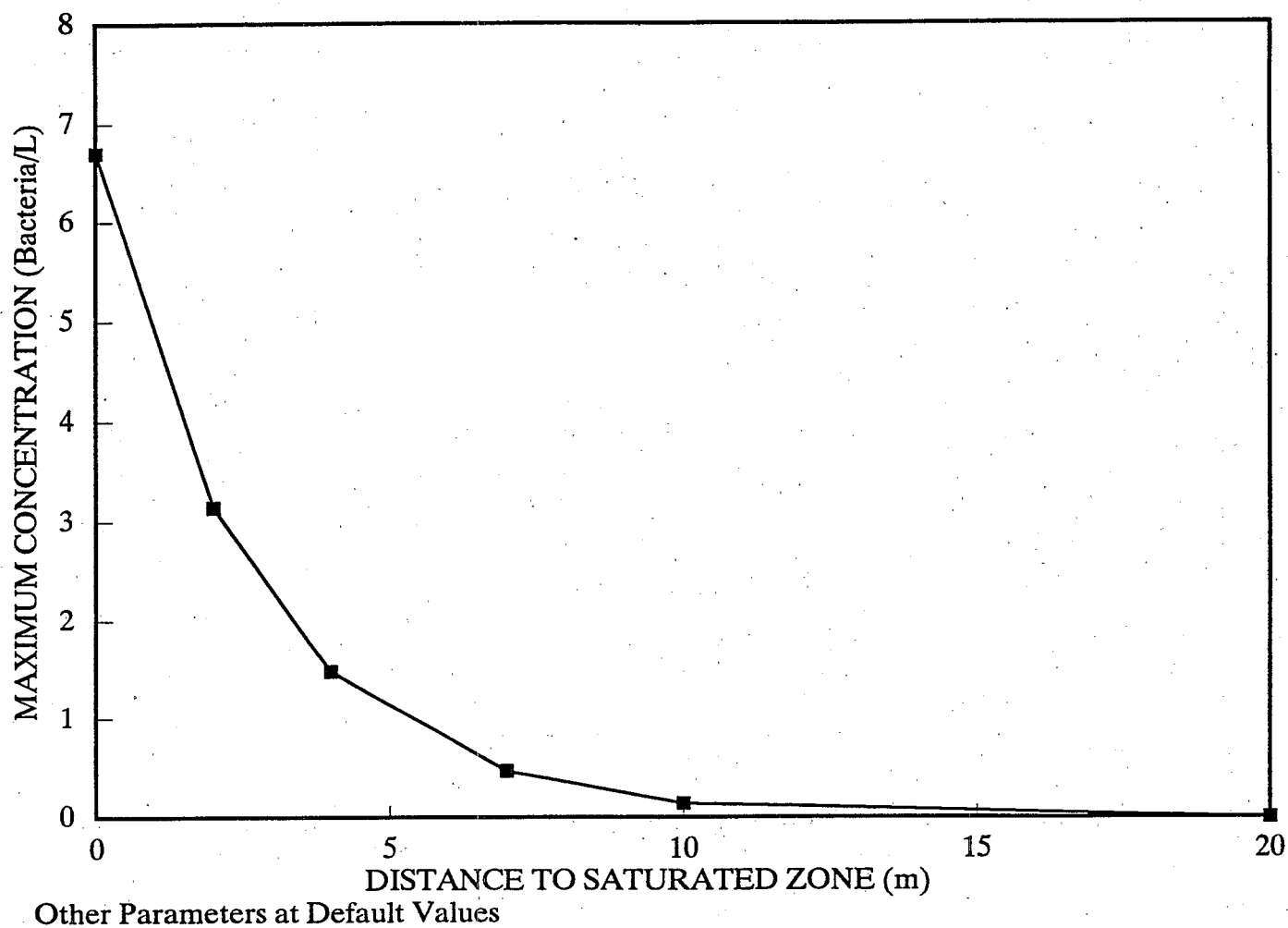


Figure 7-5. Dependence of Pathogen Transport Kinetics on DSATZN



**Figure 7-6. Dependence of Drinking Water
Bacterial Concentration on DSATZN**

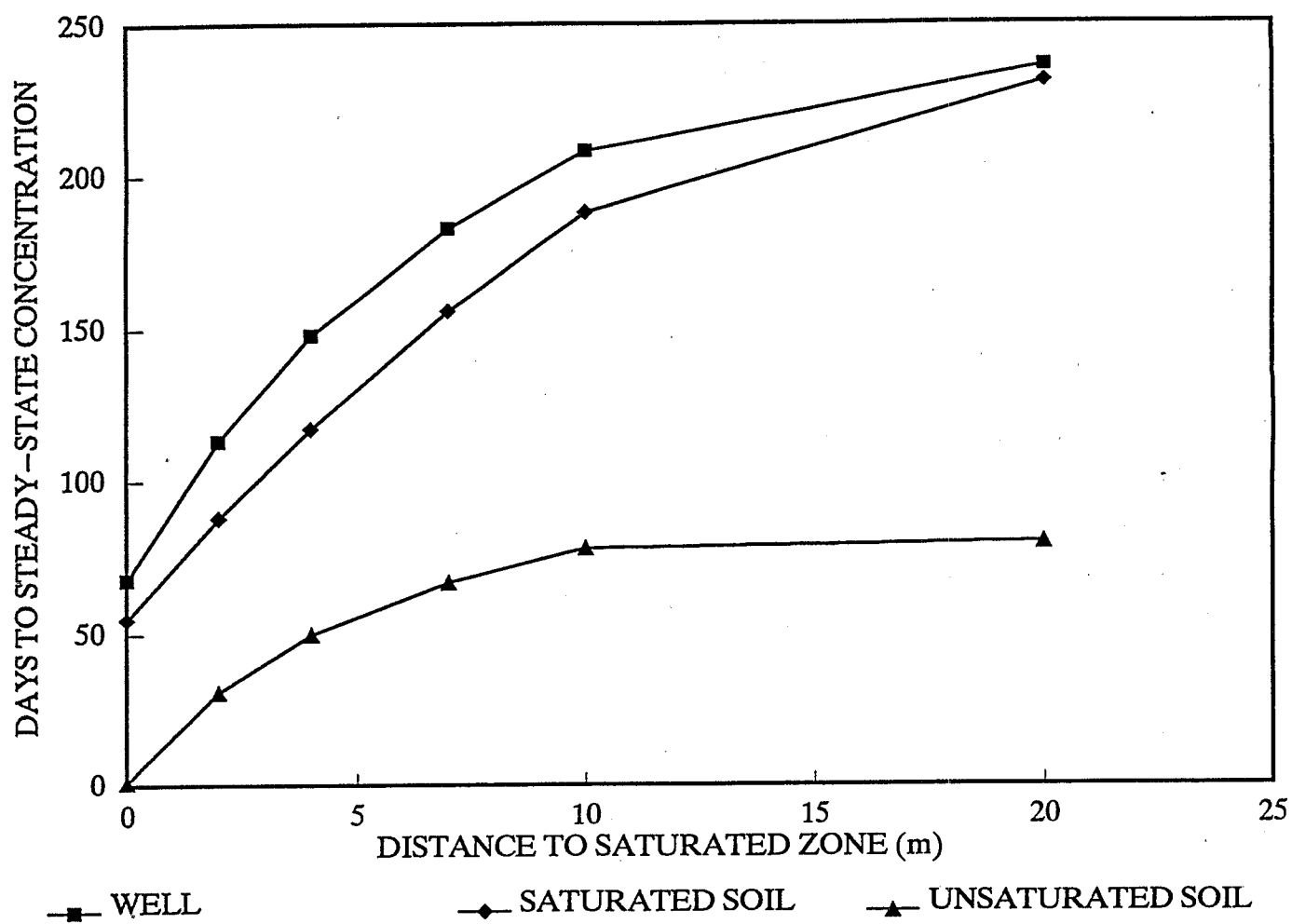


Figure 7-7. Dependence of Compartment Equilibration on DSATZN

of pathogens in the sludge [PATHDN, P(13)]. Figure 7-3 shows the relative importance of bacterial concentration when compared to other parameters. The steady-state bacterial concentration of pathogens in each compartment is directly proportional to PATHDN (Figure 7-8). As with the thickness of the aquifer, the thickness of the sludge layer [DEPTH, P(9)] has no significant effect on the steady-state concentration of bacterial pathogens. However, the length of time over which pathogens can be released without the sludge being depleted of pathogens does depend on depth. These results suggest that the concentration of pathogens in the sludge determines the magnitude of the transport to groundwater, while the amount of sludge determines the duration of the release.

7.1.2.4. Organism-specific Parameters--Organism-specific properties are INACTB [P(14)], INACTS [P(15)], INACTW [P(16)], SSPNDB [P(17)] and SSPNDS [P(18)]. Figure 7-3 shows that SSPNDB [P(17)] was one of the two most significant parameters for bacterial concentration. Figure 7-9 shows that bacterial concentrations are not directly proportional to SSPNDB, but tend to level off as SSPNDB decreases. Like PATHDN [P(13)], this parameter plays a key role in determining the concentration of pathogens in leachate entering unsaturated soil from the sludge. Reliable data on suspension of sludge pathogens in water are crucial to realistic model predictions but are not available.

The most significant parameters for risk of infection are the infective dose parameters alpha and beta [INFALF, P(20) and INFBET, P(21)], although these parameters have no effect on concentration of organisms in any compartment. Figure 7-10 shows the effects of different combinations of alpha and beta on the daily probability of infection. These default values are intended to be reasonable rather than overly conservative. As shown in Table 2-2 and as reported by Kowal (1985), Casemore (1991) and Ward et al. (1986), the estimated ranges of minimum infective doses for infection by enteroviruses are from 1 to $>10^7$, and for bacteria from 10 to 10^{10} .

U.S. EPA (1992) has proposed that the maximum allowable daily risk of infection with enteric microorganisms from ingestion of groundwater should be 2.7×10^{-7} (corresponding to an annual risk of 1×10^{-4}). Assuming the exposed individual drinks 2 L of groundwater daily, the allowable concentration of pathogens in the groundwater is determined by the infective dose. Data for rotavirus infection (Regli et al., 1991) imply that the maximum allowable virus

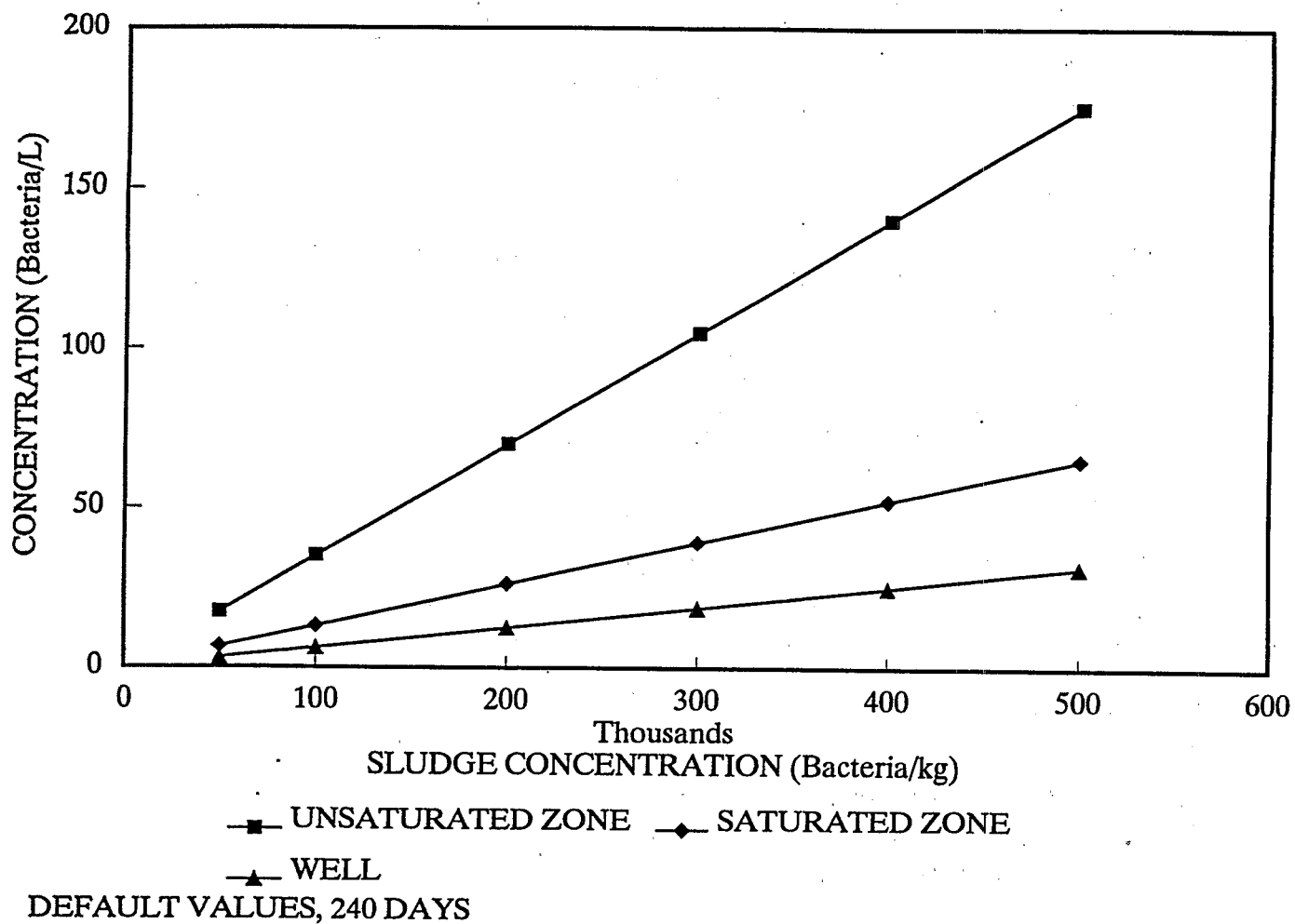


Figure 7-8. Dependence of Model Outcome on Sludge Pathogen Concentration

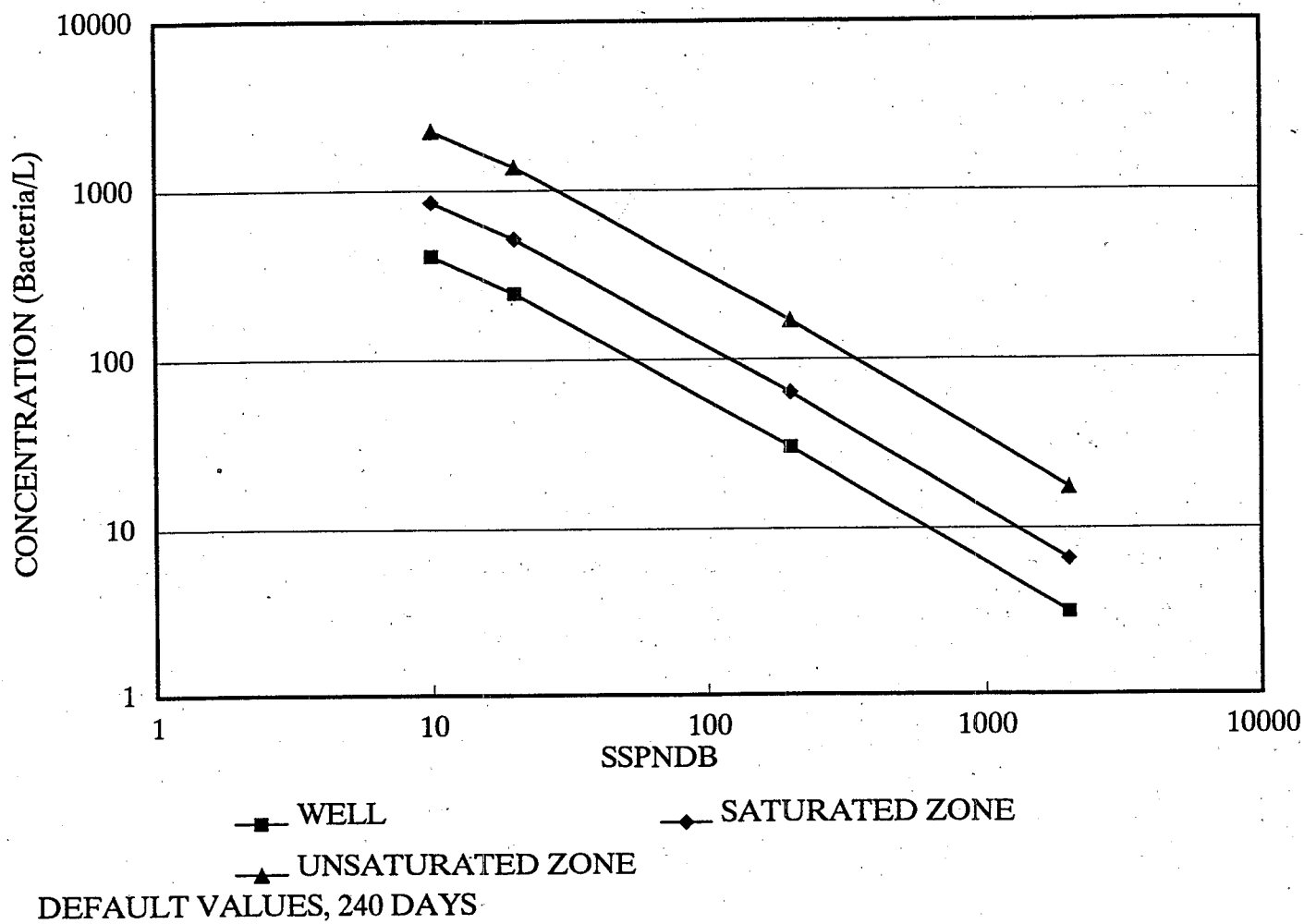


Figure 7-9. Dependence of Model Outcome on SSPNDB

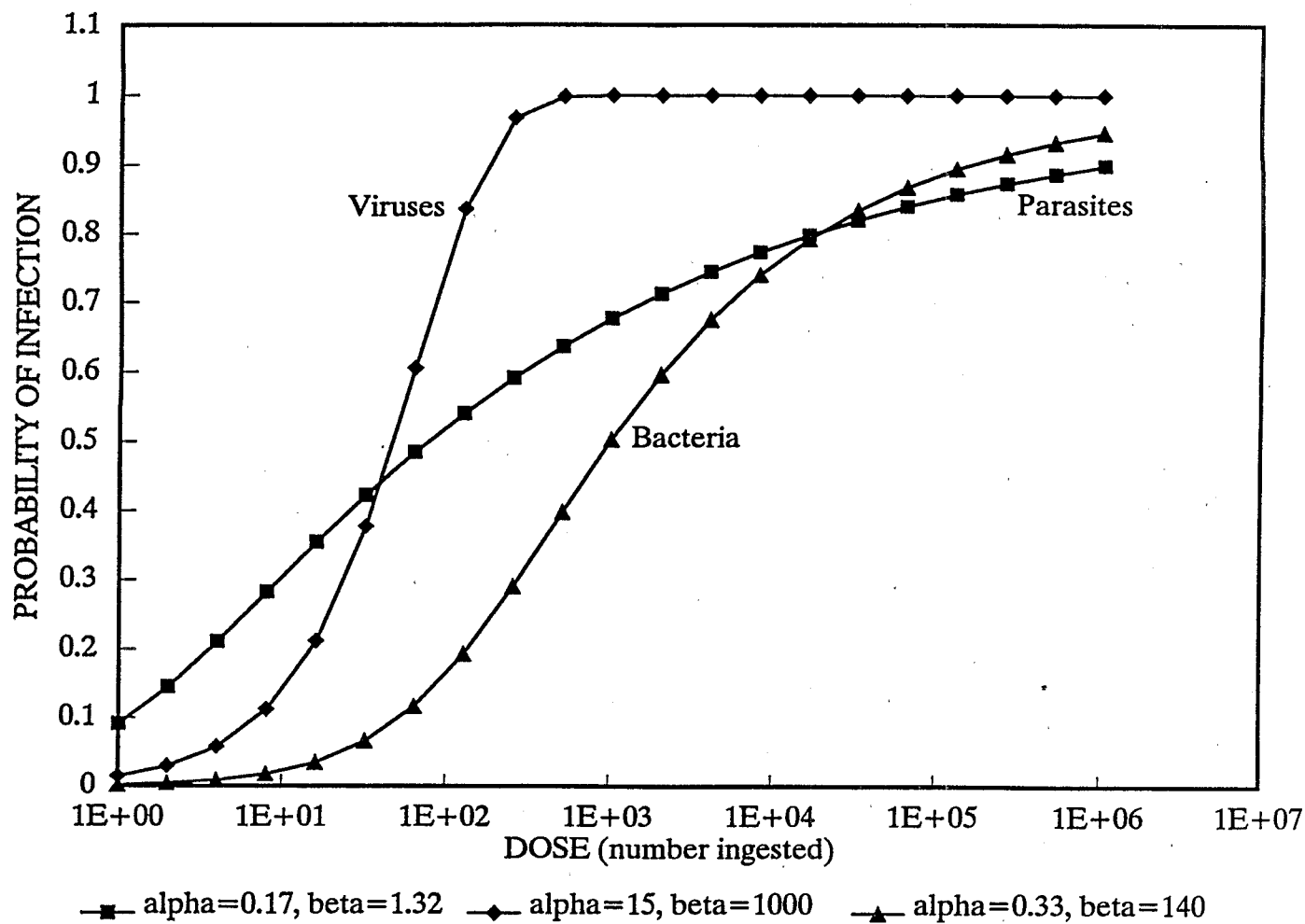


Figure 7-10. Dependence of Infection Risk on Infectivity Parameters

concentration is $2 \times 10^{-7}/L$, a concentration difficult to attain and to verify. Results of model runs in which SSPNDS is used to calculate retardation imply that even with no retardation ($RGW=1$) a concentration of 2×10^{-7} is unlikely to occur at a well, although that concentration might be achieved in groundwater in direct contact with the sludge layer.

Persistence of pathogens in the environment is typically limited because conditions in soil and water are not optimum for pathogens. Inactivation rates are described by parameters $P(14)$, $P(15)$ and $P(16)$. Of these, the inactivation rate in water [$INACTW$, $P(16)$] has the most effect on pathogen concentration, and the inactivation rate in bulk sludge [$INACTB$, $P(14)$] is also significant (Figure 7-3). Because inactivation is a composite function of inactivation rate and elapsed time, pathogen concentrations in groundwater depend on the inactivation rate, the distance to the well and the velocity of groundwater travel. As a conservative measure, it is assumed that bulk sludge is protective to pathogens found there. The protective properties of bulk sludge in the monofill are not expected to change markedly over time. Therefore, the default inactivation rate in sludge is an exponential rate of 0 logs/day. Other values for $INACTB$ [$P(14)$] should be used when/if data on die-off rates in bulk sludge become available.

Varying $INACTW$ [$P(16)$] changed the concentration of pathogens in each compartment, but in each case a steady-state level was reached (Figure 7-11). This was also true when a negative value for $INACTS$ [$P(15)$] was used to simulate growth of the bacteria in soil at an exponential rate of 0.05 logs/day. Because of the dynamic movement of organisms through the compartments, there is an equilibrium between growth or inactivation and transport: the steady-state level (and time at which steady-state is achieved) depends on that balance, but the general shape of the kinetic curve remains the same.

In contrast, with a non-zero $INACTB$ [$P(14)$], the concentration of bacteria in each compartment reached a maximum and then decreased as the pathogens in bulk sludge died off (Figure 7-12). This implies that if conditions in bulk sludge result in inactivation of pathogens, the pathogen concentration in groundwater should be much less than if there is no die-off in sludge. Conversely, if bacterial growth in the sludge layer is modeled by specifying a negative value for $INACTB$, the concentration of bacteria in the groundwater increases throughout the model run. This emphasizes the importance of maintaining conditions in the sludge that do not allow regrowth of pathogenic bacteria.

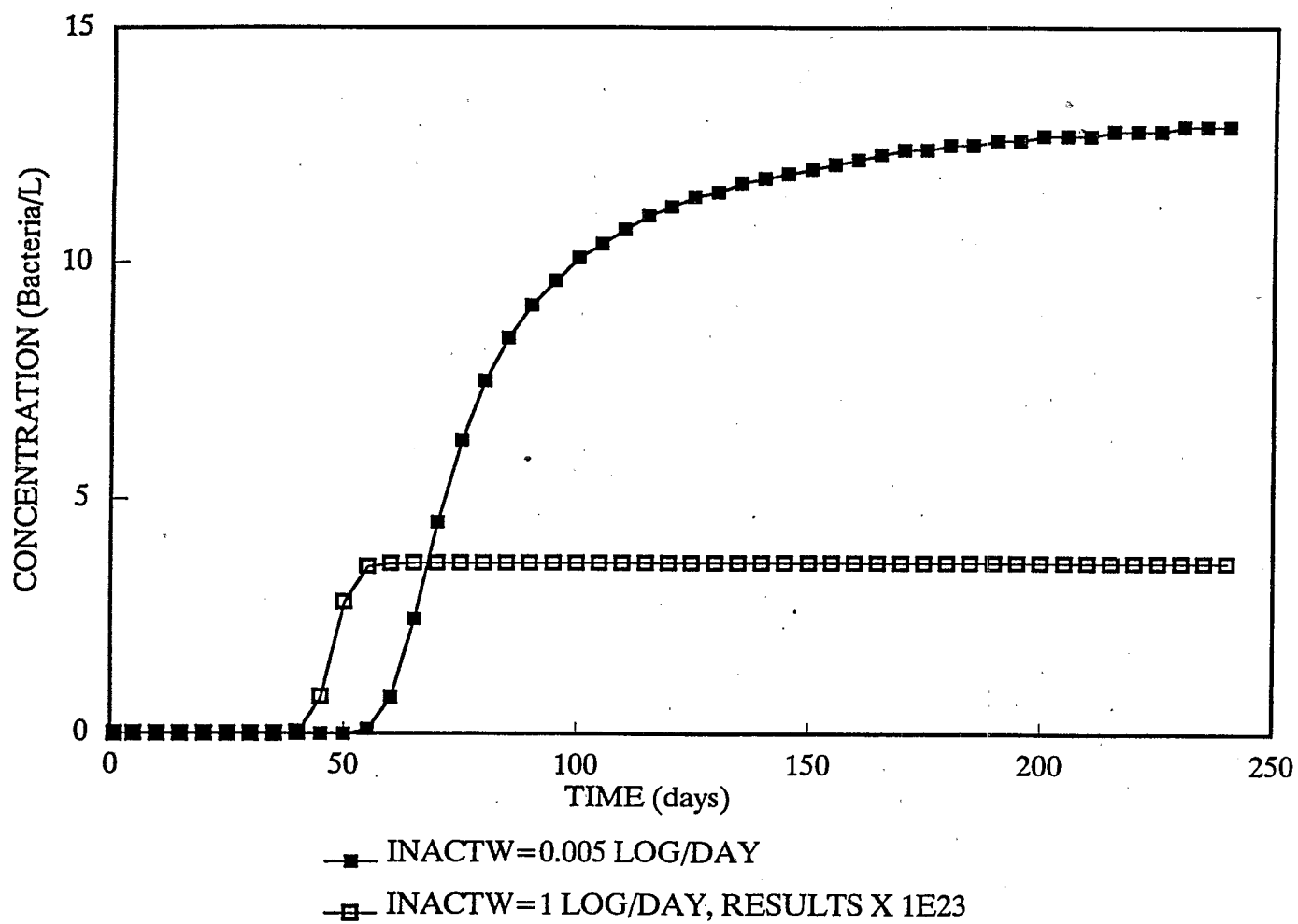


Figure 7-11. Effect of INACTW on Kinetics of Pathogen Transport

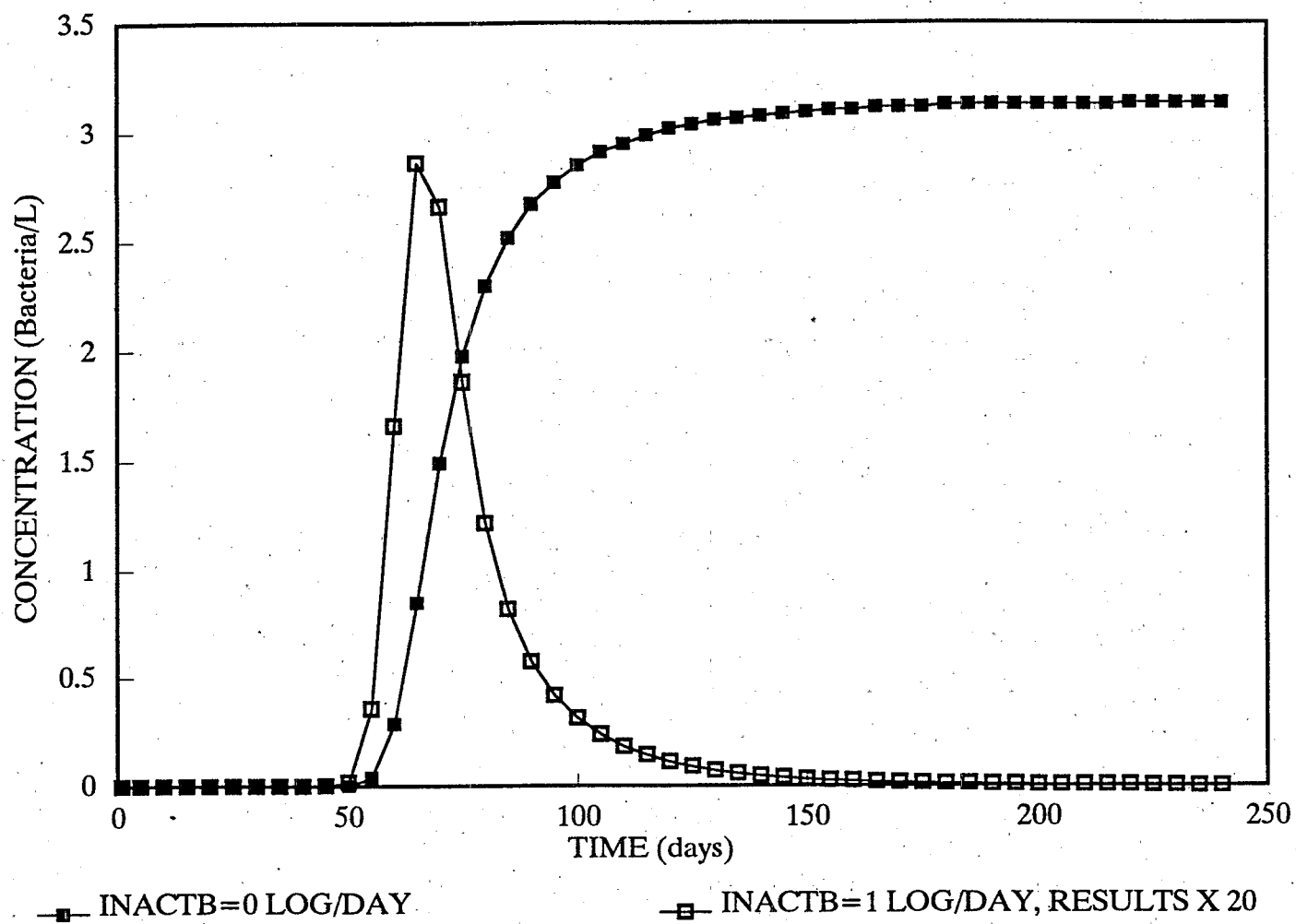


Figure 7-12. Effect of INACTB on Kinetics of Bacterial Transport

7.1.2.5. Groundwater Transport Parameters--Groundwater transport parameters describe the soil and groundwater through which the pathogens pass to the well. They are GRADI [P(22)] and XWELL [P(24)]. These parameters were tested in the model in which SSPNDS is used to calculate other groundwater parameters. Those results are discussed in the introduction to Chapter 7.

Parameters DUNSAT, VGW and DGW were tested in the modified model. DUNSAT has a small effect on bacterial concentrations in all three compartments, and VGW has a small effect on concentrations in only two, saturated soil and the well (Figure 7-4).

7.2. SENSITIVITY TESTING FOR VIRUSES

Because infection parameters are different for viruses, and because viruses are likely to provide a highly significant health risk in groundwater, a separate set of model runs was done to assess the sensitivity of predicted effects for viruses.

7.2.1. Parameter Values. The parameter values used for viral pathogens are those listed in Table 7-1. In many cases, these parameters are the same as for bacteria, except for the virus-specific default values such as density in sludge, infectivity parameters, resuspension factors and inactivation rates. These values are representative of a wide variety of viruses, and specific values for viruses of interest should be used to model their behavior. However, these values should be conservative enough to be at least as protective as is realistic for viral pathogens.

7.2.2. Results for Viruses. In every case, the concentration of viruses in soil pore water and groundwater was greater than that of bacteria. This result occurred because of the relatively low inactivation rates of viruses in soil and groundwater and because of lower values of resuspension coefficients for viruses than for bacteria. This underscores the significance of viruses as a potential health threat for surface disposal of sludge.

7.2.2.1. Kinetics--Kinetics of viral transport into the unsaturated zone, the saturated zone and then to the groundwater well are not qualitatively different from those for bacterial pathogens. Quantitative differences are discussed in the sections below dealing with parameter-specific sensitivities.

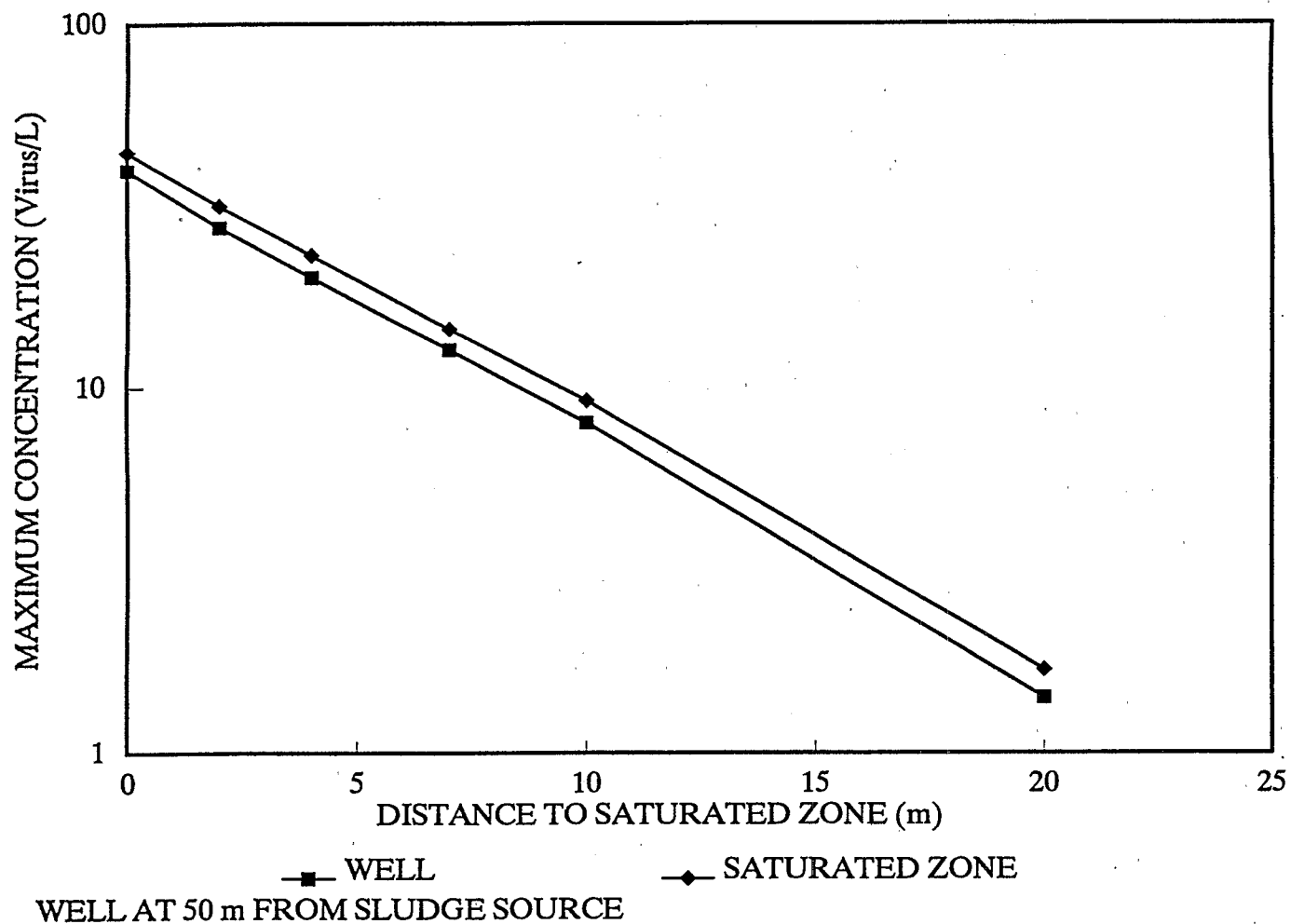
7.2.2.2. Site-specific Parameters--The only site-specific parameter showing a significant impact on concentration of viruses in the groundwater was DSATZN [P(1)], depth to the saturated zone (Figure 7-13). As with bacteria, the groundwater concentration decreased with increasing depth of unsaturated soil. Figure 7-13 shows that the maximum observed concentration was approximately logarithmically related to the depth of the unsaturated zone, reflecting the exponential inactivation of viruses with traverse time in the unsaturated zone. In these model runs the time at which a steady-state concentration was reached was also calculated. The results showed that increasing the depth of the unsaturated zone increases the time to reach steady-state concentrations in the saturated zone.

7.2.2.3. Bulk Sludge Parameters--As with bacteria, the maximum concentration of viruses in groundwater was proportional to their density in the sludge (Figure 7-14). The time required to reach steady-state levels was not affected by density. No other sludge-specific parameters had a significant effect on either the concentration of viruses in groundwater or the time at which steady-state concentrations were achieved.

7.2.2.4. Organism-specific Parameters--The inactivation rate of viruses in sludge [P(14)] had a significant effect on both the maximum predicted concentrations in groundwater and the time to achieve those levels (Figure 7-15). As in the case of bacteria (Figure 7-12), as viruses were inactivated in the source sludge, fewer viruses entered the unsaturated zone, and levels in groundwater decreased (Figure 7-16).

The effect of inactivation in groundwater was more profound than the effect of inactivation in sludge. Figure 7-17 shows that increasing inactivation rates in groundwater greatly decreased the exposure concentration at the groundwater well. An exposure concentration of 1×10^{-7} occurred when the inactivation rate in groundwater was ~ 0.2 logs/day and the retardation factor was 1.0, within a reasonable range of inactivation rates. Changes in inactivation rate in groundwater did not change the kinetics of transport to the well except to alter the concentration (Figure 7-18).

The maximum predicted virus concentrations in groundwater in the saturated zone and the well were inversely related to resuspension from soil particles. Figure 7-19 shows that as the resuspension factor SSPNDS [P(18)] increased, the maximum predicted concentrations decreased. Along with the results shown earlier (Figure 7-1), this shows the importance of



**Figure 7-13. Dependence of Virus Concentration
in the Saturated Zone and the Groundwater Well on
Depth to the Saturated Zone [P(1)]**

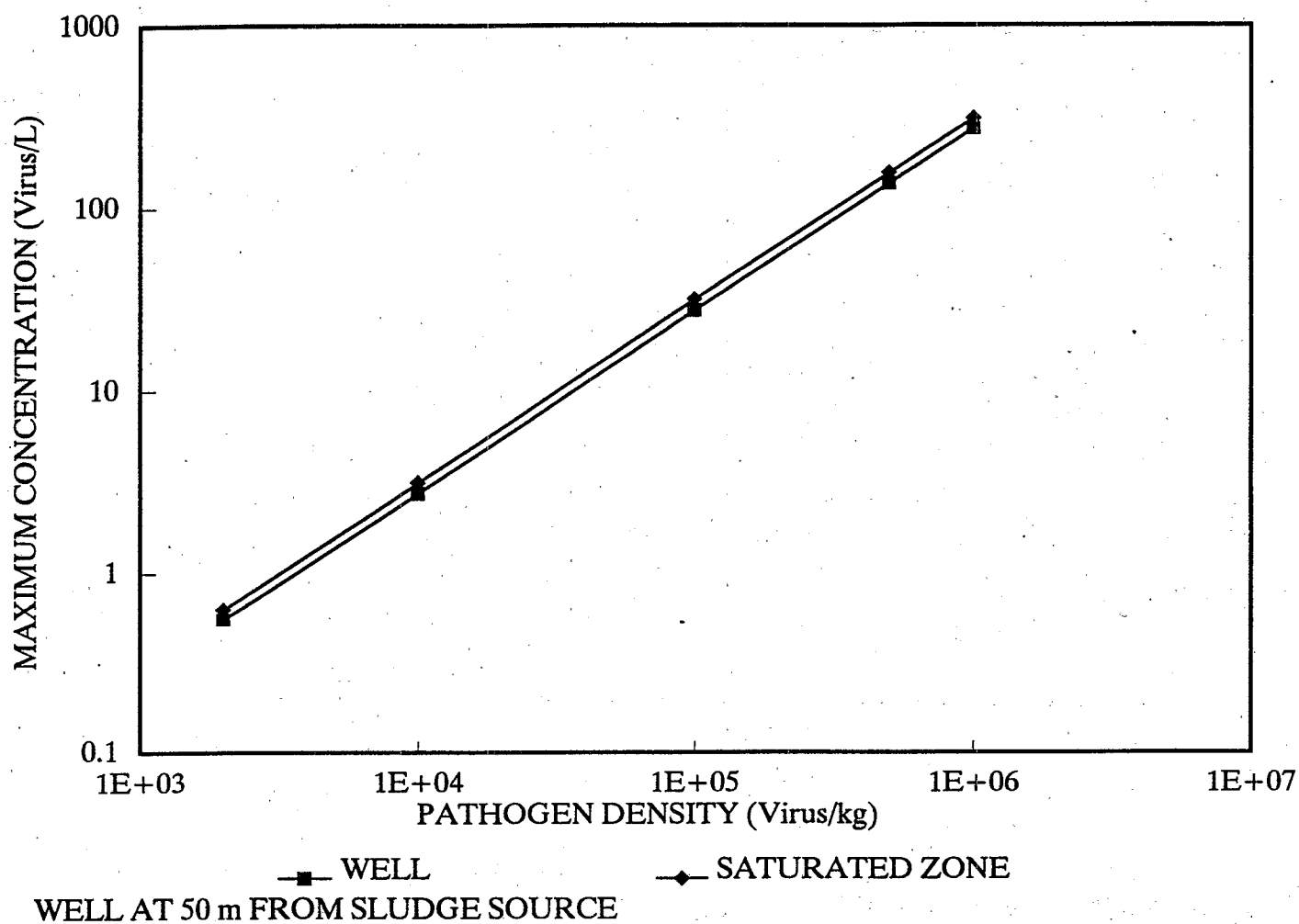


Figure 7-14. Dependence of Maximum Predicted Virus Concentration on Initial Density in Sludge [P(13)]

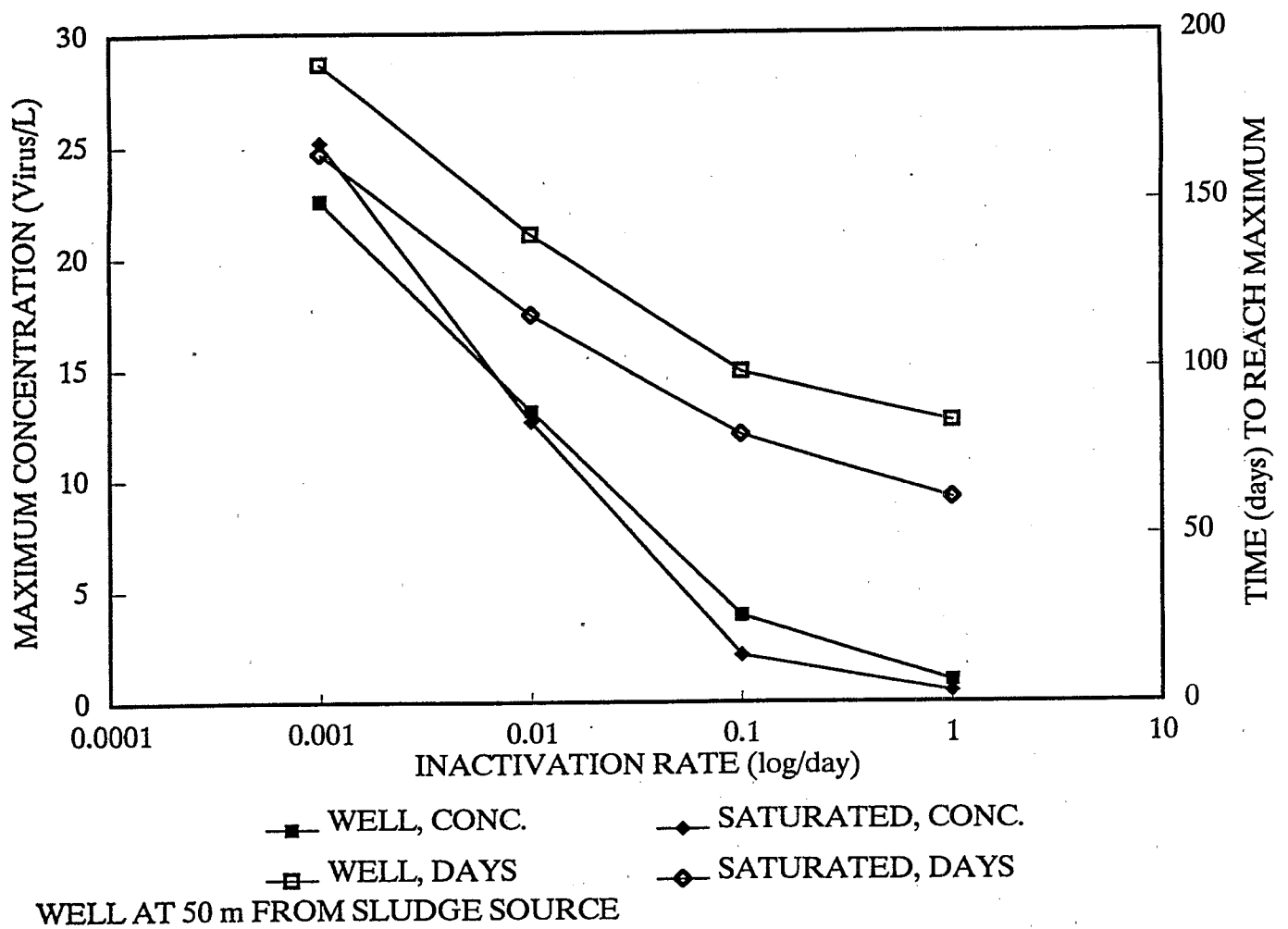


Figure 7-15. Dependence of Virus Concentration and Time to Maximum Concentration on Inactivation Rate in Sludge [P(14)]

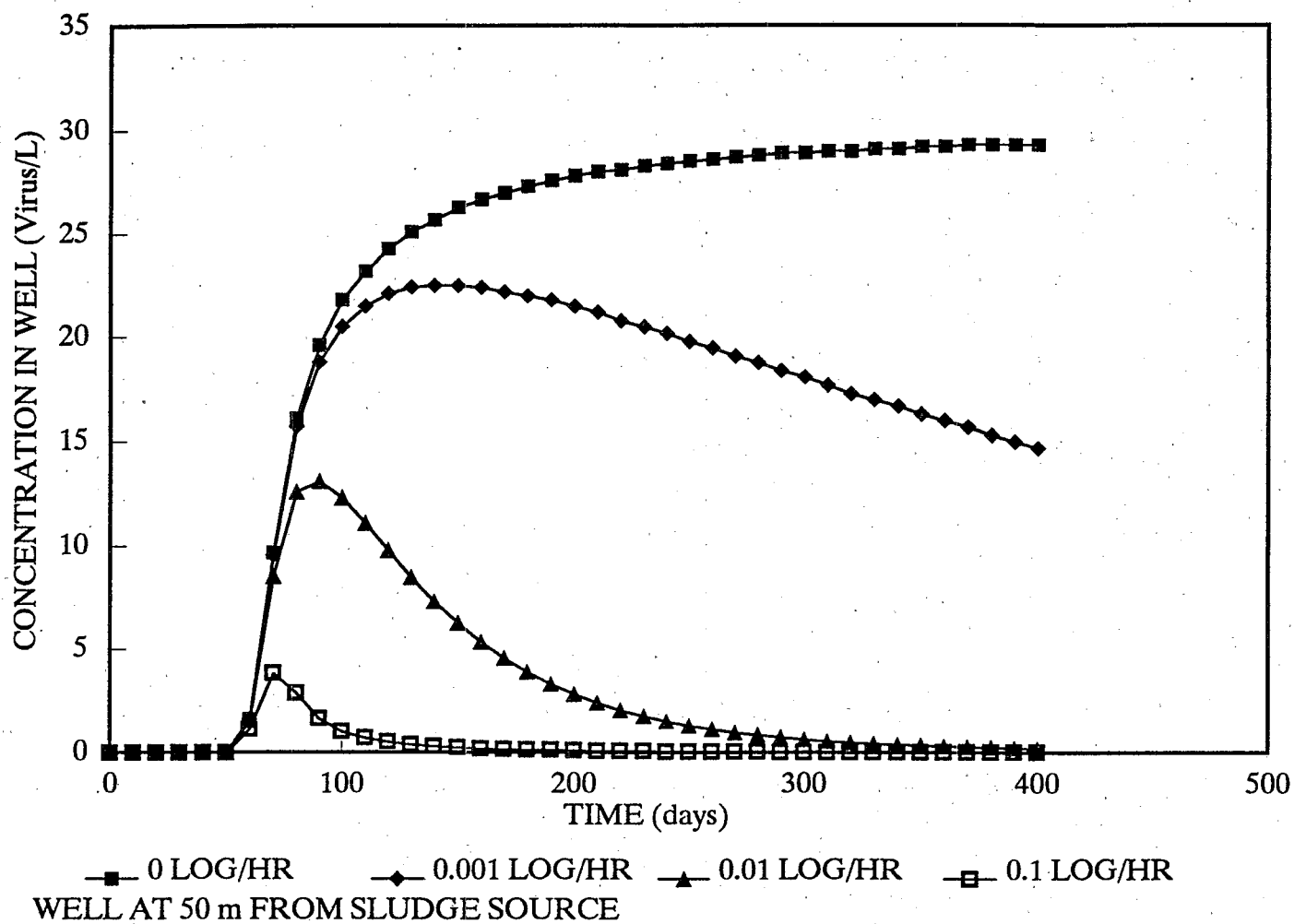


Figure 7-16. Kinetics of Virus Concentration in Groundwater as a Function of Inactivation Rate in Sludge [P(14)]

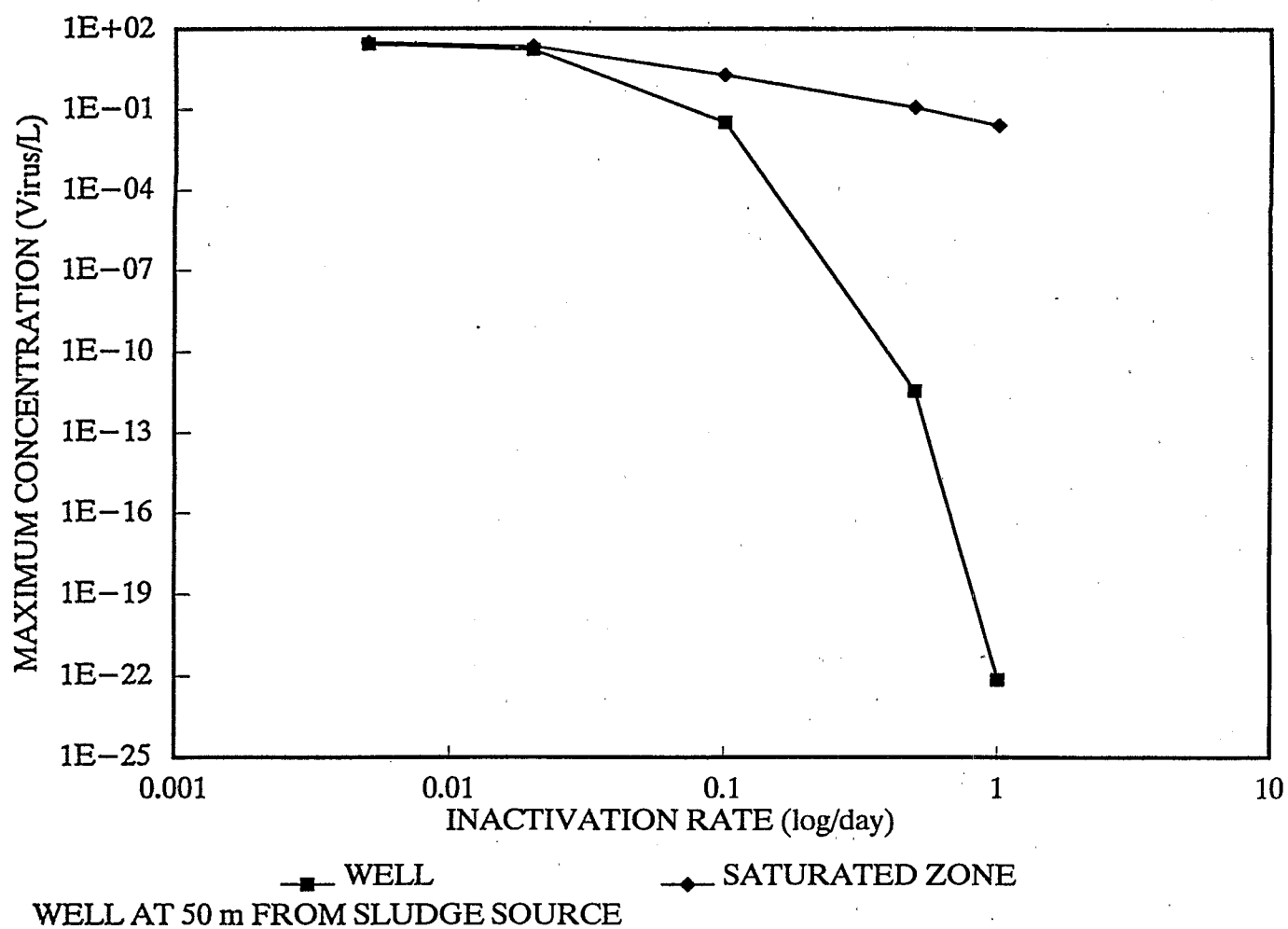
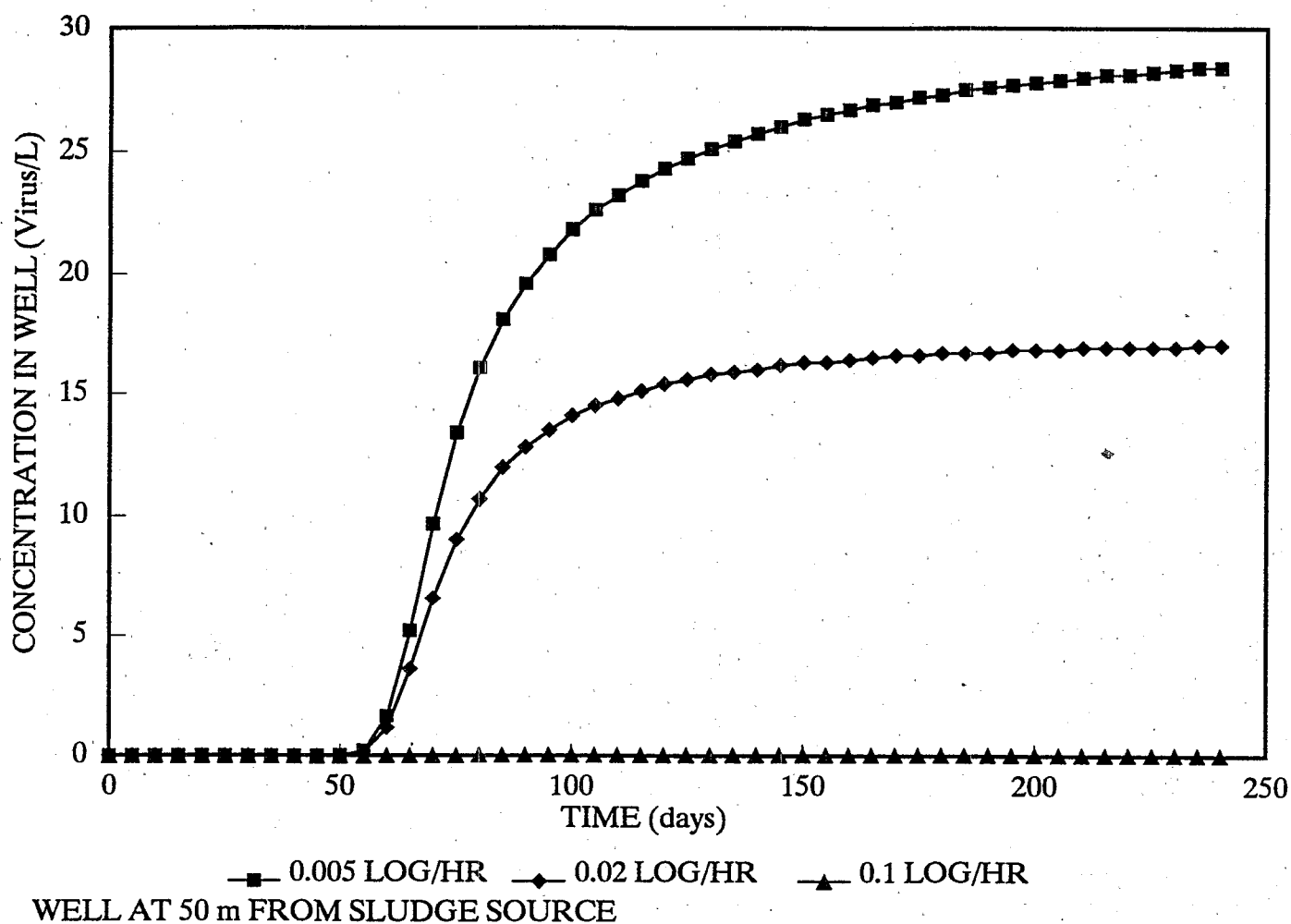


Figure 7-17. Dependence of Maximum Virus Concentration in Groundwater on Inactivation Rate in Groundwater [P(16)]



**Figure 7-18. Kinetics of Virus Concentration in Groundwater
as a Function of Inactivation Rate in Sludge [P(14)]**

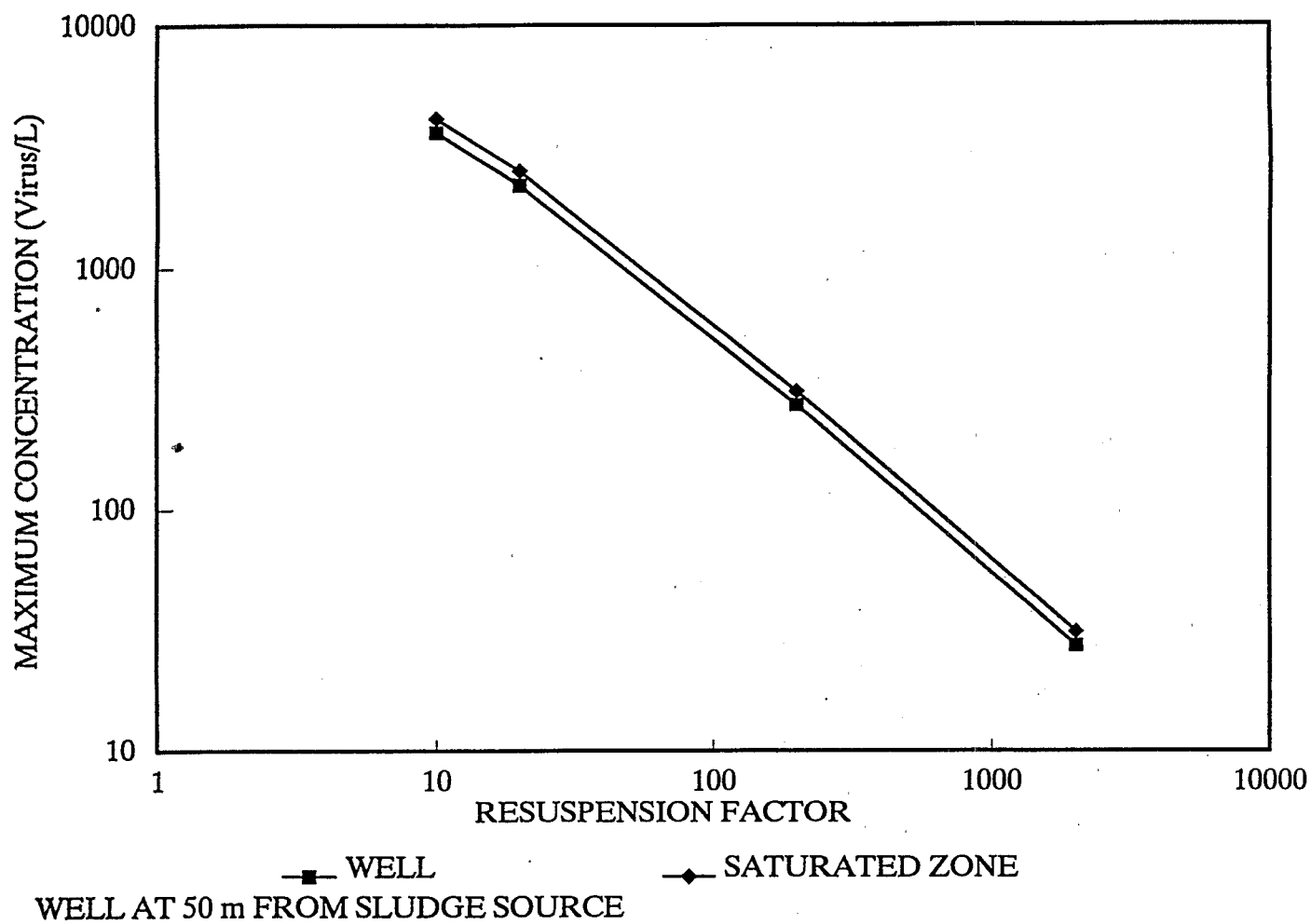


Figure 7-19. Dependence of Maximum Virus Concentration in Groundwater on Soil-to-Water Resuspension Factor [P(18)]

knowing the tightness of binding of the viruses in question to the soil particles. Published values for pathogen resuspension factors are highly variable, ranging from ~ 5 to ~ 2000 (Matson et al., 1978; Burge and Enkiri, 1978). This variability limits the accuracy of predicted groundwater transport.

Virus particles typically bind well to clay and poorly to highly organic soils (Kowal, 1985), implying that soils with a high clay content might provide a greater barrier to virus migration through the unsaturated zone than sandy or organic soils, although clay exhibits poor percolation properties. Especially in regions of low rainfall where the amount of water required to percolate out of the landfill is low, a layer of high-clay soil beneath the landfill may provide enhanced protection from contamination of groundwater.

7.2.2.5. Groundwater Transport Parameters--Some groundwater parameters were tested using the model in which SSPNDS is used to calculate retardation and dispersion. These parameters DSTAR [P(19)] and GRADI [P(22)] appear only in that model. DSTAR is used to calculate DGW, the hydrodynamic dispersion coefficient for pathogens in groundwater. It is a small number added to a term that is ~ 1 , so its value is expected to have little effect on groundwater transport. The effect of GRADI [P(22)] on rate of transport in the aquifer was tested by setting DSATZN=0 and XWELL=5 m. The time required for the virus concentration to reach $2 \times 10^{-7}/L$ was inversely proportional to GRADI: 50 days when P(22)=1, 99 days when P(22)=0.5 and 246 days when P(22)=0.2. These values extrapolate to a transport time of ~ 130 years for XWELL=50 m and GRADI=0.01.

Other groundwater parameters were tested by the model in which SSPNDS is not used to calculate retardation and dispersion. The effects of groundwater transport parameters on transport of viruses to the groundwater well were observable but relatively minor in impact. The effects of RGW and RUS are reflected in the calculated breakthrough times to the groundwater and to the well (Figures 7-1 and 7-2). Hydrodynamic dispersion in the unsaturated zone also had minor effects on the kinetics of transport and maximum predicted concentrations in groundwater. The maximum predicted concentration increased non-linearly with increasing hydrodynamic dispersion coefficient (Figure 7-20), while the time required to reach steady-state levels decreased (Figure 7-21). This effect occurs because with increasing hydrodynamic dispersion, some organisms arrive at the saturated zone, and therefore at the well, more quickly.

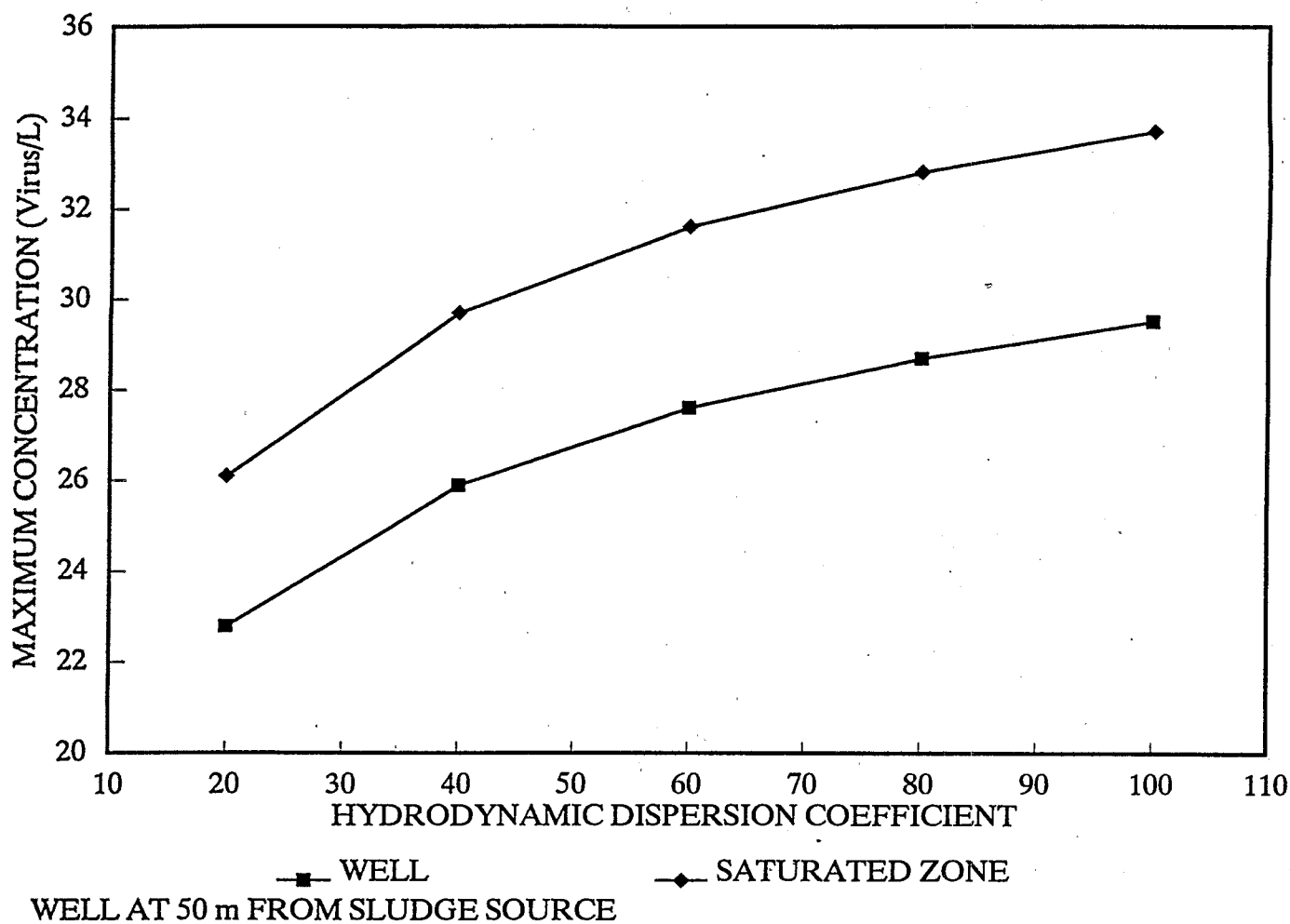


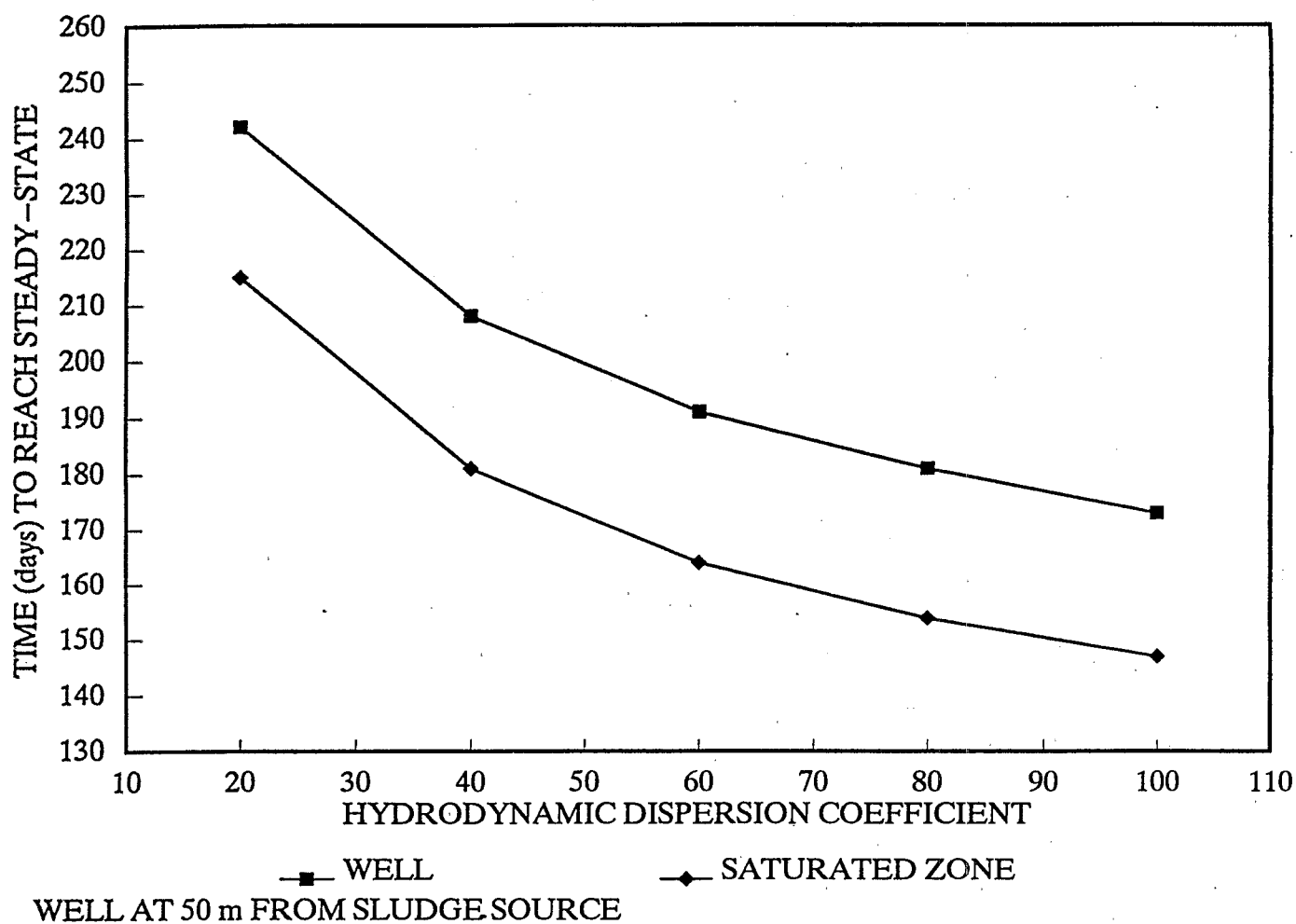
Figure 7-20. Effect of Unsaturated-Zone Hydrodynamic Dispersion on Maximum Predicted Virus Concentrations in Groundwater

Having less time for inactivation, they achieve a higher maximum concentration. Figure 7-22 demonstrates that the time at which pathogens first arrive at the saturated zone boundary is shorter and the maximum concentration is greater when DUNSAT=100 than when its value is 20.

Groundwater velocity (VGW) was an important variable at low values, but had little effect at high values. Figure 7-23 shows that the predicted virus concentrations in groundwater increased sharply as groundwater velocity increased from 0.36 to 3.6 cm/hr, and then leveled off. The time to reach steady-state levels decreased accordingly (Figure 7-24). These results reflect increased inactivation with increased travel time of the pathogens to the well.

The final groundwater parameter tested was distance to the groundwater well [P(23)]. As for bacteria, this parameter had a minor influence on maximum predicted concentrations in groundwater (Figure 7-25). However, the effect on time required to reach steady-state levels was significant (Figure 7-26). The effect of distance on concentration at the well would increase with increasing inactivation rates in groundwater, and especially with the effect of SSPNDS on retardation factors.

It is important to remember that most of the sensitivity analysis described above ignores the contribution of SSPNDS to retardation during subsurface transport. The sensitivity of the model to variations in parameters other than SSPNDS are to a large degree overwhelmed by the model's response to variations in SSPNDS. As long as it is assumed that pathogens are transported in unsaturated soil and in the aquifer by advection and dispersion rather than in cracks and solution channels, so that retardation is a significant factor, and that the binding of pathogens to soil particles is described by values of SSPNDS observed in laboratory studies, SSPNDS remains the most important parameter on which to focus additional investigation.



**Figure 7-21. Effect of Unsaturated-Zone Hydrodynamic Dispersion
on Time to Reach Steady-State Levels in Groundwater**

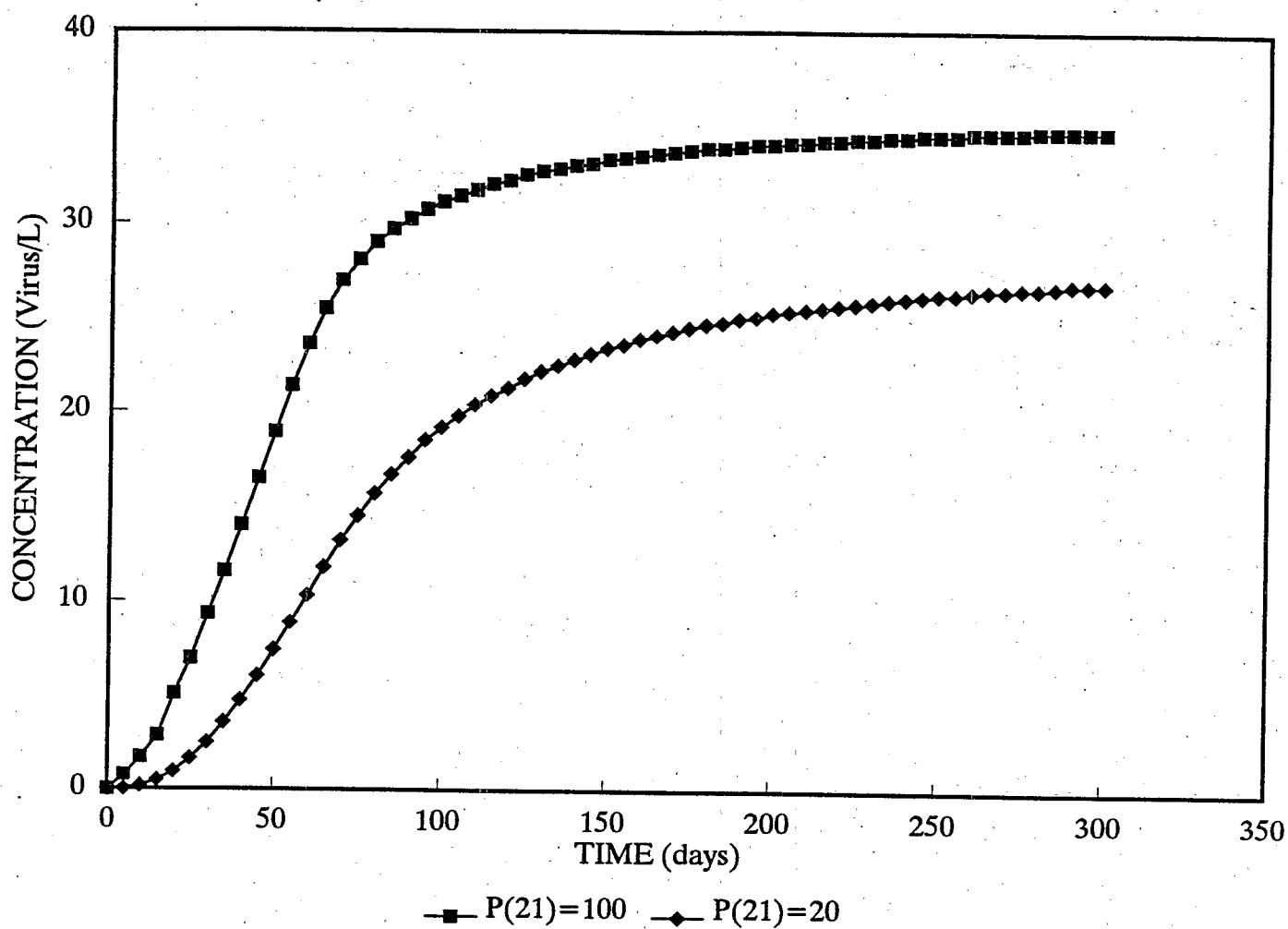
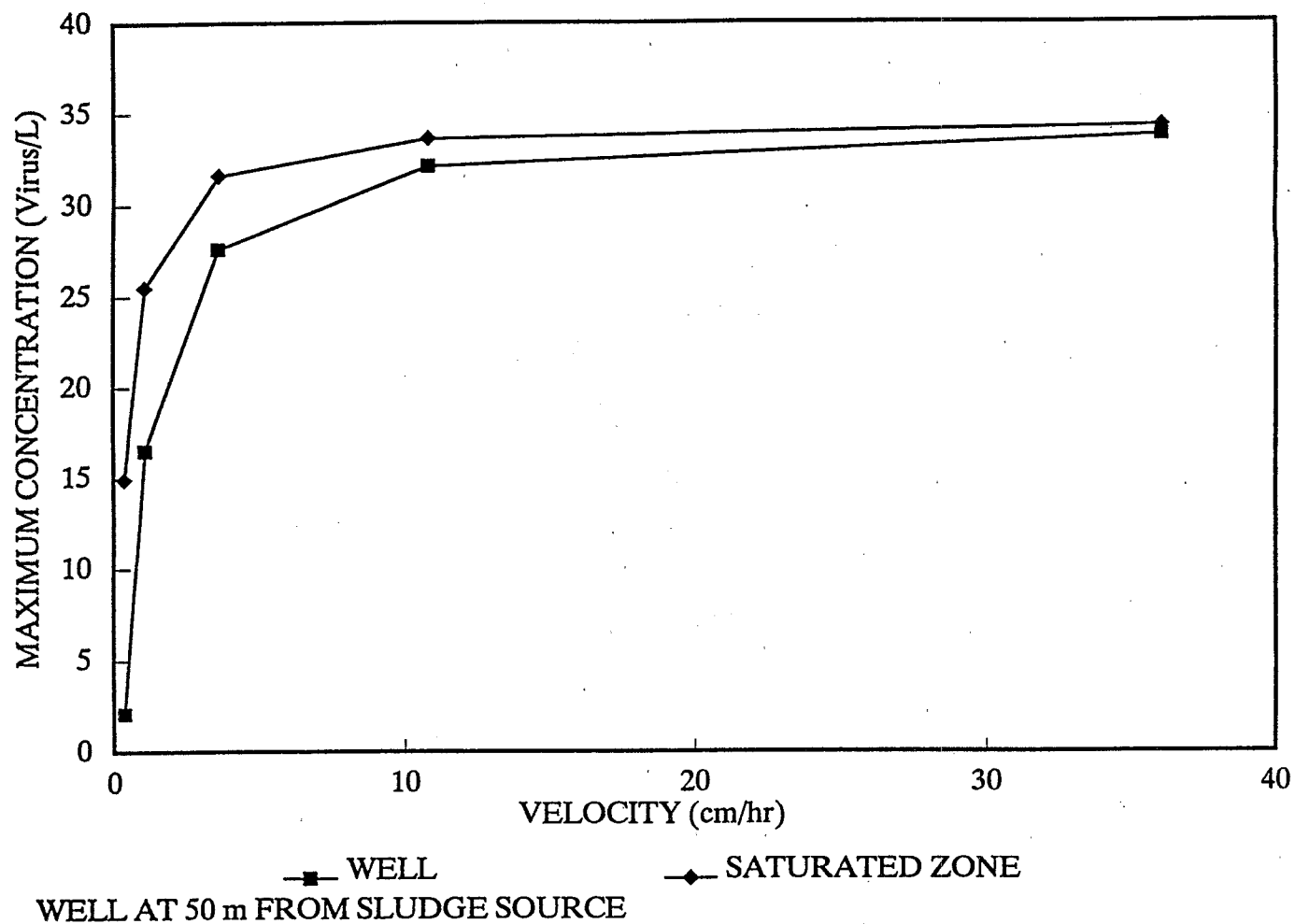
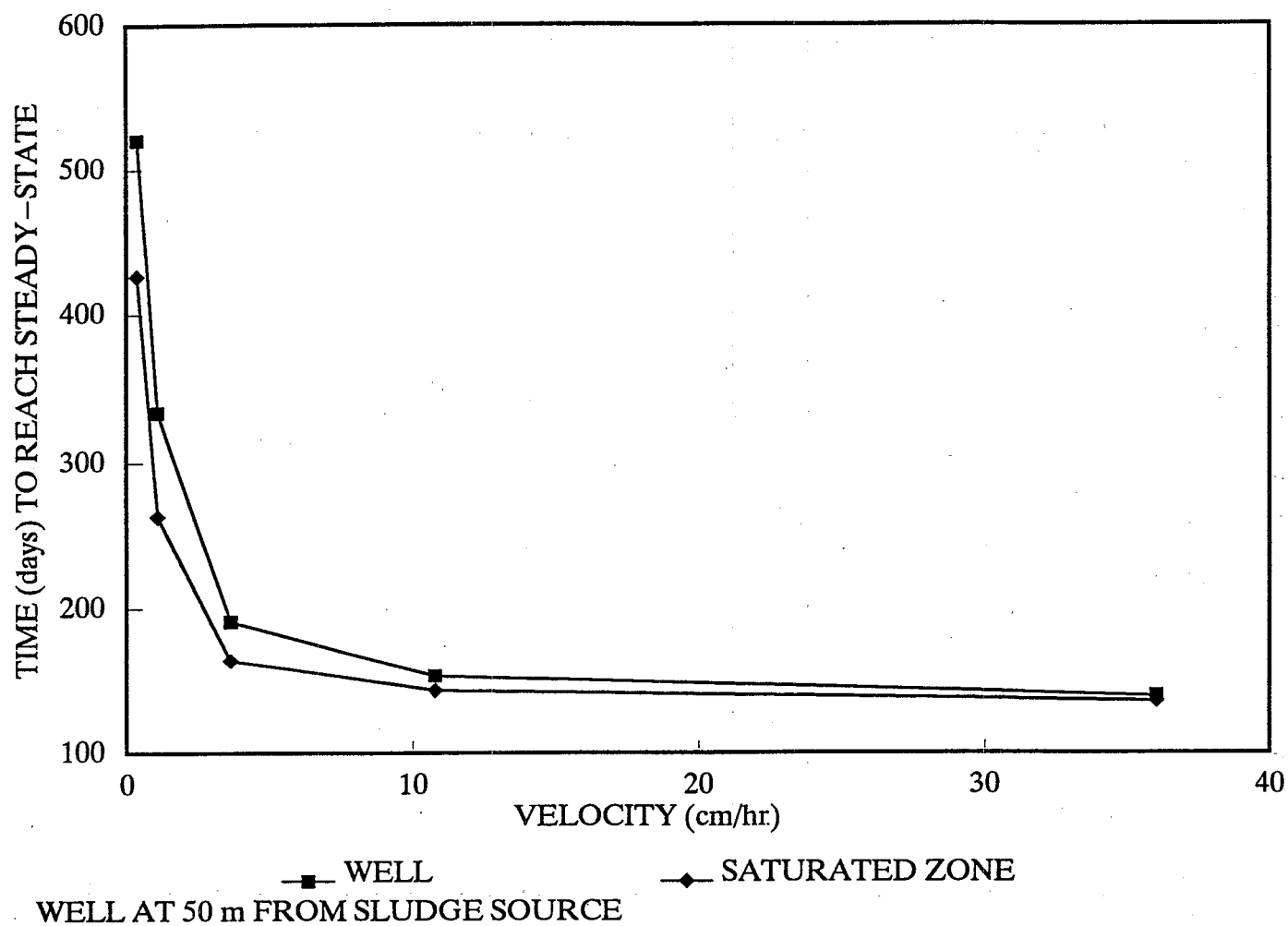


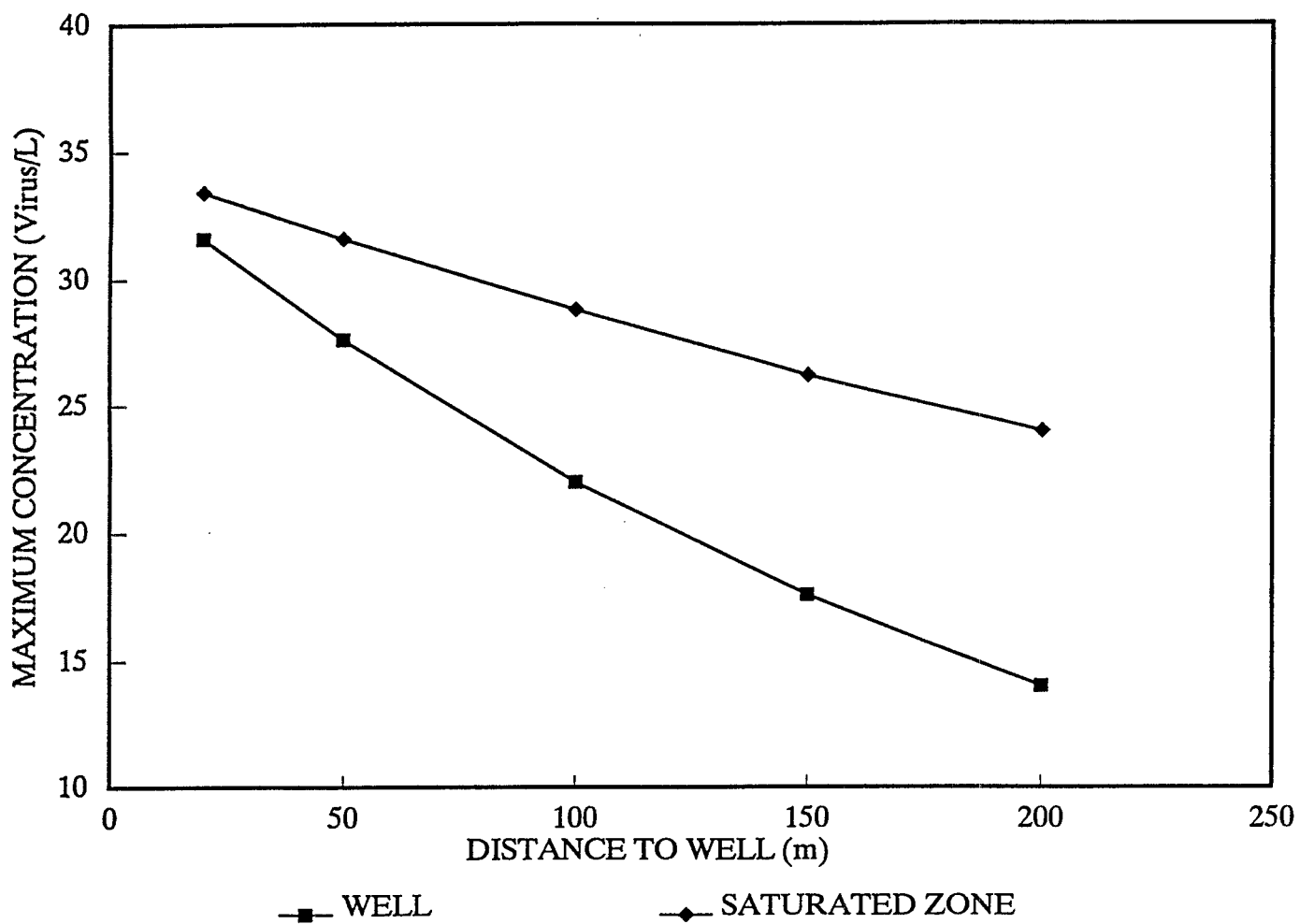
Figure 7-22. Effect of Hydrodynamic Dispersion on Time of Transfer and Concentration of Pathogens in Groundwater



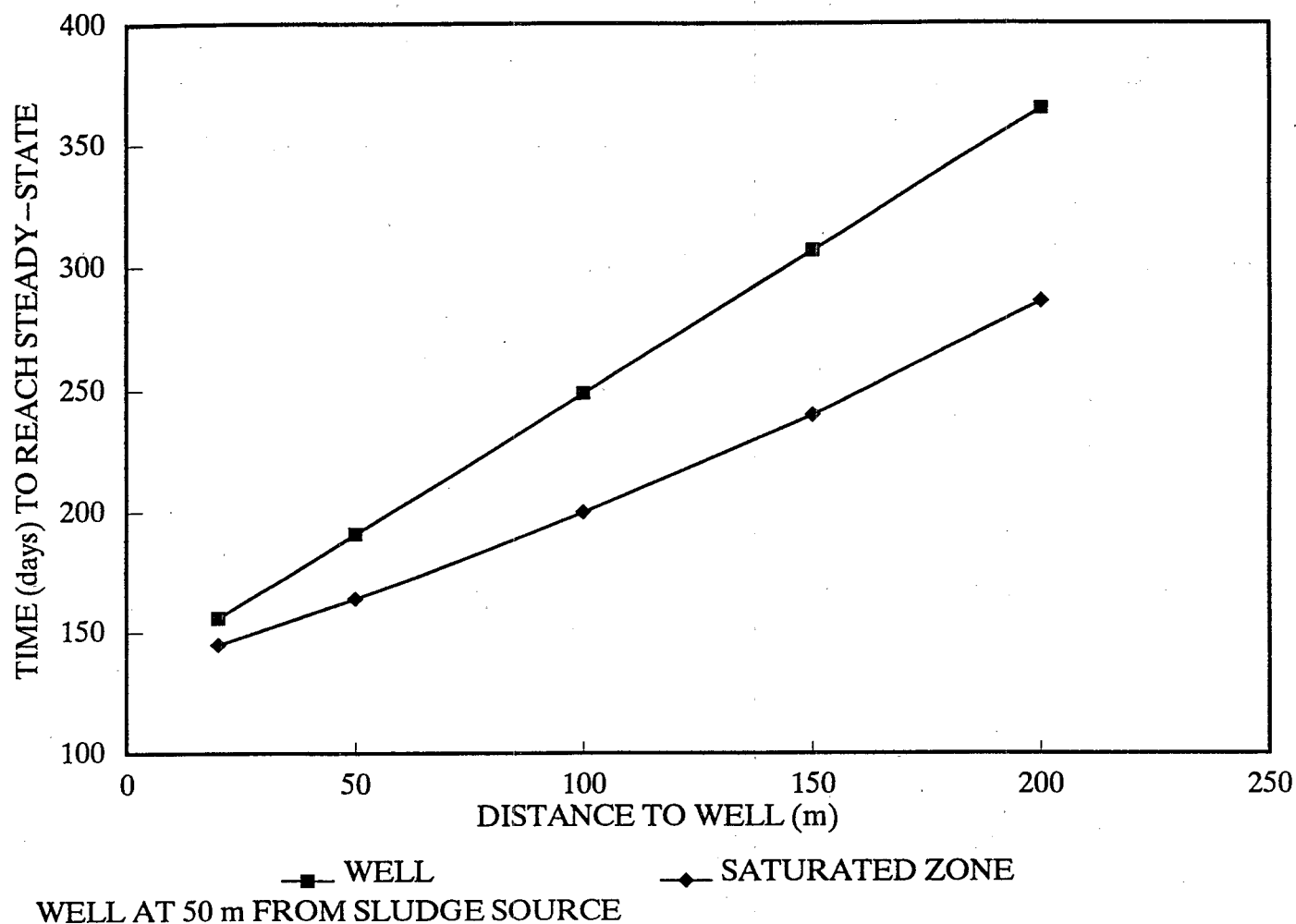
**Figure 7-23. Effect of Groundwater Velocity
on Maximum Predicted Virus Concentrations in Groundwater**



**Figure 7-24. Effect of Groundwater Velocity
on Time to Reach Steady-State Levels in Groundwater**



**Figure 7-25. Effect of Distance to the Groundwater Well [P(23)]
on Maximum Predicted Virus Concentrations in Groundwater**



**Figure 7-26. Effect of Distance to the Groundwater Well [P(23)]
on Time to Reach Steady-State Levels in Groundwater**

8. SUMMARY AND CONCLUSIONS

The SLDGFILL model for pathogen risk assessment has been run with many combinations of input parameters to simulate the transport of sludge pathogens from a disposal site to a nearby drinking-water well and the subsequent risk of infection to humans who drink from the well. Conservative exposure assumptions included a drinking water consumption rate of 2 L/day. Projections by the model show that the risk of infection by bacterial or viral pathogens is not significant at 50 m from the sludge source, even if the sludge trench or lagoon is essentially in contact with groundwater. In contrast, using average values from available data on virus transport and inactivation rates, it must be concluded that viruses present a potentially significant health hazard to consumers of well water downgradient from a sludge disposal site.

Sources of uncertainty in the model results have been discussed in Chapters 6 and 7. Uncertainties result from both the model structure and the values of the input parameters. Structural uncertainty remains because the equations that represent the model structure may not include all of the important processes or may not represent them accurately. In particular, the assumption that pathogens are transported by advection and dispersion has not been validated. Uncertainties that result from the model structure are difficult to quantify until the model results are compared with actual field measurements. Structural uncertainty can be minimized by applying the model only to the situations for which it was designed. For example, SLDGFILL should be used to predict average concentrations of pathogens in groundwater over a year. It is not designed to represent short-term changes during storm events.

Parameter uncertainty results from parameters that cannot be easily measured in the field and parameters that vary spatially and temporally. The effects of parameter uncertainty have been quantified in the sensitivity analysis in Chapter 7. The parameter uncertainty that will have the greatest effect on the results at a site depends on the potential parameter ranges for that site. A site-specific sensitivity analysis may be used to evaluate the benefit of making additional measurements to narrow parameter ranges. The resuspension coefficient SSPNDS had the most dramatic effect on pathogen transport. The effects of other parameters were tested by using a model in which SSPNDS was not used to calculate the retardation and hydrodynamic dispersion coefficients. Based on the ranges used for that sensitivity analysis, the pathogen density in the

sludge (PATHDN) and the sludge/water resuspension coefficient (SSPNDB) affected pathogen concentration in the groundwater by several orders of magnitude more than any other parameters. Other parameters that had a large effect on pathogen concentration were the depth to the saturated zone (DSATZN), the pathogen inactivation rates in water and soil (INACTB, and INACTW), and the distance of the sampling point from the disposal site (XWELL). Defining these parameter values will have the most effect on minimizing uncertainty of the results. An important question to be answered by field data is the extent to which pathogens are transported through cracks and solution channels rather than by advection and dispersion.

The model can be used to highlight aspects of sludge treatment and disposal and aspects of pathogen survival and transport under field conditions that need further study. With further validation, it could also be used to show the sludge pathogen concentration limits and well setback distances that may be necessary to ensure adequate groundwater quality to protect human health. U.S. EPA (1992) has proposed a maximum annual probability of infection by enteric pathogens of 1×10^{-4} as a limit to regulate the quality of groundwater used for human consumption. A corresponding limit on rotavirus concentration of $2 \times 10^{-7}/L$ has been derived by Regli et al. (1991). By iterative operation, the SLDGFILL model could be used to determine similar sludge concentration limits for any pathogen whose minimum infective dose is known. From that information, the well setback distances required to attain those limits could also be derived.

The dose response is characteristic of each pathogen, and alpha and beta parameters to describe it should be determined as each pathogen is identified and described. However, data on infective doses are scarce, making further research necessary for reliable use of the model to predict health risks. It is likely that viruses will present a greater health risk because they are expected to have a lower minimum infective dose.

Parasites are not expected to migrate from the bottom of the sludge trench or lagoon because their ova and cysts are too large to be transported through unsaturated soil. Transport of parasite ova and cysts through cracks and solution channels is possible, but it cannot be described by models based on advection and dispersion, because they typically assume that the soil is homogeneous.

Other than SSPNDS, after viruses reach the aquifer the most important parameter for virus concentration in the groundwater well is the inactivation rate in water [INACTW, P(16)]. At the extrapolated time required to reach the well (~ 128 years for XWELL = 50 m), all viruses would be inactivated. Changing the distance to the well changes the time over which inactivation operates to reduce the concentration of infective virus particles. Inactivation rates under field conditions are not well understood, so predictions cannot be made accurately by modeling. However, it appears that if the setback distance to the well is chosen with an eye to both groundwater velocity and inactivation rate, it should be possible to maintain the virus concentration in drinking water below the target value of $2 \times 10^{-7}/L$.

The modeled concentration of pathogens in the unsaturated zone responds proportionately to PATHDN, the concentration in the sludge. To some degree, exposure can be controlled by limiting the concentration in sludge accepted for landfilling or surface disposal. However, the concentration of viruses in well water calculated by using default parameters is at least ten orders of magnitude below the concentration ($2 \times 10^{-7}/L$) proposed by Regli et al. (1991). Therefore, the concentration of pathogens in the sludge should not be a concern for human health. An important implication of the test results is that the total number of sludge pathogens in the landfill trench or surface lagoon is less important to the pathogen concentration in the soil than is the concentration of pathogens in the sludge. Therefore, there would be no risk-based limit to the depth of sludge in the disposal units. The strong dependence on SSPNDB suggests that if resuspension of sludge pathogens into the water phase is high, a mixed landfill containing a material with a lower SSPNDB (such as clay) would reduce transport offsite. Because the default condition that pathogens will not be inactivated while they are in the sludge layer is unlikely, any treatment that delays the leaching of pathogens to groundwater will decrease both the magnitude and the duration of risk to groundwater consumers.

Several parameters were of secondary importance to model outcome. They were the depth of unsaturated soil, or depth to the saturated zone, [DSATZN, P(1)], pathogen inactivation rates in bulk sludge [INACTB, P(13)], hydraulic gradient [GRADI, P(22)] and distance from the sludge trench or lagoon to the groundwater well [XWELL, P(23)]. Of these, DSATZN [P(1)] and XWELL [P(23)] should be known accurately at any site, and GRADI [P(22)] can be measured. The uncertainty about values of the dominant parameters PATHDN [P(13)],

SSPNDB [P(17)] and especially SSPNDS [P(18)] is high enough that it becomes much less important to know the parameters P(13), P(21) and P(22) accurately.

The soil-to-water resuspension factor SSPNDS [P(18)] includes effects of adsorption/desorption to soil in the unsaturated and saturated zones, so no additional factors for retardation should be necessary. No factor is included for filtration by soil structure. Filtration is assumed to be the dominant effect for protozoa. It should be more significant for bacteria than for viruses. Therefore, because viruses appear the most likely to constitute any health risk, the inclusion of filtration may not be important to the model. Retardation allows more time for inactivation to reduce the number of infective viral particles, so it may be very important to determine resuspension coefficients accurately under field conditions.

9. RESEARCH NEEDS

Although significant research has provided an ever-increasing understanding of the risk from pathogens in sludge, there are still major information needs to be satisfied. For example, a major hurdle in any risk assessment is estimating exposure by a variety of routes or pathways to a population that varies according to activity patterns. The use of a conservatively defined human receptor is based, at least in part, on the difficulty in estimating exposure of a population to a changing level, or dose, of pathogens. Information on infective dose for most pathogens is limited. Infective doses vary greatly among pathogens, among populations of pathogens with different histories and among populations exposed to a given pathogen. Infective doses are extremely important to model outcome and need to be predicted accurately. The limited set of beta-Poisson parameters will probably be expanded in the near future. However, the large number of potentially pathogenic strains that may be found in sludge makes it impractical to characterize dose responses for all of them. Instead, a few indicator strains should be chosen that are widely prevalent, resistant to inactivation and highly infective and that have a significant impact to health. Dose responses of these indicators should be determined. In addition, rapid and reliable quantitative assays for the indicator strains in sludge should be developed and uniformly applied among sewage treatment operators. Protecting against these "worst-case" indicators would at the same time protect against less hardy or less infectious pathogens.

There is no accurate quantification of distribution of pathogens in soil or groundwater. This model assumes random distribution of pathogens in environmental media, but data are not available to verify this assumption. Further research on pathogen exposure pathways and infectious dose levels would facilitate the predictive accuracy of this model and its successors.

Another obvious data gap, illustrated by this methodology and model development, is the degree of survival and transport of pathogens in the environment. Inactivation rates under field conditions are not known with much accuracy. Information on the fate of pathogens in landfilled or surface-disposed sludge, subsurface soil and groundwater is extremely limited. The concentration and survival rates of pathogens leaching through soil into groundwater are unavailable for viruses, protozoa and helminths, and bacterial concentration data are few. More data are needed concerning the transport of pathogens through sludge, particularly as transport

rate is related to percentage solids in the sludge and to climatic factors. There is not yet sufficient information about the relationships of the many interacting factors to allow reliable predictions of pathogen resuspension from sludge and soil, or solids-to-water suspension factors. Groundwater transport parameters such as retardation coefficients for pathogens and hydrodynamic dispersion coefficients are not known under field conditions. More research should be done to find reliable ways to determine the leaching characteristics of sludge-bound pathogens. Likewise, data are needed on pathogen transport through the unsaturated zone, including quantification of subsurface transport rates of pathogens as a function of soil type and percentage of organic matter, soil chemistry, climatic factors, etc.

This model should be laboratory- and field-verified before using it for purposes other than research and development. Input of actual field data, should it become available, would reveal other research needs and would indicate any needed additional development and refinement of the model. When field data on pathogen survival and transport become available, the many different models for subsurface transport can be compared to determine which model features are the most important, which ones provide sufficient accuracy and which ones need further refinement.

In summary, future research should be oriented toward satisfying the following information needs to allow for more realistic modeling of human health risk from pathogens from landfilled and surface-disposed municipal sludge:

- field data on subsurface transport, in both the saturated and unsaturated zones, of bacteria and viruses;
- inactivation rates of pathogens under field conditions in sludge, soil and water;
- solids-to-water suspension factors applicable to sludge- and soil-bound pathogens;
- leaching characteristics of sludge-bound pathogens;
- interaction of factors affecting pathogen resuspension from sludge and soil; and
- parameters needed to describe infective doses of selected indicator species and strains of pathogens in sludge.

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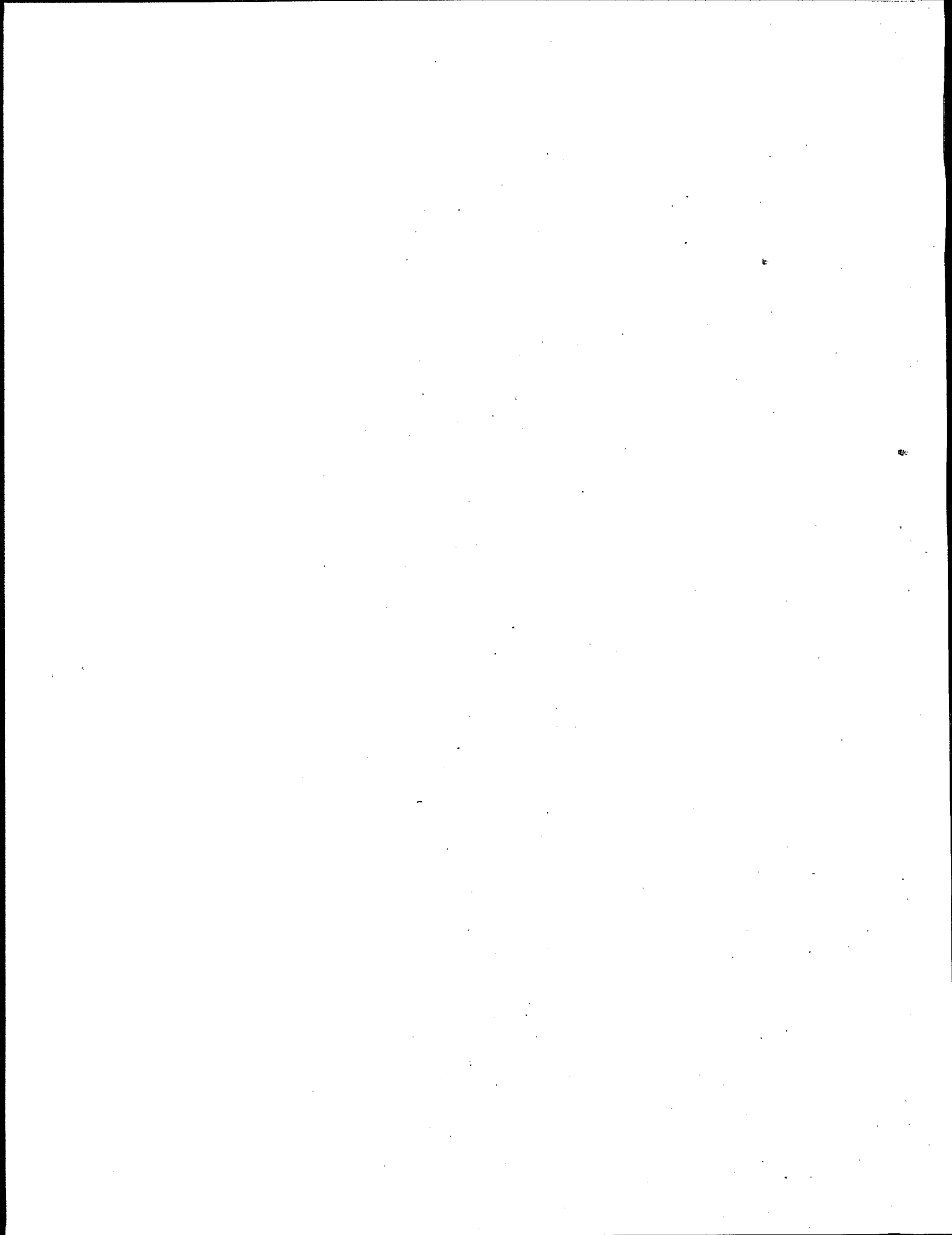
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APPENDIX A
USER'S MANUAL FOR THE PATHOGEN RISK ASSESSMENT MODEL FOR
MUNICIPAL SEWAGE SLUDGE LANDFILLING AND SURFACE DISPOSAL
(SLDGFILL)

INTRODUCTION. The purpose of this appendix is to provide directions for the use of the SLDGFILL model, which has been described in the body of this document. Before you begin, you should have a floppy disk containing SLDGFILL.EXE. To run the model, simply follow the steps outlined below.

A.1. MODEL OVERVIEW. This model evaluates the potential risk to humans from pathogenic microorganisms following surface disposal or landfilling of municipal sewage sludge. Surface disposal includes land application of dewatered sludge to dedicated non-agricultural land or long-term storage or disposal of liquid sludge in surface impoundments or lagoons. Landfilling is the disposal of dewatered sludges ($\geq 15\%$ solids) in sludge-only landfills (monofills) that use trench, area fill, or diked containment methods. The methodology and computer model deal only with the groundwater pathway. The other exposure routes are less significant or better regulated through good sludge management practices. The receptor is a person ingesting water directly from a drinking water well near the site.

This pathogen risk assessment model, SLDGFILL, is a compartment-vector model with four compartments: bulk sludge, unsaturated soil, saturated soil, and groundwater well. The model begins with a trench filled with dewatered sludge as the worst case for the source term. The number of organisms in each compartment is calculated for a column with square cross-section, 1 m on each side and the entire depth of the column. The number of organisms may increase or decrease by transfer from one compartment to another or they may decrease by inactivation. Transfers are assumed to be unidirectional, from sludge to the well from which the exposed individual drinks.

The program begins with a short initial sequence that calls a subroutine to enter operating parameters. Default values of these parameters are contained in BLOCK DATA statements, but any parameter can be changed from the keyboard at menu prompts or from an input data file.

This subroutine is read only once during each model run. Parameters are then converted to consistent units, and the initial compartment loadings are calculated. The subsurface transport subroutines for unsaturated soil and saturated soil are then initialized. Following the model run, the model will display a summary table showing daily calculations made of the probability of infection and of pathogen concentrations in each subsurface compartment. The output file, which the user specified during the initial phase of parameter selection, can be read with a text editor or word processor.

A.2. SOFTWARE AND HARDWARE REQUIREMENTS. This program requires a personal computer installed with MS-DOS. It does not require a math coprocessor, but it will run significantly faster if one is present. The CONFIG.SYS file must be written to allow at least 20 open files, i.e. CONFIG.SYS must contain the line FILES=20. The CONFIG.SYS file must also contain the line DEVICE=ANSI.SYS, DEVICE=VANSI.SYS, or an equivalent statement for an ANSI device driver.

A.3. HOW TO RUN THE MODEL. The SLDGFILL model is provided to the user on a floppy disk. Although the model can be run using the floppy disk only, it is recommended that the SLDGFILL file be copied to a hard disk, if available, before the model run. Output files also should be saved to the hard disk, if possible. (Important: Any existing data files that will be retrieved for the current model run should be copied to the same disk drive as SLDGFILL before starting the model).

To start the SLDGFILL model, the user will retrieve the file from the hard disk or load the program diskette into one of the PC floppy drives. Then the user will enter the drive name and the model name, for example:

C:SLDGFILL

This will load the model into the computer and the following screen will appear:

**PATHOGEN RISK ASSESSMENT MODEL FOR
MUNICIPAL SLUDGE LANDFILLING OR SURFACE DISPOSAL**

**VERSION 2
U.S. EPA, 1994**

**DO YOU WANT TO ENTER PARAMETER VALUES FROM
THE KEYBOARD OR FROM A FILE?
(ENTER "K" OR "F")**

A.4. PRELIMINARY DECISIONS ABOUT THE MODEL RUN. The model will ask the user a series of preliminary questions about the model run. Each of the user's keyed-in responses appears on the screen on the left margin beneath the question. Then each response is entered by pressing the "enter" key. Once a response has been entered the screen will "scroll" to the next question. (Warning: It is not possible to scroll backwards to a previous question once a response has been entered. However, the output file can be saved for future editing - see Section A.6.)

A.4.1. Selecting a File for Parameter Values. The model asks the user if parameter values will be entered from the keyboard or an existing file. If the user enters "F" for FILE, the following message will appear:

**ENTER THE NAME OF THE INPUT FILE.
(8 CHARACTER MAXIMUM)
(.IN SUFFIX IS ASSUMED)**

If the user enters "K" for KEYBOARD entry of parameter values, the model will ask for a name for the output file. In this example the user entered the name "TEST" for the output file.

**ENTER A NAME FOR THE OUTPUT FILE.
(YOU MAY USE UP TO 8 CHARACTERS)
(THE SUFFIX .OUT WILL BE APPENDED TO THE NAME)**

TEST

Once the existing file is accessed or a new file is created, the model will report the name of the file to be used for parameter selection:

**OUTPUT FILES
PARAMETER AND RESULTS SUMMARY: TEST.OUT**

The user is then asked to select either landfilling or surface disposal as the model option.

**SELECT MODEL OPTION:
1 = LANDFILLING
2 = SURFACE DISPOSAL**

1

At this point, the user may review and edit the parameter values.

DO YOU WANT TO VIEW OR MODIFY PARAMETERS?

Y

If the user enters "N" , indicating that no parameter review is needed, the model run will be initiated, as shown in Section A.6. In most cases, the user will answer "Y" so that parameters can be viewed and modified if necessary. The following screen will appear:

***** SLUDGE MONOFILL PATHOGEN MODEL *****

**ENTER VALUES FOR THE FOLLOWING
(PRESS RETURN AFTER EACH):**

1. END TIME OF PRACTICE IN DAYS

300

2. FREQUENCY FOR PRINTING RESULTS IN DAYS

1

A.4.2. End Time of Practice and Frequency of Printing. The "end time of practice" refers to the total number of days for which the model will simulate transport following introduction of the sludge to the landfill or surface disposal site. The SLDGFILL model can simulate pathogen transport to other subsurface compartments for periods as short as one day and as long as 2000 days, or about 5.5 years.

Frequency of printing results refers to the reporting intervals for the model output. Usually, the user will request the results to be reported in daily intervals (response = 1).

A.4.3. Selection of Pathogen Type. The user now is asked to specify one category of pathogen for the model run. The answer to this question determines the default values for pathogen-specific parameters that will appear later in the program. In this example, the user selected number 3, enterovirus, a viral pathogen.

PROVIDE PATHOGEN TYPE

YOUR CHOICES ARE:

- | | |
|----------------------|-------------------|
| 1 SALMONELLA | (BACTERIA) |
| 2 ASCARIS | (PARASITE) |
| 3 ENTEROVIRUS | (VIRUS) |

3

A.4.4. Pathogen Loadings from Other Sources. The initial pathogen loadings to the unsaturated zone and saturated zone are presumed to be zero (0). This scenario could be modified if there are sources of pathogen contamination other than the sludge disposal site. The

initial pathogen loadings of the unsaturated and saturated soils are referred to as POPL(2) and POPL(3). Loading data are expressed as PATHOGENS/KG DRY WT and are limited to the same category of pathogen as previously selected for the model run (in Section A.4.3).

INITIAL PATHOGEN LOADINGS

2: UNSATURATED ZONE	.00000	(POPL(2)- PATHOGENS/KG DRY WT)
3: SATURATED ZONE	.00000	(POPL(3)- PATHOGENS/KG DRY WT)

**TO CHANGE INITIAL PATHOGEN LOADINGS ENTER THE
COMPARTMENT NUMBER.
(ENTER 0 TO ACCEPT THE CURRENT VALUES.)**

0

In this example, only sources of viral pathogens were considered. (Important: POPL(2) and POPL(3) are not parameters that are saved to the output file; any modifications of these values must be re-entered for each model run). See Section 6.4.2 for a discussion of POPL(2) and Section 6.4.3 for POPL(3).

A.5. SELECTING VALUES FOR KEY PARAMETERS. The next series of screens allows the user to view and modify the 23 key parameters of the SLDGFILL model. These parameters are organized under the following headings: physical, pathogen-specific, and groundwater transport.

A.5.1. Physical Parameters. The first set of parameters presented are physical parameters. The physical properties of the sluge and of the site's underlying soil layers are addressed under this heading. Default values appear on the screen for each parameter. (Note: If an existing file had been retrieved, the file values would appear instead of the default values.)

The model allows the user to edit any or all parameter values. Parameter definitions and ranges of probable values are presented at the end of Appendix A in Table A-1. The values within a heading can be edited and re-edited in any order. However, once the user accepts a set of values by keying in "0", the model will proceed to the next set of parameters.

PHYSICAL PARAMETERS

PARAMETER	NAME	VALUE	UNITS	CHANGED
1:	DSATZN	3.5000	(M)	
2:	AQUIFR	10.000	(M)	
3:	PORWTR	0.32000	(FRACTION)	
4:	ANRAIN	150.00	(CM)	
5:	EVAP	0.50000	(FRACTION)	
6:	WCSAT	0.43700	(FRACTION)	
7:	USATCND	6.40000E-07	(M/S)	
8:	GSATCND	5.80000E-05	(M/S)	
9:	DEPTH	3.50000	(M)	
10:	SOLIDS	0.17000	(FRACTION)	
11:	BLKDEN	1.38000	(G/CM3)	
12:	SMRSLP	8.52000		

TO CHANGE PHYSICAL PARAMETERS ENTER THE
PARAMETER NUMBER. (ENTER 0 TO ACCEPT ALL VALUES.)

4

If the user wishes to change the value of a physical parameter, the number of that parameter must first be entered. For example, in this model run, the user has entered "4" for the annual rainfall parameter ANRAIN P(4). The model then asks for the new value for ANRAIN.

ENTER NEW VALUE FOR PARAMETER ANRAIN IN (CM)

100

PHYSICAL PARAMETERS

PARAMETER NAME	VALUE	UNITS	CHANGED
1: DSATZN	3.5000	(M)	
2: AQUIFR	10.000	(M)	
3: PORWTR	0.32000	(FRACTION)	
4: ANRAIN	100.00	(CM)	*
5: EVAP	0.50000	(FRACTION)	
6: WCSAT	0.43700	(FRACTION)	
7: USATCND	6.40000E-07	(M/S)	
8: GSATCND	5.80000E-05	(M/S)	
9: DEPTH	3.50000	(M)	
10: SOLIDS	0.17000	(FRACTION)	
11: BLKDEN	1.38000	(G/CM3)	
12: SMRSLP	8.52000		

TO CHANGE PHYSICAL PARAMETERS ENTER THE
PARAMETER NUMBER. (ENTER 0 TO ACCEPT ALL VALUES.)

0

Table A-1 shows that the default value for ANRAIN is 150 cm and that the rationale for selecting a value is site-specific. In this example, the user selects and then enters "100", indicating an average rainfall of 100 cm/yr for the sludge disposal site. The model substitutes "100.00" under VALUE for ANRAIN. An asterisk (*) appears in the far right column indicating that a change has been made to that parameter. The same procedure would be used to edit values for an existing file.

A.5.2. Pathogen-specific Parameters. The next set of parameters presented are pathogen-specific. If possible, the user should enter a site-specific value for PATHDN, the pathogen density in the sludge, because the model results are sensitive to this parameter. A range of values specific to each type of pathogen is presented in Table A-1.

THE PATHOGEN ENTEROVIRUS WAS SELECTED.

PATHOGEN-SPECIFIC PARAMETERS

PARAMETER NAME	VALUE	UNITS	CHANGED
13: PATHDN	1.00000E+05	(NO./KG)	
14: INACTB	0.00000	(LOG10/DAY)	
15: INACTS	1.70000E-03	(LOG10/DAY)	
16: INACTW	7.50000E-03	(LOG10/DAY)	
17: SSPNDB	20.0000	(CM3/G)	
18: SSPNDS	100.000	(CM3/G)	
19: DSTAR	1.00000E-06	(CM2/SEC)	
20: INFALF	15.0000		
21: INFBET	1000.00		

**TO CHANGE PATHOGEN-SPECIFIC PARAMETERS ENTER THE
PARAMETER NUMBER. (ENTER 0 TO ACCEPT ALL VALUES.)**

0

A.5.3. Groundwater Transport Parameters. The final set of parameters are those related to movement of groundwater between the landfill and the well.

TRANSPORT MODEL PARAMETERS

PARAMETER NAME	VALUE	UNITS	CHANGED
22: GRADI	1.00000E-02		
23: XWELL	50.000	(M)	

**TO CHANGE TRANSPORT MODEL PARAMETERS ENTER THE
PARAMETER NUMBER. (ENTER 0 TO ACCEPT ALL VALUES.)**

23

ENTER NEW VALUE FOR PARAMETER XWELL IN (M)

30

The actual distance to the well from the sludge area source (XWELL) should be entered if available. In this example, XWELL was known to be 30 m and therefore parameter number "23" was selected and a new value of "30" was entered.

TRANSPORT MODEL PARAMETERS			
PARAMETER NAME	VALUE	UNITS	CHANGED
22: GRADI	1.00000E-02		
23: XWELL	30.000	(M)	*

TO CHANGE TRANSPORT MODEL PARAMETERS ENTER THE
PARAMETER NUMBER. (ENTER 0 TO ACCEPT ALL VALUES.)

0

A.6. SAVING THE MODIFIED PARAMETER VALUES AND INITIATING THE RUN.

Once the parameter review is completed, the model allows the user to create a unique descriptive header for the run.

DESCRIPTIVE HEADER FOR RUN FOLLOWS:

1:
2:
3:
4:
5:

TO REPLACE A LINE ENTER THE LINE NUMBER.
(ENTER 0 TO ACCEPT ALL LINES.)

1

In this example, the first line of the header was selected and the text was entered: "MOST PROBABLE VALUES." Other pertinent information was then entered for lines 2-4. (Note: lines do not "wrap" around; each header entry is limited to one line).

DESCRIPTIVE HEADER FOR RUN FOLLOWS:

- 1: MOST PROBABLE VALUES**
- 2: ENTEROVIRUS**
- 3: ABC LANDFILL, ANYTOWN USA**
- 4: 30 METERS FROM LANDFILL TO WELL**
- 5:**

**TO REPLACE A LINE ENTER THE LINE NUMBER.
(ENTER 0 TO ACCEPT ALL LINES.)**

0

The user can save the modified parameter values for future reference by entering "Y" for YES and by naming the parameter file. All files should be saved to a hard disk if possible, or to the same floppy diskette as SLDGFILL if there is capacity on the diskette.

**DO YOU WANT TO SAVE THE REVISED PARAMETERS?
(ENTER "Y" OR "N")**

Y

The user is asked to name the file of modified parameters. In this example, the parameter file was named "ABC-RUN1."

**ENTER A NAME FOR THE PARAMETER FILE.
(8 CHARACTER MINIMUM)
(.IN SUFFIX IS ASSUMED)**

ABC-RUN1

*******MODEL INITIALIZED-RUN STARTED*******

Once the parameters are saved and the file is named, the model run will be initiated. (Hint: If the user wishes to stop the model run, type "CTRL C". Results will not be displayed, and the run must be re-initiated.) The model will display a summary table showing daily calculations made of the probability of infection and of pathogen concentrations in each subsurface compartment.

CONCENTRATION IN COMPARTMENT					
***** (PATHOGENS/LITER) *****					
INFECTION	DAY	**PROB**	**WELL**	*SAT ZN*	UNSAT ZN *SLUDGE*
1	0.00E+00	0.00E+00	0.00E+00	4.02E+02	4.02E+03
2	0.00E+00	0.00E+00	0.00E+00	4.02E+02	4.02E+03
3	0.00E+00	0.00E+00	0.00E+00	4.02E+02	4.02E+03
4	0.00E+00	0.00E+00	0.00E+00	4.02E+02	4.02E+03
5	0.00E+00	0.00E+00	0.00E+00	4.02E+02	4.02E+03
6	0.00E+00	0.00E+00	0.00E+00	4.02E+02	4.02E+03
7	0.00E+00	0.00E+00	0.00E+00	4.02E+02	4.02E+03
8	0.00E+00	0.00E+00	0.00E+00	4.02E+02	4.02E+03
10	0.00E+00	0.00E+00	0.00E+00	4.02E+02	4.02E+03
11	0.00E+00	0.00E+00	0.00E+00	4.02E+02	4.02E+03
12	0.00E+00	0.00E+00	0.00E+00	4.02E+02	4.02E+03
13	0.00E+00	0.00E+00	0.00E+00	4.02E+02	4.02E+03

A.7. RECOVERING THE DATA. After the run has been completed, you will see the reminder,

... RUN COMPLETE

OUTPUT FILES

PARAMETER AND RESULTS SUMMARY: TEST.OUT

To view the results, the user can use a word processor, text editor, or the TYPE command using the filename provided by the user at the second prompt after invoking the model: "TYPE FILENAME.OUT". The file will then scroll up the monitor screen. The scrolling can be stopped by typing "CTRL S". It will continue if the user strikes any key.

To print the results, use the print functions of a word processor or text editor, or type "CTRL P" to activate the printer echo mode of the computer; then "TYPE FILENAME.OUT". The file should then be printed. This file contains the descriptive header supplied by the user, a summary of the parameters accepted, and the probability of infection for each day. The output file also gives the concentration of pathogens in each of the four compartments in pathogens/liter. (WARNING: Avoid specifying a font that uses

proportional spacing for the output files. Proportional spacing may result in unaligned columns.)

A.8. SAMPLE INPUT AND OUTPUT. The test.out file contains input parameters, default values and those modified by the user, for the model run. Results are also included in this file. The following pages are a portion of the test.out file that resulted from the preceding examples.

FILE test.OUT

MOST PROBABLE VALUES
 ENTEROVIRUS
 ABC LANDFILL, ANYTOWN USA
 30 METERS FROM LANDFILL TO WELL

PRACTICE STOP TIME= 300 DAYS

PRINT SAMPLING RATE (IPRNT) = 1 DAYS

PATHOGEN = 3 ENTEROVIRU

INITIAL POPULATIONS FOR COMPARTMENTS (PATHOGENS/KG DRY WGT):

1: SLUDGE = 5.0000E+04
 2: UNSATURATED ZONE = 0.0000
 3: SATURATED ZONE = 0.0000

THE FOLLOWING INPUT PARAMETERS WERE ACCEPTED:

	VARIABLE	VALUE	CHANGED BY USER	UNITS
1	DSATZN	3.500		(M)
2	AQUIFR	10.00		(M)
3	PORWTR	0.3200		(FRACTION)
4	ANRAIN	100.0	*	(CM)
5	EVAP	0.5000		(FRACTION)
6	WCSAT	0.4370		(FRACTION)
7	USATCND	6.4000E-07		(M/S)
8	GSATCND	5.8000E-05		(M/S)
9	DEPTH	3.500		(M)
10	SOLIDS	0.1700		(FRACTION)
11	BLKDN	1.380		(G/CM3)
12	SMRSLP	8.520		
13	PATHDN	1.0000E+05		(NO./KG)
14	INACTB	0.0000		(LOG10/DAY)
15	INACTS	1.7000E-03		(LOG10/DAY)
16	INACTW	7.5000E-03		(LOG10/DAY)
17	SSPNDB	20.00		(CM3/G)
18	SSPNDS	100.0		(CM3/G)
19	DSTAR	1.0000E-06		(CM2/SEC)
20	INFALF	15.00		
21	INFBET	1000.		
22	GRADI	1.0000E-02		
23	XWELL	30.00	*	(M)

*****CONCENTRATION IN COMPARTMENT*****
 INFECTION ***** (PATHOGENS/LITER) *****
 DAY **PROB** **WELL** *SAT ZN* UNSAT ZN* *SLUDGE*

[illegible]

[illegible]

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[illegible]

[illegible]

271	0.00E+00	0.00E+00	0.00E+00	3.95E+02	3.95E+03
272	0.00E+00	0.00E+00	0.00E+00	3.95E+02	3.95E+03
273	0.00E+00	0.00E+00	0.00E+00	3.95E+02	3.95E+03
274	0.00E+00	0.00E+00	0.00E+00	3.95E+02	3.95E+03
275	0.00E+00	0.00E+00	0.00E+00	3.95E+02	3.95E+03
276	0.00E+00	0.00E+00	0.00E+00	3.95E+02	3.95E+03
277	0.00E+00	0.00E+00	0.00E+00	3.95E+02	3.95E+03
278	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
279	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
280	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
281	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
282	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
283	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
284	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
285	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
286	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
287	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
288	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
289	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
290	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
291	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
292	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
293	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
294	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
295	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
296	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
297	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
298	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
299	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03
300	0.00E+00	0.00E+00	0.00E+00	3.94E+02	3.94E+03

Table A-1. Parameters for Pathogen Risk Assessment Methodology: Sludge Landfilling and Surface Disposal

Parameter	Definition	Value ^a	Description of Values
DSATZN P(1)	Depth to saturated zone (m)	Range 0-42.7; Median 21.3 ^b ; Default 3.5	Site-specific
AQUIFR P(2)	Aquifer thickness (m)	1 10 40	Site-specific Shallow, confined aquifer. Default value. Deep, extensive aquifer.
PORWTR P(3)	Volumetric moisture content, or pore water (θ , standard groundwater model term)	0.06 0.32 0.30	Site-specific Clay. Default value; medium sand aquifer. Coarse sand.
ANRAIN P(4)	Annual rainfall (cm)	150	Site-specific
EVAP P(5)	Fraction of rainfall lost by evaporation and runoff	0.5	Site-specific
WCSAT P(6)	Saturated water content of subsurface soil (fraction) ^c	Range: 0.332-0.582 0.437 0.464 0.475	Site-specific Default value; sand. Clay loam. Clay.
USATCND P(7)	Unsaturated conductivity rate (m/s)	6.4E-7	Site-specific
GSATCND P(8)	Saturated conductivity rate (m/s) of subsurface soil ^c	1.7E-7 5.8E-5 6.4E-7	Site-specific Clay. Default value; sand. Clay loam.

Table A-1. (continued)

Parameter	Definition	Value ^a	Description of Values
DEPTH P(9)	Depth of sludge (m)	Range 0.9-6.1; Median 3.5 ^b	Application-specific Default value.
SOLIDS P(10)	Fractional solids of sludge	Range .03-.30 Median .17 ^b 0.05	Application-specific Default value. Surface disposal in lagoons.
BLKDEN P(11)	Bulk density of sludge (g/cm ³)	1.3	
SMRSLP P(12)	Slope of soil moisture retention curve (dimensionless) ^c	8.52 10.40 11.40	Site-specific Default value; clay loam. Silt clay. Clay.

Table A-1. (continued)

Parameter	Definition	Value ^a	Description of Values
PATHDN P(13)	Pathogen density in the sludge (pathogens/kg dry weight)	2×10^2 5×10^3 1.1×10^4 1×10^2 5×10^4 5×10^6 2×10^3 1×10^5 1×10^6	Parasites: Low reported value; lower value expected to be insignificant. Default value. Highest reported value. Bacteria: Lower value expected to be insignificant. Default value. Above highest reported values. Virus: Lower value expected to be insignificant. Default value. Above highest reported values.
INACTB P(14)	Inactivation rate in sludge (\log_{10}/day)	0	Assumes protective effect of bulk sludge; to be changed as data allow
INACTS P(15)	Inactivation rate in moist soil (\log_{10}/day)	0.0037-0.033 0.016-6.39 0.0017-3.69	Parasites (Default 0.0037) Bacteria (Default 0.016) Viruses (Default 0.0017)

Table A-1. (continued)

Parameter	Definition	Value*	Description of Values
INACTW P(16)	Inactivation rate in water (\log_{10}/day)	0.0128	Parasites (Default 0.0128)
		0.0228-3.01	Bacteria (Default 0.0228)
		0.0039-2.383	Viruses (Default 0.0075)
SSPNDB P(17)	Sludge/water resuspension coefficient (cm^3/g)	Range 20-2000	Pathogen-specific
		200	Parasites (conservative)
		2000	Bacteria
		20	Viruses
SSPNDS P(18)	Soil/water resuspension coefficient (cm^3/g)	Range 10-1000	Pathogen-specific
		100	Parasites (conservative)
		1000	Bacteria
DSTAR P(19)	Diffusivity (cm^2/s)	1.000E-6	Viruses
INFALF P(20)	beta-Poisson alpha	0.17	Parasites
		0.33	Bacteria
		15	Viruses
INFBET P(21)	beta-Poisson beta	1.32	Parasites
		139.9	Bacteria
		1000	Viruses
GRADI P(22)	Hydraulic gradient (unitless)	0.01	Site-specific

Table A-1. (continued)

Parameter	Definition	Value ^a	Description of Values
XWELL P(23)	Distance of well from sludge area source (m)	20	Arbitrary lower value.
		50	Default value.
		150	Arbitrary upper value.

^a Values from literature were used where possible to establish ranges and default values.

^b Source: Walsh, 1978. *Process Design Manual: Municipal Sludge Landfills*.

^c Source: Superfund Exposure Assessment Manual. USEPA Office of Remedial Response EPA/540/1-88/001; OSWER 9285.5-1, April 1988.

