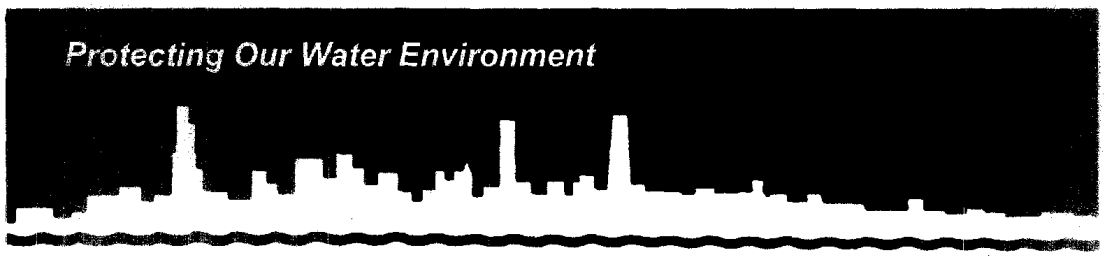

Metropolitan Water Reclamation District of Greater Chicago

RESEARCH AND DEVELOPMENT DEPARTMENT

REPORT NO. 01-1-A

PEER REVIEW OF

METROPOLITAN WATER RECLAMATION DISTRICT

OF GREATER CHICAGO'S APPLICATION FOR DESIGNATION

OF PROCESSES TO FURTHER REDUCE PATHOGENS

ADDENDUM

STATISTICAL EVALUATION OF PATHOGEN INACTIVATION FOR A CONVENTIONAL LOW-COST TECHNOLOGY CLASS A BIOSOLIDS PROCESS

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Statistical Evaluation of Pathogen Inactivation for a Conventional Low-Cost Technology Class A Biosolids Process

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ABSTRACT: Statistical methods were developed for analyzing the results of a study of pathogen densities for sludge samples taken over the four stages of the solids processing trains (SPTs) operating at the Stickney and Calumet Water Reclamation Plants of the Metropolitan Water Reclamation District of Greater Chicago, Illinois (District). These methods also apply to pathogen density studies for other biosolids processes. Analysis of covariance models were used to estimate expected pathogen densities for individual solids processing stages. Cross-validation was used to select appropriate analysis of covariance models. Nonparametric methods were used to estimate distributions for pathogen density reductions between solids processing stages and to assess the effect of hypothetical surges and expansions in initial stage pathogen densities on final stage pathogen densities. These statistical analyses demonstrate that the District's SPTs achieve target reductions in enteric virus and viable helminth ova densities with high probabilities. Furthermore, the District's SPTs would still meet U.S. Environmental Protection Agency Class A restrictions for these pathogens with high probabilities, even if the initial stage pathogen densities observed in the study undergo extreme hypothetical surges or extreme hypothetical uniform expansions, that is, exceptionally large isolated bursts of pathogens or exceptionally large sustained increases in pathogens in the feed to the SPTs. *Water Environ. Res.*, **72,** 423 (2000).

KEYWORDS: analysis of covariance, biosolids, Class A biosolids, cross-validation, helminths, $\log_{10}$ reductions, processes to further reduce pathogens, viruses.

On February 19, 1993, U.S. Environmental Protection Agency (U.S. EPA) published the 40 CFR Part 503 regulations (U.S. EPA, 1993), which included criteria for biosolids quality relative to indicator organisms and pathogen content. The criteria define biosolids to be of Class A designation when the final biosolids product before use contains less than 1000 fecal coliforms (most probable number [MPN]) per gram of dry solids, less than three *Salmonella* organisms (MPN) per 4 g of dry solids, less than one enteric virus per 4 g of dry solids, and less than one viable helminth ovum per 4 grams of dry solids.

U.S. EPA identified in the regulations five specific alternative solids processing technologies as processes to further reduce pathogens (PFRPs). A sixth alternative under which a petitioner is required to demonstrate to the U.S. EPA with review by its Pathogen Equivalency Committee (PEC) that a process or scheme is equivalent to a PFRP was also included in the regulations. The Metropolitan Water Reclamation District of Greater Chicago, Illinois (District), has elected to petition U.S. EPA to have its solids processing trains (SPTs)—that is, the complete process trains consisting of mesophilic anaerobic digestion followed by lagoon storage of anaerobically digested solids and centrifuge cake and then air drying—to be certified as PFRPs under this sixth alternative.

The regulations are clear in their requirements for Class A equivalency certification. The final biosolids product from the proposed equivalent process must satisfy Class A requirements for pathogen content levels before use. Although specific log reductions in indicator and pathogenic organisms are not required by the Part 503 regulations, U.S. EPA's PEC requested that the District demonstrate specific log reductions to establish that the District's SPTs are equivalent to PFRPs. The requested level for enteric viruses is a reduction by an amount with a $\log_{10}$ of -3 or better and for viable helminth ova by an amount with a $\log_{10}$ of -2 or better. The PEC suggests that they are concerned about sudden surges in indigenous pathogen content of raw wastewater entering the treatment facility. In addition, the PEC seems to desire that the above-indicated $\log_{10}$ reductions be demonstrated through seeding of the solids treatment process with target pathogens, particularly helminth ova, and tracking their decay through the process.

Because of the obvious problems of worker safety and public health risk and, indeed, the impracticality of seeding viable helminth ova into a full-scale system and because of the lack of similitude between the results of bench-scale seeding experiments and a full-scale system that produces 0.5 million kilograms (1 million pounds) of dry weight of biosolids per day, the District proposed an alternative approach to the PEC. This approach consists of operating the District's full-scale conventional SPTs under codified operating conditions, increasing the sensitivity of helminth ova analysis by increasing the sample size of the final air-dried biosolids product from approximately 4 to approximately 100 to 400 g, collecting numerous samples from different locations of the SPTs for pathogen analysis, and subjecting the resulting analytical data to rigid statistical analyses. See Tata et al. (2000) for further details.

Statistical methods are presented in this paper that may be used to analyze pathogen densities for any biosolids process. These methods are used to analyze the results of the District's study of its SPTs to address the following issues: (1) determination of the $\log_{10}$ reduction capability of the SPTs and (2) prediction of the effect of surges and expansions of viable helminth ova in the digester feed on the final biosolids product.

Methodology

Statistical methods were developed for analyzing the results of a study of pathogen densities for samples taken across the four stages of the District's SPTs. Analysis of covariance models were used to estimate expected pathogen densities for individual solids

processing stages. Cross-validation was used to select appropriate analysis of covariance models for computing these estimates. Nonparametric methods were used to estimate distributions of pathogen density reduction between solids processing stages and to assess the effect of hypothetical surges (exceptionally large isolated bursts) and expansions (exceptionally large sustained increases) in initial stage pathogen densities on final stage pathogen densities.

These methods apply to general pathogen studies of biosolids processes that consist of multiple stages. Specifically, pathogen densities, as determined from observed pathogen counts within samples of varying solids content, can be modeled using analysis of covariance models based on classification variables such as solids processing stage and season or on covariates such as solids content, time of the month, ammonia-nitrogen levels, solar radiation levels during the drying season, and so forth. The selection of appropriate models of this type may then be conducted using the modeling framework presented in this section. Furthermore, analysis of pathogen reduction–inactivation for the actual process and hypothetical surges and expansions in the inputs may be conducted using the nonparametric methods of this section.

Analysis of Covariance Models. Under the analysis of covariance model (Snedecor and Cochran, 1967), the expected value of a response variable, Y, conditioned on the value x of a covariate, X (or a vector of covariates), is modeled as a linear function of x with the same slope(s) for all classes, j, but with different intercepts, that is, $E(Y|X = x) = \alpha_j + \beta x$. The one-way analysis of variance model with $E(Y|X = x) = \alpha_j$ is a special case. For the purposes of this paper, the response variable may be pathogen count, C, pathogen density, D, per 4 g of solids, or some transform of either of these two quantities and is classified on the basis of the single factor, the solids processing stage j, when $1 \le j \le J$. The transforms to be considered for response variables are $Y = C^p$ and $Y = D^p$ for positive powers $p > 0$. Solids content, S, or any transform of it is the choice considered for the covariate. The transforms to be considered for the covariate are $X = S^q$ for $q \neq 0$ and $X = \ln(S)$. Thus, there are quite a few choices for the form of the observed data values (x_{ij}, y_{ij}), for $1 \le i \le n_j$, $1 \le j \le J$, to which to apply the analysis of covariance model, and each choice may be applied to the observed data for each type of pathogen.

General Modeling Framework. The analysis of covariance model and the associated one-way analysis of variance model generate an extensive family of modeling procedures corresponding to the various adjustments of experimental data that may be used in computing initial parameter estimates. A selection procedure is required to discriminate between all these modeling procedures, M, on the basis of some appropriate measure of the fidelity of generated predictions to actual values, for example, the standard deleted prediction error [SDPE(M)] defined below. The actual values, and hence their predictions, also need to be expressed in some standard form. The appropriate form for the purposes of this paper is pathogen densities per 4 g solids content so that the selection procedure reflects the definition of a Class A biosolids. Whatever form for the data (x_{ij}, y_{ij}) used in initial parameter estimation, the associated initial predictions $\hat{y}_{ij}$ may be adjusted to obtain predictions $\hat{d}_{ij}$ of associated pathogen densities. Better modeling procedures produce predictions of this kind that are closer to the actual pathogen densities, d_{ij}, in an overall sense as measured by a criterion like SDPE(M).

As a consequence of these considerations, a variant of the procedure statisticians call cross-validation (Stone, 1974) was chosen as the selection procedure. Specifically, for the ijth observation (x_{ij}, y_{ij}), whatever its form, parameter estimates were calculated using a modeling procedure, M, applied to all the other observations, that is, with the ijth observation deleted. Then, these deleted estimates were used to calculate the deleted predictions, $\hat{d}_{ij}$, and the associated deleted prediction errors, $d_{ij} - \hat{d}_{ij}$. These prediction errors were then combined into a composite squared standard deleted prediction error for pathogen densities at all stages.

$$\mathrm{SDPE}^2(M) = \frac{\sum_{j=1}^{J} \sum_{i=1}^{n_j} (d_{ij} - \hat{d}_{ij})^2}{\sum_{j=1}^{J} n_j} \tag{1}$$

Better modeling procedures, M, produce smaller SDPE(M)s, and the appropriate modeling procedure to use from a set of modeling procedures is one with minimal SDPE over that set or any other modeling procedure with a close SPDE, one within a few percentage points of the best.

The cross-validation scheme used here is often referred to as leave-one-out cross-validation. More general schemes are also possible; in the under k-fold cross-validation (Efron and Tibshirani, 1993), data are sorted in a fixed random order and decomposed into k approximately equal subsets. Subsets are removed one at a time, the model fit to the remaining data, and the corresponding estimated parameters used to predict all observations in the currently deleted subset. Leave-one-out cross-validation is a special case with k equal to the sample size. Kohavi (1995) recommends the use of 10-fold cross-validation in practice. However, when 10-fold cross-validation was used, it produced almost exactly the same results as leave-one-out cross-validation for the data analyzed in this paper; so only leave-one-out cross-validation results are reported.

For all of these models, with possibly transformed pathogen counts or densities expressed as functions of possibly transformed solids content, the estimated conditional expected values $\widehat{EY}|S = s_{ij}$ may be adjusted to estimates of conditional expected pathogen densities $\widehat{ED}|S = s_{ij}$ per 4 g solids. In particular, for models with $Y = C^p$,

$$\widehat{ED}|S = s_{ij} \equiv 4 \times \frac{(\max(\widehat{EY}|S = s_{ij}, 0))^{1/p}}{s_{ij}} \tag{2}$$

and, for models with $Y = D^p$,

$$\widehat{ED}|S = s_{ij} \equiv (\max(\widehat{EY}|S = s_{ij}, 0))^{1/p} \tag{3}$$

The expected pathogen density ED_j for each stage j adjusted to reflect the varying level of solids content at that stage may then be estimated by averaging the estimated conditional expected densities over solids content values, s_{ij}, observed at that stage, that is

$$\widehat{ED}_j \equiv \frac{\sum_{i=1}^{n_j} \widehat{ED}_j|S = s_{ij}}{n_j} \tag{4}$$

Estimating Pathogen Reduction Distributions. A desirable property of solids processing is the reduction of pathogen densities from stage 1 to a subsequent stage j by at least a specified target

amount. Such target reductions are typically expressed as $\log_{10}$ reductions. More specifically, denote by D_j the pathogen density per 4 g for the same random sample at each solids processing stage j, $1 \le j \le J$, and define the reduction $R_{1\to j}$ from stage 1 to stage $j > 1$ for that sample as

$$R_{1\to j} = \begin{cases} 0 & \text{if } D_1 = 0 \\ \dfrac{D_j}{D_1} & \text{otherwise} \end{cases} \tag{5}$$

and its associated $\log_{10}$ reduction $LR_{1\to j}$ as

$$LR_{1\to j} = \begin{cases} -\infty \text{ if } D_1 = 0 & \text{or } D_j = 0 \\ \log_{10}\left(\dfrac{D_j}{D_1}\right) & \text{otherwise} \end{cases} \tag{6}$$

The PEC-suggested target $\log_{10}$ reduction level by the final stage J for viable helminth ova and, hence, also for viable ascaris ova is $LR_{1\to J} \le -2$ and for enteric viruses is $LR_{1\to J} \le -3$.

Solids processing can reduce the pathogen density per 4 g solids content for a fixed sample or even leave it unchanged, but it does not increase the pathogen density, that is, $D_j \le D_1$ with probability 1; so the reduction $R_{1\to j}$ is at most 1. The set of pairs of observed pathogen densities consistent with this constraint is

$$E_{1\to j} = \{(d_{i1}, d_{kj}) | d_{kj} \le d_{i1},\ 1 \le i \le n_1,\ 1 \le k \le n_j\} \tag{7}$$

where d_{i1} denotes the ith of the n_1 pathogen densities observed for the first stage and d_{kj} is the kth of the n_j pathogen densities observed at a stage $j > 1$. This set has cardinality $|E_{1\to j}|$ and associated constant empirical weights $1/|E_{1\to j}|$ for pairs in the set. The empirical reduction variable $\hat{R}_{1\to j}$ over $E_{1\to j}$ is defined by

$$\hat{R}_{1\to j}(d_1, d_j) = \begin{cases} 0 & \text{if } d_1 = 0 \\ \dfrac{d_j}{d_1} & \text{otherwise} \end{cases} \quad \text{for } (d_1, d_j) \text{ in } E_{1\to j} \tag{8}$$

with the probability density function given by

$$\hat{p}_{1\to j}(r) = \frac{\#(\hat{R}_{1\to j} = r)}{|E_{1\to j}|} \tag{9}$$

where $\#(\cdot)$ denotes the number of observations satisfying the condition enclosed in parentheses. Let $r_{1\to j,h}$ denote the distinct reduction values, indexed by h, from stage 1 to stage $j > 1$ determined from equation 8. The associated empirical estimate of the probability distribution function for the reduction variable $R_{1\to j}$ is given by

$$\hat{P}(R_{1\to j} \le r) = P(\hat{R}_{1\to j} \le r) = \sum_{r_{1\to j,h} \le r} \hat{p}_{1\to j}(r_{1\to j,h}) \tag{10}$$

The expected reduction, $ER_{1\to j}$, may then be estimated as the weighted average of the $r_{1\to j,h}$

$$\widehat{ER}_{1\to j} = \sum_h r_{1\to j,h} \times \hat{p}_{1\to j}(r_{1\to j,h}) \tag{11}$$

Because the $\log_{10}$ reduction $LR_{1\to j}$ equals $-\infty$ with nonzero probability in practical situations, its expected value is not usually finite. However, a typical value for the $\log_{10}$ reduction can be estimated by taking the logarithm of the estimated expected reduction

$$\widehat{LR}_{1\to j} = \log_{10}(\widehat{ER}_{1\to j}) \tag{12}$$

This is finite except in the special case when the reduction equals zero.

The distribution for the $\log_{10}$ reduction $LR_{1\to j}$ for a random sample moving through solids processing from stage 1 to stage j satisfies

$$\begin{aligned} P(LR_{1\to j} \le u) &= P(D_1 = 0 \text{ or } D_j = 0) \\ &\quad + P(D_j \le 10^u \times D_1,\ D_1 > 0,\ D_j > 0) \\ &= P(D_1 = 0) + P(D_1 > 0,\ D_j = 0) \\ &\quad + P(D_j \le 10^u \times D_1,\ D_1 > 0,\ D_j > 0) \\ &= P(D_1 = 0) + P(D_j \le 10^u \times D_1,\ D_1 > 0) \\ &= P(R_{1\to j} \le 10^u) \\ &= P_{1\to j}(10^u) \end{aligned} \tag{13}$$

for $-\infty \le u \le 0$. This distribution may be estimated from the estimate of the distribution for $R_{1\to j}$ from equation 10.

Relationship Between Distributions of Initial and Final Pathogen Densities. Suppose that solids processing produces consistent proportional reductions regardless of the initial stage pathogen densities. More precisely, assume that the conditional distributions $P(R_{1\to J} \le r | D_1 = d_1)$ for reductions by the final SPT stage given nonzero initial densities, $d_1 > 0$, are all the same with a common value $P(R_{1\to J} \le r | D_1 > 0)$ and that this conditional distribution is the same no matter what the initial distribution is for D_1. As a consequence, the distribution $P_J(d_J)$ of the pathogen density D_J at the final stage J can be related to the distribution P_1 for D_1.

$$\begin{aligned} P_J(d_J) \equiv P(D_J \le d_J) &= \int_{u \ge 0} P(R_{1\to J} \times D_1 \le d_J | D_1 = u) dP_1(u) \\ &= P(D_1 = 0) + \int_{u > 0} P(R_{1\to J} \le d_J/u | D_1 > 0) dP_1(u) \end{aligned} \tag{14}$$

for $d_J \ge 0$. An estimate of the integrand of equation 14 may be computed as in equation 10. Specifically, define $E_{1\to J,+}$ to be the subset of $E_{1\to J}$ consisting of observed pairs with positive initial pathogen density values ($d_{i1} > 0$). There are $|E_{1\to J,+}|$ pairs in this set with constant empirical weights $1/|E_{1\to J,+}|$. The associated conditional reduction variable, $\hat{R}_{1\to J,+}$, is the unconditional reduction variable, $\hat{R}_{1\to J}$, restricted to the set $E_{1\to J,+}$ with probability density function

$$\hat{p}_{1\to J,+}(r) = \frac{\#(\hat{R}_{1\to J,+} = r)}{|E_{1\to J,+}|} \tag{15}$$

and with the distribution function providing the desired empirical estimate

$$\hat{P}(R_{1\to J} \le r | D_1 > 0) = P(\hat{R}_{1\to J,+} \le r) = \sum_{r_{1\to J,h+} \le r} \hat{p}_{1\to J,+}(r_{1\to J,h+}) \tag{16}$$

determined by the distinct reduction values $r_{1\to J,h+}$, indexed by $h+$, from positive initial stage pathogen densities to final stage

values. Equations 14 and 16 are used below in the estimation of the effect of surges and expansions in initial pathogen densities, D_1, on final pathogen densities, D_J.

Effect of Large Surges in Initial Pathogen Densities. Let $D_1^{(0,\cdot)}$ denote the random initial pathogen density in effect for solids processing during sampling with probability density $p_1^{(0,\cdot)}$ and distribution $P_1^{(0,\cdot)}$. These may be estimated by the associated empirical density function $\hat{p}_1^{(0,\cdot)}$ and empirical distribution function $\hat{P}_1^{(0,\cdot)}$). Suppose that a surge occurs in the random initial pathogen density in the sense that this random pathogen density $D_1^{(\varepsilon,\Delta)}$ is a large positive value Δ with small probability ε and is otherwise unchanged with probability $1 - \varepsilon$. This means that the distribution for the random initial pathogen density is given by

$$P_1^{(\varepsilon,\Delta)} = (1 - \varepsilon) \times P_1^{(0,\cdot)} + \varepsilon \times 1_{(\cdot \geq \Delta)} \tag{17}$$

where $1_{(\cdot \geq \Delta)}$ indicates whether the initial pathogen density is no smaller than the value Δ. The associated probability density is given by $p_1^{(\varepsilon,\Delta)} = (1-\varepsilon) \times p_1^{(0,\cdot)} + \varepsilon \times 1_{(\cdot = \Delta)}$ where $1_{(\cdot = \Delta)}$ indicates whether the initial pathogen density equals the value Δ. This may be estimated by

$$\hat{P}_1^{(\varepsilon,\Delta)} = (1 - \varepsilon) \times \hat{P}_1^{(0,\cdot)} + \varepsilon \times 1_{(\cdot \geq \Delta)} \tag{18}$$

with associated probability density function

$$\hat{p}_1^{(\varepsilon,\Delta)} = (1 - \varepsilon) \times \hat{p}_1^{(0,\cdot)} + \varepsilon \times 1_{(\cdot = \Delta)} \tag{19}$$

where

$$\hat{p}_1^{(0,\cdot)}(d_1) = \frac{\#(d_{i1} = d_1)}{n_1} \tag{20}$$

and where d_{i1} are the n_1 pathogen densities observed at stage 1. In other words, the estimated distribution $\hat{P}_1^{(\varepsilon,\Delta)}$ is concentrated on the observed initial pathogen densities d_{i1}, $1 \leq i \leq n_1$ and on the value Δ of the surge in the initial pathogen densities. The distribution $P_J^{(\varepsilon,\Delta)}$ induced by a surge of size $\Delta > 0$ with probability ε at stage 1 may then be estimated, using equations 14 and 17 and then the estimates of equations 16 and 20, by

$$\begin{aligned}\hat{P}_J^{(\varepsilon,\Delta)}(d_J) &= \hat{P}(R_{1\to J} \times D_1^{(\varepsilon,\Delta)} \leq d_J) \\ &= (1 - \varepsilon) \\ &\quad \times \left(\frac{\#(d_{i1} = 0)}{n_1} + \sum_{d_{i1} > 0} \frac{\hat{P}(R_{1\to J} \leq d_J/d_{i1} | D_1 > 0)}{n_1} \right) \\ &\quad + \varepsilon \times \hat{P}(R_{1\to J} \leq d_J/\Delta \,|\, D_1 > 0) \\ &= (1 - \varepsilon) \times \hat{P}_J^{(0,\cdot)}(d_J) + \varepsilon \times \hat{P}(R_{1\to J} \leq d_J/\Delta | D_1 > 0)\end{aligned} \tag{21}$$

The quantity $\hat{P}_J^{(\varepsilon,\Delta)}(RL)$ estimates the probability that a random sample at the final stage J of solids processing is at or below the Class A required level RL of 1 pathogen per 4 g for viable helminth ova, for viable ascaris ova, or for enteric viruses under a surge of size Δ with probability ε at the initial stage of solids processing. For enteric viruses, $\hat{P}(R_{1\to J} = 0) = 1$, and so $\hat{P}_J^{(\varepsilon,\Delta)}(RL) = 1$ for all ε and Δ. However, for viable helminth ova and for viable ascaris ova, $\hat{P}(R_{1\to J} = 0) < 1$, and so, for large enough Δ and for positive ε, $\hat{P}_J^{(\varepsilon,\Delta)}(RL) < 1$; in other words, there will be a nonzero estimated probability of a sample that does not meet Class A requirements for the associated type of pathogen.

Effect of Expansions of Initial Pathogen Densities. Let $D_1^{(1)}$ denote the random initial pathogen density at stage 1 in effect for solids processing during sampling with probability density $p_1^{(1)}$ and distribution $P_1^{(1)}$. These may be estimated by the associated empirical density function $\hat{p}_1^{(1)}$ and empirical distribution function $\hat{P}_1^{(1)}$. Suppose that the random pathogen density at stage 1 is expanded by a factor $\eta > 0$ in the sense that the random pathogen density is changed to $D_1^{(\eta)} = \eta \times D_1^{(1)}$ and so has distribution function

$$P_1^{(\eta)}(d_1^{(\eta)}) \equiv P(D_1^{(\eta)} \leq d_1^{(\eta)}) = P_1^{(1)}(d_1^{(\eta)}/\eta) \tag{22}$$

This may be estimated by

$$\hat{P}_1^{(\eta)}(d_1^{(\eta)}) = \hat{P}_1^{(1)}(d_1^{(\eta)}/\eta) \tag{23}$$

with associated estimated probability density function

$$\hat{p}_1^{(\eta)}(d_1^{(\eta)}) = \frac{\#(d_{i1}^{(1)} = d_1^{(\eta)}/\eta)}{n_1} \tag{24}$$

where $d_{i1}^{(1)}$ is the n_1 pathogen density observed at stage 1. In other words, the estimated distribution $\hat{P}_1^{(\eta)}$ is concentrated on the unique values determined by $\eta \times d_{i1}^{(1)}$, $1 \leq i \leq n_1$. The distribution $P_J^{(\eta)}$ induced by the expansion factor η at stage 1 may then be estimated, using equations 14 and 22 and then the estimates of equations 16 and 24, by

$$\begin{aligned}\hat{P}_J^{(\eta)}(d_J^{(\eta)}) = \hat{P}(R_{1\to J} \times D_1^{(\eta)} \leq d_J^{(\eta)}) &= \frac{\#(d_{i1}^{(1)} = 0)}{n_1} \\ &\quad + \sum_{d_{i1}^{(1)} > 0} \frac{\hat{P}(R_{1\to J} \leq d_J^{(\eta)}/(\eta \times d_{i1}^{(1)}) | D_1 > 0)}{n_1}\end{aligned} \tag{25}$$

The quantity $\hat{P}_J^{(\eta)}(RL)$ estimates the probability that a random sample at the final stage J of solids processing is at or below the Class A required level RL of 1 pathogen per 4 g for viable helminth ova, for viable ascaris ova, or for enteric viruses under an expansion factor of η at the initial stage of solids processing. For enteric viruses, $\hat{P}(R_{1\to J} = 0) = 1$; so $\hat{P}_J^{(\eta)}(RL) = 1$ for all η. However, for viable helminth ova and for viable ascaris ova, $\hat{P}(R_{1\to J} = 0) < 1$; so for large enough η, $\hat{P}_J^{(\eta)}(RL) < 1$. In other words, there will be a nonzero estimated probability of a sample that does not meet Class A requirements for the associated type of pathogen.

Results

The District's SPTs were operated under codified conditions for a period of approximately 3 years. Numerous samples were collected for pathogen analyses at the four SPT stages of digester feed, digester draw, lagoon draw, and air-dried product. Counts of viable helminth ova, viable ascaris ova, and enteric viruses were recorded for samples of varying sizes, and densities per 4 g dry solids were computed for each of these samples. See the companion paper by Tata et al. (2000) for a more detailed discussion of the results of that study.

Summary statistics are presented in Table 1. Observed densities for viable helminth ova, viable ascaris ova, and enteric viruses for all SPT stages combined are plotted in Figure 1 versus $\log_{10}$-transformed solids content. These plots indicate a nonlinear relationship between pathogen density and solids content and suggest the consideration of analysis of covariance models with transformed solids content as the covariate.

The analysis of covariance model and its associated one-way analysis of variance model generate an extensive family of mod-

Table 1—Summary statistics.

Stage	Viable helminth ova				Enteric viruses		
	Samples	Total dry solids, g[a]	Total ova[b]	Ascaris ova[b]	Samples	Total dry solids, g[a]	Total PFUs[c]
Digester feed	57	1 032.25	177	77	46	537.01	197
Digester draw	52	804.33	53	34	49	623.20	15
Lagoon draw	56	2 069.83	35	19	36	3 844.25	0
Dried product	155	22 520.61	26	22	119	22 800.16	0
Total	320	26 427.02	291	152	250	27 804.63	212

[a] Total mass of solids from all samples for a given stage or for all stages combined.
[b] Number of viable ova found in the associated total mass of solids.
[c] Number of plaque-forming units found in the associated total mass of solids.

eling procedures corresponding to the various adjustments that may be used to compute initial parameter estimates from pathogen-count data collected during solids processing. In particular, the response variable Y may be pathogen counts C as originally recorded, pathogen densities D per 4 g solids content, or some transform of either of these. Also, the covariate(s) X may be any transform of solids content S or of any other available supplementary variables.

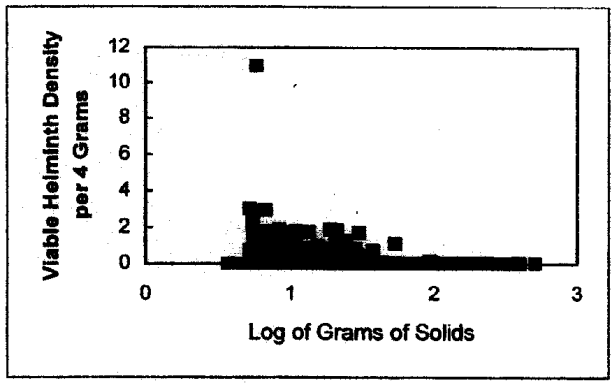
A cross-validation procedure is used to select an appropriate model from this family for each of the three types of pathogens, minimizing SDPEs for predicting pathogen densities per 4 g solids content. This criterion is based on the prediction of pathogen densities per 4 g to conform with the requirements for Class A biosolids. The predictions used in computing this criterion are deleted predictions in the sense that each observed pathogen density is predicted using all of the remaining data, not including the density value being predicted; so the selection procedure is a leave-one-out cross-validation.

The possible response variables Y that were considered included power transforms of pathogen counts, C^p, and power transforms of pathogen densities, D^p, over multiples of 0.25 for the power p. The possible covariates X that were considered included the natural log transform, $\ln(S)$, and power transforms of solids content, S^q, over multiples of 0.5 for the power q and the analysis of variance model constant in S. The selected transforms of solids content for all of the three types of pathogen were hyperbolic in form, that is, S^q with $q < 0$. However, for viable helminth ova and for viable ascaris ova, simpler analysis of variance models that do not depend on solids content may be used in place of the chosen hyperbolic models. For enteric viruses, on the other hand, the chosen hyperbolic model provides tangibly better predictions than the associated analysis of variance model indicating a more complex dependence on solids content than for the other two pathogen types.

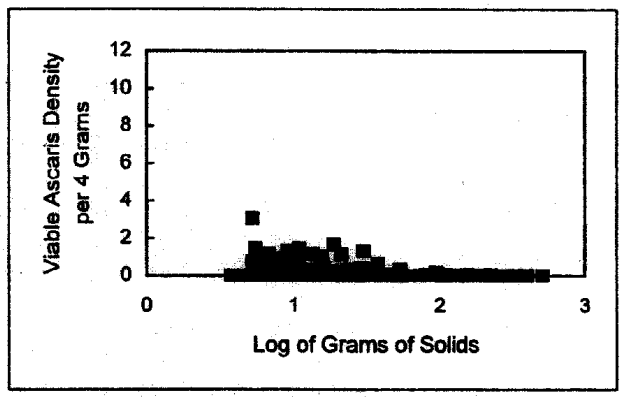
Table 2 reports SDPEs for a variety of models applied to data from the District's study of its SPTs. The nine models designated in Table 2 as M_1 through M_9 either minimize SDPE within indicated classes of models or provide suboptimal choices with simplified structure. Models are chosen, one for each pathogen type, from among these nine models to generate the estimates reported in Table 3 of the expected pathogen density at each solids processing stage. Estimated expected pathogen densities at the fourth and final stage are less than the Class A required level of 1 per 4 g viable helminth ova, viable ascaris ova, and enteric viruses and are also below the required level for enteric viruses at the second and third stages as well. These estimates adjust for varying levels of solids content in cases in which the chosen model indicates this is warranted.

For viable helminth ova and viable ascaris ova, pathogen densities may be reasonably predicted using analysis of variance models (models M_3 and M_6, respectively) that are independent of solids content values. For enteric viruses, however, there is a tangible benefit to using an analysis of covariance model (model M_7) with a nontrivial dependence on solids content. Pathogen densities for viable helminth ova and ascaris ova are reasonably predicted indirectly by first modeling possibly transformed pathogen counts using an analysis of variance model with expected pathogen counts that depend only on the stage of solids processing and then by adjusting predicted pathogen counts into predicted pathogen densities. Pathogen densities for enteric viruses are also reasonably predicted indirectly by first modeling transformed pathogen counts but using an analysis of covariance model with expected pathogen counts that depend on both the stage of solids processing and the associated levels of solids content. Details are provided in later subsections.

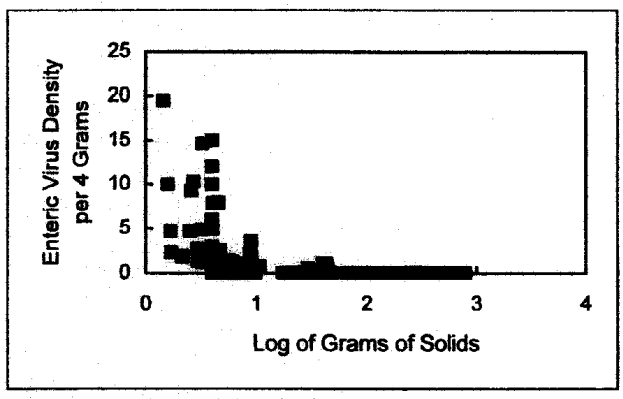
It is also desirable that solids processing reduce pathogen densities from the initial to the final solids processing stage by at least specified target amounts. In particular, the PEC-suggested target $\log_{10}$ reduction level is -2 for viable helminth ova and, hence, also for viable ascaris ova and -3 for enteric viruses. Estimated distributions are given in Table 4 for the percent pathogen inactivation (i.e., 100% minus the percent pathogen reduction). These distributions are highly asymmetric and so are distributions for pathogen reductions. Typical values for pathogen reduction thus need to be reported with nonparametric measures of their variability rather than with standard symmetric confidence intervals. For this reason, Table 5 reports not only estimates for the District's SPTs of typical values for reductions and for associated $\log_{10}$ reductions but also estimates of probabilities of $\log_{10}$ reductions at or below PEC-suggested target levels. The estimated typical $\log_{10}$ reductions from stage 1 to 4 for viable helminth ova, viable ascaris ova, and enteric viruses are less than the associated target levels. Moreover, there is approximately a 0.91 probability of meeting the PEC-suggested target $\log_{10}$ reduction level for viable helminth ova, approximately a 0.94 probability for viable ascaris ova, and approximately a 1.00 probability for enteric viruses. See the Pre-



Viable helminth densities vs. solids content



Viable ascaris densities vs. solids content



Enteric virus densities vs. solids content

Figure 1—Pathogen densities versus solids content.

diction of Pathogen Reduction–Inactivation Capability subsection for further details.

Suppose solids processing produces consistent proportional reductions, that is, the distributions for reductions, conditioned on positive initial pathogen density values, are the same with the same common value whatever the distribution is for initial pathogen densities. This assumption means that the chance that pathogen densities are reduced by at least some percentage, for example, by 10, 25, 50, or 100%, is assumed to be the same no matter what the initial pathogen density is. It is the basis for the assessment of the effect of changes in initial stage pathogen densities on final stage pathogen densities.

Suppose further that a surge (a large isolated burst) occurs in the random pathogen density at stage 1 in the sense that the random initial pathogen density is a large positive value Δ with small probability ε and is otherwise unchanged with probability $1 - \varepsilon$. Table 6 of Tata et al. (2000) contains estimates of the probability, for selected values of Δ and ε, that pathogen densities at stage 4 will satisfy Class A requirements for viable helminth ova and ascaris ova. Results presented in that table indicate that, even if surges as great as 10 000 pathogens per 4 g occur with probability as great as 0.05, the estimated probabilities of meeting Class A requirements for viable helminth and viable ascaris ova are still greater than 0.99. Estimated probabilities of meeting Class A requirements for enteric viruses are not reported in that table because all such values are 1.00 because all observed enteric virus densities at stage 4 are 0.00.

Alternatively, suppose that the random initial pathogen density is expanded by a large factor $\eta > 0$ (a large sustained increase). Table 6 of Tata et al. (2000) also contains estimates for selected values of η of the probability that pathogen densities at stage 4 will satisfy Class A requirements for viable helminth ova and ascaris ova. The results of that table indicate that, even if the initial pathogen densities are expanded by a factor of 100, the estimated probability of meeting Class A requirements for viable helminth ova is greater than 0.92 and for viable ascaris ova it is greater than 0.96. Thus, even under such exceptionally large expansions, Class A requirements for viable helminth ova and viable ascaris ova still hold with quite high estimated probabilities. As noted previously, estimated probabilities of meeting Class A requirements for enteric viruses are not reported in Table 6 of Tata et al. (2000) because all such values are 1.00 because all observed enteric virus densities at stage 4 are 0.00. See the Effect of Hypothetical Surges–Expansions in Initial Densities on Final Pathogen Densities subsection for further details.

Modeling Viable Helminth Ova Counts and Densities. When the response variable Y is a transform of pathogen counts C for viable helminth ova, the best model M_1 [in the sense of lowest SDPE(M) for associated pathogen densities D] uses the untransformed counts $Y = C$ and the transform $X = S^{-8.5}$ of solids content. On the other hand, when the response variable Y is a transform of pathogen densities D for viable helminth ova per 4 g solids content, the best model M_2 [also in the sense of lowest SDPE(M) for pathogen densities D] uses the transforms $Y = D^{0.75}$ and $X = S^{-1.0}$. Standard deleted prediction errors for related models are presented in Table 2.

The score SDPE(M_1) = 0.660 0 for the first of these models, with estimates based on pathogen counts, is somewhat smaller, approximately 4.8% smaller, than the score SDPE(M_2) = 0.693 3 for the other model, with estimates based on pathogen densities. Thus, the first model is more appropriate to use to estimate expected pathogen densities for viable helminth ova. However, the analysis of variance model M_3 corresponding to model M_1 using untransformed $Y = C$ and independent of solids content S has a score of 0.663 5, only 0.5% greater than the score for model M_1; it provides essentially the same predictions as the best model and is preferable because of its simpler level of dependence on solids content. For this reason, model M_3 is used in computing the values reported in Table 3.

Modeling Viable Ascaris Ova Counts and Densities. When the response variable Y is a transform of pathogen counts C for viable ascaris ova, the best model M_4 [in the sense of lowest SDPE(M) for associated pathogen densities D] uses the transforms $Y = C^{0.75}$ and $X = S^{-11.5}$. On the other hand, when the response

Table 2—Standard deleted prediction errors.

Densities	p	$g(S)$	Best SDPE	SDPE $g(S) = 0$
Viable helminth ova densities[a]				
Model-transformed helminth ova counts, $Y = C^p$, versus transformed solids content, $X = g(S)$	0.75	$S^{-12.0}$	0.662 1	—
	1.00	$S^{-8.5}$	0.660 0 (M_1)	0.663 5 (M_3)
	1.25	$S^{-7.0}$	0.667 4	—
Model-transformed helminth ova densities, $Y = D^p$, versus transformed solids content, $X = g(S)$	0.50	$S^{-1.0}$	0.704 2	—
	0.75	$S^{-1.0}$	0.693 3 (M_2)	0.725 8
	1.00	$S^{-1.0}$	0.697 5	—
Viable ascaris ova densities[b]				
Model-transformed ascaris ova counts, $Y = C^p$, versus transformed solids content, $X = g(S)$	0.50	$S^{-17.5}$	0.303 8	—
	0.75	$S^{-11.5}$	0.292 1 (M_4)	0.293 4 (M_6)
	1.00	$S^{-4.5}$	0.293 6	—
Model-transformed ascaris ova densities, $Y = D^p$, versus transformed solids content, $X = g(S)$	0.75	$S^{-0.5}$	0.299 7	—
	1.00	$S^{-0.5}$	0.297 8 (M_5)	0.305 0
	1.25	$S^{-1.0}$	0.304 3	—
Enteric virus densities[c]				
Model-transformed virus counts, $Y = C^p$, versus transformed solids content, $X = g(S)$	0.50	$S^{-12.5}$	1.640 9	—
	0.75	$S^{-35.0}$	1.618 8 (M_7)	1.715 2 (M_9)
	1.00	0	1.706 1	—
Model-transformed virus densities, $Y = D^p$, versus transformed solids content, $X = g(S)$	0.75	$S^{-8.0}$	1.761 8	—
	1.00	$S^{-10.0}$	1.753 6 (M_8)	2.076 2
	1.25	$S^{-12.0}$	1.772 0	—

[a] Models M_1, M_2, and M_3 are discussed in the Modeling Viable Helminth Ova Counts and Densities subsection.
[b] Models M_4, M_5, and M_6 are discussed in the Modeling Viable Ascaris Ova Counts and Densities subsection.
[c] Models M_7, M_8, and M_9 are discussed in the Modeling Enteric Virus Counts and Densities subsection.

variable Y is a transform of pathogen densities D for viable ascaris ova per 4 g solids content, the best model M_5 [also in the sense of lowest SDPE(M) for pathogen densities D] uses untransformed densities $Y = D$ and transformed solids content $X = S^{-0.5}$. Standard deleted prediction errors for related models are presented in Table 2.

The score SDPE(M_4) = 0.292 1 for the first of these models, with estimates based on pathogen counts, is only 1.9% smaller than the score SDPE(M_5) = 0.297 8 for the other model, with estimates based on pathogen densities. Thus, either model may be reasonably used to estimate expected pathogen densities for viable ascaris ova. Moreover, the analysis of variance model M_6 corresponding to the better of these two models using transformed pathogen counts $Y = C^{0.75}$ and independent of solids content S has a score of 0.293 4, only 0.4% greater than the best model M_4; it provides essentially the same predictions as the best model while depending on a simpler level of dependence on solids content. For this reason, model M_6 is used in place of the best model M_4 in computing results reported in Table 3.

Table 3—Expected pathogen density per 4 g adjusted for varying solids content.

Stage	Helminth ova: Total ova per 4 g[a]	Helminth ova: Ascaris ova per 4 g[b]	Enteric viruses per 4 g[c]
Digester feed	1.014×10^{0}	3.519×10^{-1}	3.641×10^{0}
Digester draw	3.705×10^{-1}	1.521×10^{-1}	1.168×10^{-1}
Lagoon draw	1.690×10^{-1}	5.381×10^{-2}	4.084×10^{-22}
Dried product	1.041×10^{-2}	3.991×10^{-3}	7.104×10^{-22}

[a] Estimated using model M_3 of Table 2.
[b] Estimated using model M_6 of Table 2.
[c] Estimated using model M_7 of Table 2.

Modeling Enteric Virus Counts and Densities. When the response variable Y is a transform of pathogen counts C for enteric viruses, the best model M_7 [in the sense of lowest SDPE(M) for associated pathogen densities D] uses the transforms $Y = C^{0.75}$ and $X = S^{-35.0}$. On the other hand, when the response variable Y is a transform of pathogen densities D for enteric viruses per 4 g solids

Table 4—Estimated distributions[a] of the percent pathogen inactivation.

	Viable helminth ova		Viable ascaris ova	
Inactivation, %	Frequency	Cumulative frequency	Frequency	Cumulative frequency
0–5	2.30×10^{-4}	2.30×10^{-4}	0.00	0.00
5–10	0.00	2.30×10^{-4}	0.00	0.00
10–15	2.30×10^{-4}	4.60×10^{-4}	2.35×10^{-4}	2.35×10^{-4}
15–20	2.30×10^{-4}	6.90×10^{-4}	0.00	2.35×10^{-4}
20–25	2.30×10^{-4}	9.20×10^{-4}	2.35×10^{-4}	4.70×10^{-4}
25–30	2.30×10^{-4}	1.15×10^{-3}	0.00	4.70×10^{-4}
30–35	1.15×10^{-4}	1.27×10^{-3}	1.18×10^{-4}	5.88×10^{-4}
35–40	1.15×10^{-4}	1.38×10^{-3}	1.18×10^{-4}	7.06×10^{-4}
40–45	5.75×10^{-4}	1.96×10^{-3}	2.35×10^{-4}	9.41×10^{-4}
45–50	6.90×10^{-4}	2.65×10^{-3}	0.00	9.41×10^{-4}
50–55	4.60×10^{-4}	3.11×10^{-3}	2.35×10^{-4}	1.18×10^{-3}
55–60	3.45×10^{-4}	3.45×10^{-3}	3.53×10^{-4}	1.53×10^{-3}
60–65	6.90×10^{-4}	4.14×10^{-3}	1.18×10^{-4}	1.65×10^{-3}
65–70	1.50×10^{-3}	5.64×10^{-3}	1.53×10^{-3}	3.18×10^{-3}
70–75	1.73×10^{-3}	7.37×10^{-3}	1.65×10^{-3}	4.83×10^{-3}
75–80	3.91×10^{-3}	1.13×10^{-2}	2.94×10^{-3}	7.77×10^{-3}
80–85	5.06×10^{-3}	1.64×10^{-2}	4.59×10^{-3}	1.24×10^{-2}
85–90	9.66×10^{-3}	2.61×10^{-2}	9.18×10^{-3}	2.16×10^{-2}
90–95	1.87×10^{-2}	4.48×10^{-2}	1.59×10^{-2}	3.75×10^{-2}
95–100	9.55×10^{-1}	1.00	9.63×10^{-1}	1.00

[a] The estimated distribution for enteric viruses is concentrated on 100% inactivation.

content, the best model M_8 [also in the sense of lowest SDPE(M) for pathogen densities D] uses untransformed densities $Y = D$ and transformed solids content $X = S^{-10.0}$. Standard deleted prediction errors for related models are presented in Table 2.

The score SDPE(M_7) = 1.618 8 for the first of these models, with estimates based on pathogen counts, is 7.7% smaller than the score SDPE(M_8) = 1.753 6 for the other model, with estimates based on pathogen densities, and is more appropriate to use to estimate expected pathogen densities for enteric viruses. Moreover, the analysis of variance model M_9 corresponding to model M_7 using transformed pathogen counts $Y = C^{0.75}$ and independent of solids content S has a score of 1.715 2, 6.0% greater than the best model M_7. Thus, it is preferable to model enteric virus densities using an analysis of covariance model with a nontrivial dependence on solids content. For this reason, the best model M_7 is used in computing results reported in Table 3.

Prediction of Pathogen Reduction–Inactivation Capability. To assess the pathogen reduction–inactivation capability of the District's solids treatment process, observed data for each of the three pathogen types are used to compute empirical estimates of probability density functions and cumulative distribution functions for the pathogen reductions. Specifically, distributions are estimated for reductions $R_{1\to j}$, for $j > 1$ (i.e., for the second, third, and fourth SPT stages) in pathogen density resulting for a random sample with first stage pathogen density D_1 and subsequent pathogen density D_j. Distributions are also estimated for the associated

Table 5—Estimated reduction capability features.

Pathogen type	Stage j	Expected reduction $\widehat{ER}_{1\to 4}$	Log_{10} expected reduction, $\log_{10}(\widehat{ER}_{1\to 4})$	Target level	Probability of $\log_{10}$ reduction at or below the target level
Viable helminth ova	2	1.52×10^{-1}	−0.82		0.622
	3	5.48×10^{-2}	−1.26		0.844
	4	8.83×10^{-3}	−2.05	−2	0.913
Viable ascaris ova	2	6.65×10^{-2}	−1.18		0.856
	3	3.39×10^{-2}	−1.46		0.911
	4	6.46×10^{-3}	−2.19	−2	0.935
Enteric viruses	2	3.66×10^{-2}	−1.44		0.863
	3	0.00	$-\infty$		1.000
	4	0.00	$-\infty$	−3	1.000

quantity: the percent pathogen inactivation $(1 - R_{1\to4}) \times 100\%$ by the final stage $J = 4$. These quantities are estimated by assuming that the conditional distributions of the subsequent pathogen densities $D_j|D_1 = d_1$, conditioned on the initial density value, d_1, are the same for all $d_1 > 0$ and so are the same as the distribution for subsequent pathogen densities $D_j|D_1 > 0$ conditioned on D_1 being positive. Estimated distributions for the percent pathogen inactivation by the end of solids processing are provided in Table 4.

Estimates of associated expected reductions and probabilities of meeting target reduction levels are provided in Table 5. An estimated $\log_{10}$ reduction of -2.05 by the end of solids processing with an estimated probability of 0.913 of being less than the target level of -2 is achieved for viable helminth ova, while an estimated $\log_{10}$ reduction of -2.19 by the end of solids processing with estimated probability of 0.935 of being less than the target level of -2 is achieved for viable ascaris ova. Because all observed enteric virus densities at stage 4 are zero, the estimated $\log_{10}$ reduction is $-\infty$ with an estimated probability of 1.000 of being less than the target level of -3.

Note that the distributions of Table 4 and, as a consequence, also the distributions for reductions and $\log_{10}$ reductions are quite asymmetric; so the probabilities of Table 5 provide nonparametric measures of the variability about associated estimates of $\log_{10}$ expected reductions relative to target levels and are more appropriate in this case than typically used symmetric confidence intervals.

Effect of Hypothetical Surges–Expansions in Initial Densities on Final Pathogen Densities. Table 6 of Tata et al. (2000) contains estimates of the probability of a random sample meeting Class A requirements, first under various probabilities and sizes of surges and then under various expansion factors. These estimates are based on the assumption that solids processing produces consistent proportional reductions in the sense that the conditional distributions for $R_{1\to4}|D_1 = d_1$ are all the same for any positive initial pathogen density $d_1 > 0$. Surges of size Δ pathogens per 4 g with probability ε are modeled by the family of random initial pathogen densities $D_1^{(\varepsilon,\Delta)}$ equal to Δ with probability ε and otherwise equal to the random density $D_1^{(0,\cdot)}$ in effect during sampling. Expansions of size $\eta > 0$ are modeled by the family of random initial pathogen densities $D_1^{(\eta)} = \eta \cdot D_1^{(1)}$ obtained by rescaling the random initial pathogen density $D_1^{(1)}$ in effect during sampling by the multiple η. Results of Table 6 of Tata et al. (2000) indicate that even for very large surges of $\Delta = 10\,000$ viable helminth–ascaris ova per 4 g and under the relatively large probability of occurrence of 0.05, Class A biosolids requirements will be met with an estimated probability greater than 0.99. Furthermore, even under the exceptionally large expansion factor of 100 times the level of observed viable helminth–ascaris densities, Class A biosolids requirements will be met with an estimated probability greater than 0.92.

Conclusions and Engineering Significance

Statistical methods are presented for assessing the pathogen reduction capability of a biosolids process. These methods are used to assess the District's SPTs by analyzing the data of a full-scale study of those SPTs. Data are analyzed for three pathogen types: viable helminth ova, viable ascaris ova, and enteric viruses. However, these methods are also applicable to the study of inactivation of any microorganisms or pathogens in other solids processing operations consisting of unit processes that may or may not be similar to the District's SPTs.

Parametric analysis of covariance models are used to analyze pathogen density levels at the four stages of solids processing from digester feed to air-dried product of the District's SPTs. Models are selected through a cross-validation scheme that minimizes the SDPE for predicting pathogen density levels per 4 g because the U.S. EPA standards for Class A biosolids are stated in terms of such densities.

Nonparametric methods are developed for determining pathogen reduction and inactivation distributions and selected summary measures for these distributions. Estimates of expected pathogen reduction from the initial to the final solids processing stage are computed, as are estimates of the probability of meeting target pathogen reduction levels.

Nonparametric methods are developed for conducting sensitivity analyses to assess the effect of changes in initial stage pathogen densities on final stage pathogen densities. Two kinds of changes are supported: (1) surges representing exceptionally large isolated bursts of pathogens in the digester feed and (2) uniform expansions representing exceptionally large and sustained increases in pathogens in the digester feed. Results for these sensitivity analyses are presented in the companion paper by Tata et al. (2000).

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