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Estimation of Infectious Risks in Residential Populations Exposed to Airborne Pathogens During Center Pivot Irrigation of Dairy Wastewaters

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Supporting Information

ABSTRACT: In the western United States where dairy wastewaters are commonly land applied, there are concerns over individuals being exposed to airborne pathogens. In response, a quantitative microbial risk assessment (QMRA) was performed to estimate infectious risks after inhalation exposure of pathogens aerosolized during center pivot irrigation of diluted dairy wastewaters. The dispersion of pathogens (*Campylobacter jejuni*, *Escherichia coli* O157:H7, non-O157 *E. coli*, *Listeria monocytogenes*, and *Salmonella* spp.) was modeled using the atmospheric dispersion model, AERMOD. Pathogen concentrations at downwind receptors were used to calculate infectious risks during one-time (1, 8, and 24 h) and multiday (7 d at 1 h d^{-1}) exposure events using a β-Poisson dose−response model. This assessment considered risk of infection in residential populations that were 1 to 10 km from a center pivot operation. In the simulations, infectious risks were estimated to be the greatest in individuals closest to the center pivot, as a result of a higher pathogen dose. On the basis of the results from this QMRA, it is recommended that wastewaters only be applied during daylight hours when inactivation and dilution of airborne pathogens is highest. Further refinement of the dispersion and dose−response models should be considered to increase the utility of this QMRA.

INTRODUCTION

Dairy wastewaters, which are a combination of manure (feces and urine), waters used to flush milking parlors and feed lanes, and sometimes lot runoff, are placed in engineered ponds to contain the material and prevent leaching. Because the capacity of storage ponds can eventually be exceeded, it is necessary to remove wastewater at regular intervals. In the arid west, where dairy forage crops must be irrigated, the wastewaters are a valuable water and nutrient resource. As a result, the wastewaters are commonly blended with irrigation water and then land applied through center pivots, wheel lines, and spray guns.[1] Since cattle feces, manures, and wastewaters are known to harbor zoonotic pathogens,[2,3] the unintended consequence is that microbes within the discharge from pressurized irrigation systems can become aerosolized and drift off site. Once airborne, the pathogens could potentially cause infection in downwind individuals if they are directly inhaled or, in the case of enteric pathogens, swallowed after becoming lodged in the upper respiratory tract (nasal cavity and nasopharynx mucus). Though this latter mode of transmission has not been demonstrated in clinical trials with humans,[4] there is evidence from animal studies suggesting that airborne transmission of enteric pathogens is possible.[5−7]

Since there is substantial interest in understanding the health impacts of airborne pathogens and other biologically derived agents,[8,9] a number of studies to date have attempted to quantify and characterize bioaerosols at livestock facilities and manure wastewater application sites.[10] The overarching trend seen in these types of studies is that the bioaerosol concentrations decrease with increasing downwind distance from the source.[11,12] One interesting fact among bioaerosol studies is that, despite the excretion of zoonotic pathogens by livestock, very few if any are detected downwind of manure application sites and housing units.[13−15] A number of factors could be at play here, as pathogen levels in manures tend to be lower than that of indicator organisms;[16] plus the aerosol sampling methods and ambient conditions (e.g., temperature, humidity, solar radiation) could be impacting the viability of microorganisms resulting in fewer detects if culture-dependent techniques are used for enumeration. While molecular-based methods such as quantitative PCR (qPCR) have increased sensitivity over traditional culture techniques, they cannot distinguish between inactive and infectious organisms.[17] Due to the difficulties in detecting airborne pathogens and deciphering positive results, many researchers instead target indicator organisms in bioaerosol studies.[12,18−20] However, the survival

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Table 1. Calculation of Pathogen Emission Rates from the Simulated 396 m Long Center Pivot Spraying Diluted Dairy Wastewater

scenario[a]	water flow rate (L min^{-1})	pathogen concentration[b] (cells 100 mL^{-1} wastewater)	wastewater (%)	aerosolization efficiency (%)	pathogen emission rate[c] (cells s^{-1})
A (low)	3217	10^3	5	0.1	2.7×10^1
B (medium)	3217	10^4	10	1.5	8.0×10^3
C (high)	3217	10^5	10	1.5	8.0×10^4
D (very high)	3217	10^6	20	3.0	3.2×10^6

[a]Low, medium, high, and very high represent potential risk levels associated with irrigation scenarios A, B, C, and D, respectively. [b]Data source: See ref. 3. Pathogen concentrations orginally determined using qPCR. [c]Emission values divided by 1.0×10^6 prior to use in AERMOD so model output units were in cells m^{-3}.

of airborne indicator organisms is likely different from that of pathogens, making them a poor pathogen surrogate.[21−23]

In lieu of empirical assessments, another approach is to approximate the dispersion and transport of airborne pathogens using atmospheric models.[24,25] Various studies to date have used steady-state Gaussian plume models to estimate bioaerosol dispersion at biosolids land application sites,[26] green waste composting facilities,[27] and during spray irrigation events of food-processing and municipal wastewaters.[21,28,29] More complex short- and long-range dispersion models have also been developed to understand and manage the airborne spread of highly contagious livestock diseases.[30−33] Most atmospheric dispersion models have been developed to address concerns from gaseous and particulate pollutants, but their use to model bioaerosol dispersion has generally been conducted without validation, except in a few cases.[28] Because of the labor intensive methods required to monitor airborne microorganisms,[17] validation of dispersion models can be difficult and is often not realistic. Despite this potential limitation, dispersion models can be a useful and cost-effective tool, especially if long-term monitoring is required for a large number of sources.

Recently, several researchers have used atmospheric transport models to estimate the dispersion of airborne pathogens during land application events of biosolids.[26,34] Pathogen dispersion data from these studies was then applied to a quantitative microbial risk assessment (QMRA) approach to estimate occupational and public infectious risks via inhalation exposures. A QMRA is composed of four basic steps: (1) hazard identification, (2) exposure assessment, (3) dose−response, and (4) risk characterization. Using this approach, the objective of this study was to perform a QMRA to estimate the risk of human infection from the inhalation exposure and subsequent ingestion of bacterial pathogens (*Campylobacter jejuni*, *Escherichia coli* O157:H7, non-O157 *E. coli*, *Listeria monocytogenes*, and *Salmonella* spp.) aerosolized during center pivot irrigation of dairy wastewaters. Four irrigation scenarios and associated pathogen emission rates were developed on the basis of available information. The dispersion of select bacterial pathogens found in dairy wastewaters was modeled using the steady-state Gaussian plume model, AERMOD.[35] After a sensitivity analysis was conducted on model inputs and settings, the infectious risks were calculated using modeled airborne pathogen concentrations. This assessment considered risk of infection to nonimmunocompromised residential populations that were 1 to 10 km from a center pivot that was spraying diluted dairy wastewater, which is the first QMRA to address such an issue. This QMRA provides a reasonable starting point to evaluate public infectious risks (incidental exposure) associated with land application of livestock wastewaters.

EXPERIMENTAL SECTION

Dispersion Model Setup. The dispersion model AERMOD[35] was operated using the graphical interface BEEST for Windows (Oris Solutions, Austin, TX, USA). An area source that was 396 m × 15 m was used to mimic the droplet pattern produced by a center pivot with sprinklers mounted 3 m above ground level. Sprinklers mounted at 3 m is typical for the irrigation of silage corn. Discrete receptors were placed in a polar grid with rings at 1, 2, 3, 4, 5, 7, and 10 km from the midsection of the source with 10 degrees of separation between receptors. Therefore, each polar ring contained 36 receptors, with a total of 252 for the complete grid. The center of the field being irrigated in each scenario was located at UTM coordinates 296581 m E and 4735519 m N (zone 12T) in Minidoka, Idaho, at an elevation of 1305 m above sea level.

A five-year meteorological data set (2000−2004) of hourly readings for use in AERMOD was prepared using AERMET[36] on available surface and upper air meteorological data. Surface data were obtained from a meteorological tower maintained by the Idaho National Laboratory in Minidoka, Idaho. Missing onsite data were supplemented with surface observations from the National Weather Service (NWS) station in Burley, Idaho, which is 32 km to the south. The winds were predominantly from the west, west-southwest, or east with an average annual wind speed of 4.4 m s^{-1} and calm conditions occurred <0.12% of the time (Figure S1, Supporting Information). Upper air data were obtained from the NWS site in Boise, Idaho. Missing upper soundings were substituted with available model-generated data nearest Boise using the Pennsylvania State University/National Center for Atmospheric Research mesoscale model, MM5 (http://www.mmm.ucar.edu/mm5/).

Surface characteristic values for AERMET, including albedo, Bowen ratio, and surface roughness length, were calculated for the area surrounding Minidoka using AERSURFACE[37] along with the USGS 1992 National Land Cover Data set.[38] Seasonal surface roughness values were calculated for twelve 30 degree sectors within 1 km of the Minidoka meteorological tower. A summary of the AERSURFACE results is presented in Table S1, Supporting Information.

Irrigation Scenarios, Pathogen Emission Rates, and Assumptions. On the basis of information obtained from peer-reviewed literature, government reports, and direct communication with local dairymen, pathogen emission rates (cells s^{-1}) for use in AERMOD were calculated as presented in Table 1. Four scenarios of low, medium, high, and very high risk (i.e., A, B, C, and D, respectively) were developed to account for a wide range of pathogen emission rates that may occur during the center pivot irrigation of dairy wastewaters. The hypothetical 396 m long center pivot (with no end gun) was assumed to have 94 flat plate sprinklers (34.2 L min^{-1}

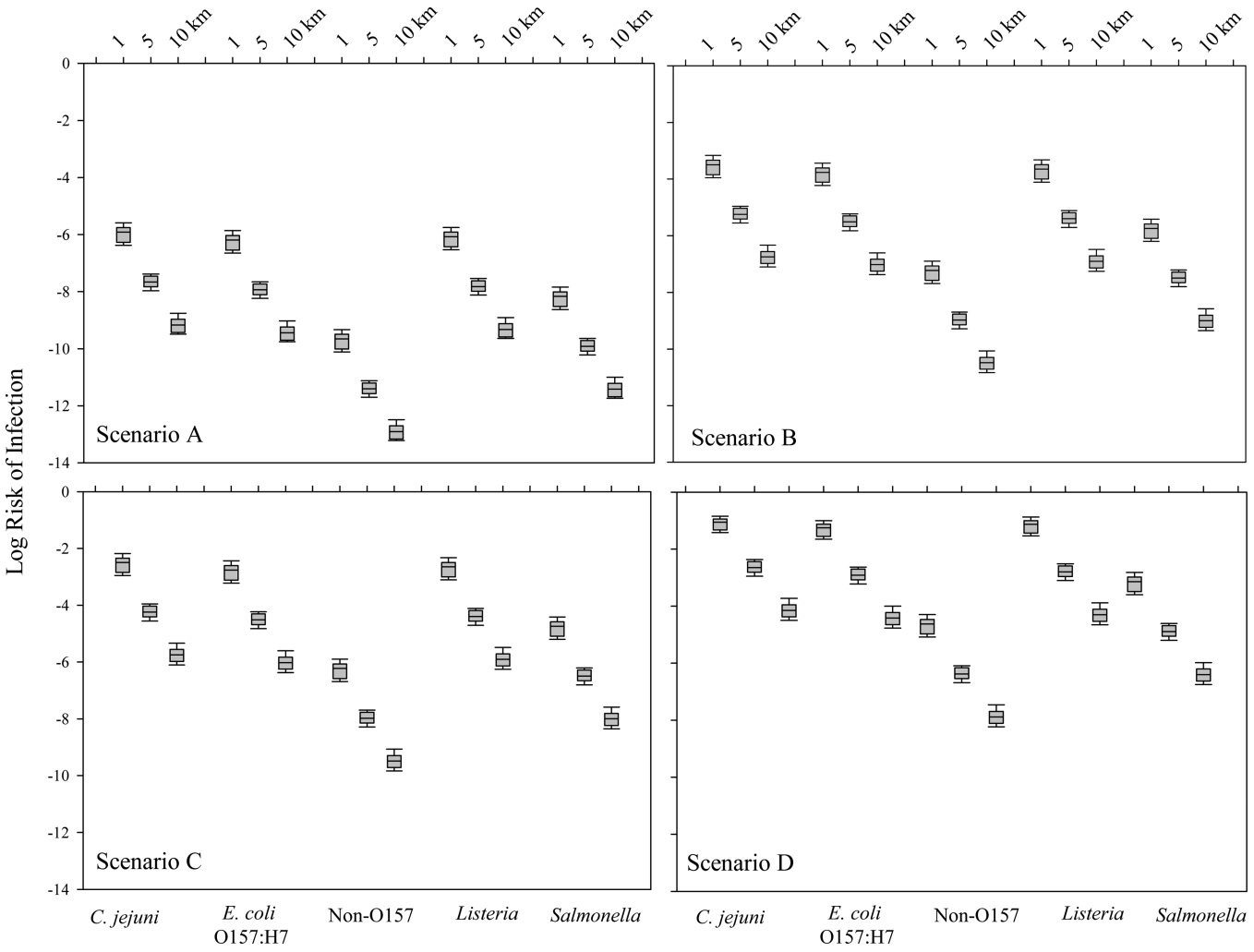

Figure 1. Log probability of infectious risk from bacterial pathogens associated with aerosols emitted during center pivot irrigation of dairy wastewater. The risk estimates, as determined using the β-Poisson dose−response model, were based on a one-time exposure of 1 h, first highest 3 h concentrations from AERMOD runs using meteorological data from 2004 (April to Oct), and accounted for a low rate of pathogen inactivation, where $\lambda = 0.002$. The horizontal lines in the box plots, from bottom to top including the whisker caps, represent the 5th, 25th, 50th, 75th, and 95th percentiles.

sprinkler^{-1} at 138 kPa) and discharge a total of 3217 L of irrigation water min^{-1} with uniform application occurring under all pivot sections. Data from the literature indicates that drift losses are greatest from flat plate sprinklers due to the formation of smaller droplets;[39−41] thus, one can infer that increased aerosolization of microorganisms might also occur under these conditions.

The sprinkler impact factor (I) was set to zero, as previous research by our laboratory demonstrated little or no effect of pressure and spray plate type on postsprinkler culturable microorganism concentrations.[42] Pathogen levels were set to values (i.e., 10^3 to 10^6 cells 100 mL^{-1} of wastewater) as determined in a study of dairy wastewaters in Idaho.[3] Wastewater dilution rates were set at 5%, 10%, or 20% (v/v), with the latter value being the greatest percentage of wastewater that is typically applied through center pivot irrigation systems. Aerosolization efficiency (E), which is the fraction of total water sprayed that leaves the vicinity of the irrigation system as dry or semidry aerosols, was set to encompass values (i.e., 0.1% to 3%) in the literature.[29,43−45] Using Rhodamine dye as a tracer, values for E were determined to range from 0.08% to 2.7% (median, 0.33%) during irrigation runs where impact sprinklers were utilized.[29]

Given the uncertainty associated with some parameters, the following assumptions were applied: (1) dispersion behavior among all pathogens was similar; (2) all bioaerosol particles were inhalable at ≤100 μm in aerodynamic diameter; (3) due to the arid climate of Idaho, only dry deposition was considered; (4) aerosol density was 1.1 g cm^{-3} with an even mass fraction of particles 1 to 100 μm; (5) inactivation of airborne pathogens occurred; and (6) aerosol age was based on an average wind speed (i.e., 4.4 m s^{-1}) during the irrigation season. Because AERMOD cannot be used to model moving sources, the center pivot was treated as a stationary source during the irrigation scenarios, but effect of directional orientation (i.e., north/south or east/west) on downwind bioaerosol concentrations was considered.

Sensitivity Analysis. AERMOD sensitivity was investigated to find out how accurately input parameters and settings need to be specified in the model. The sensitivity analysis[46] was performed on the emission rate, release height, receptor (flagpole) height, concentration averaging period, highest value at each receptor, meteorological data sets, and center

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Table 2. Risk of Infection from Inhalation Exposures to Airborne Bacterial Pathogens in Residential Populations 1 km Downwind from a Center Pivot Spraying Diluted Dairy Wastewater[a]

		log risk of infection[c]											
		duration of one-time exposure events									multiday exposure event[d]		
		1 h			8 h			24 h			7 days		
organism	scenario[b]	min	med	max	min	med	max	min	med	max	min	med	max
Campylobacter jejuni	A	−13.2	−12.6	−12.2	−12.3	−11.7	−11.3	−11.8	−11.2	−10.8	−12.3	−11.8	−11.4
	B	−10.8	−10.2	−9.8	−9.9	−9.3	−8.9	−9.4	−8.8	−8.4	−9.9	−9.4	−8.9
	C	−9.8	−9.2	−8.8	−8.9	−8.3	−7.9	−8.4	−7.8	−7.4	−8.9	−8.4	−7.9
	D	−8.2	−7.6	−7.2	−7.2	−6.7	−6.3	−6.8	−6.2	−5.8	−7.3	−6.8	−6.3
E. coli O157:H7	A	−13.4	−12.9	−12.5	−12.5	−12.0	−11.6	−12.1	−11.5	−11.1	−12.6	−12.1	−11.6
	B	−11.0	−10.5	−10.0	−10.1	−9.6	−9.1	−9.6	−9.1	−8.7	−10.2	−9.6	−9.2
	C	−10.0	−9.5	−9.0	−9.1	−8.6	−8.1	−8.6	−8.1	−7.7	−9.2	−8.6	−8.2
	D	−8.4	−7.9	−7.4	−7.5	−7.0	−6.5	−7.0	−6.5	−6.1	−7.6	−7.0	−6.6
non-O157	A	–[e]	–	–	–	–	–	–	–	−14.5	–	–	–
	B	−14.5	−13.9	−13.5	−13.6	−13.0	−12.6	−13.1	−12.6	−12.1	−13.6	−13.1	−12.7
	C	−13.5	−12.9	−12.5	−12.6	−12.0	−11.6	−12.1	−11.6	−11.1	−12.6	−12.1	−11.7
	D	−11.9	−11.3	−10.9	−11.0	−10.4	−10.0	−10.5	−10.0	−9.5	−11.0	−10.5	−10.1
Listeria monocytogenes	A	−13.3	−12.8	−12.4	−12.4	−11.9	−11.4	−12.0	−11.4	−11.0	−12.5	−11.9	−11.5
	B	−10.9	−10.4	−9.9	−10.0	−9.5	−9.0	−9.5	−9.0	−8.5	−10.1	−9.5	−9.1
	C	−9.9	−9.4	−8.9	−9.0	−8.5	−8.0	−8.5	−8.0	−7.5	−9.1	−8.5	−8.1
	D	−8.3	−7.8	−7.3	−7.4	−6.9	−6.4	−6.9	−6.4	−5.9	−7.5	−6.9	−6.5
Salmonella spp.	A	–	–	−14.4	−14.5	−14.0	−13.5	−14.0	−13.5	−13.1	–	–	−13.6
	B	−13.0	−12.4	−12.0	−12.1	−11.5	−11.1	−11.6	−11.1	−10.6	−12.2	−11.6	−11.2
	C	−12.0	−11.4	−11.0	−11.1	−10.5	−10.1	−10.6	−10.1	−9.6	−11.2	−10.6	−10.2
	D	−10.4	−9.8	−9.4	−9.5	−8.9	−8.5	−9.0	−8.5	−8.0	−9.6	−9.0	−8.6

[a]Assuming a high rate of pathogen inactivation, where λ = 0.07. [b]A, B, C, and D represent low, medium, high, and very high risk scenarios, respectively. For complete details, see Table 1. [c]Risk of infection calculated using the first highest 3 h concentrations from AERMOD. [d]Assumes 1 h of daily exposure to aerosolized pathogens over a 7 d period; calculated using eq 4. [e]Risk of infection calculated to be near zero.

pivot orientation. A complete discussion of the results from the sensitivity analysis are presented in the Supporting Information.

Microorganism Die-Off Factor. To account for death of airborne microorganisms, the modeled pathogen concentrations at the receptors were corrected using the following eq 1:[29]

$$M_d = e^{-\lambda a_d} \quad (1)$$

where M_d is the microorganism die-off factor, λ is the viability decay rate (s^{-1}), and a_d is the aerosol age (s) calculated by dividing the downwind distance (m) by the average wind speed (m s^{-1}). To account for microbial inactivation that would occur during nighttime (low solar radiation, high humidity) and daytime (high solar radiation, low humidity) conditions, the decay rates used were 0.002 or 0.07 s^{-1}, respectively.[29,47−49]

Pathogen Dose and Risk Analysis. As described by Brooks et al.,[4] exposure to airborne pathogens was estimated using the following eq 2:

$$d = ec \times br \times t \times ag \quad (2)$$

where d is the number of pathogens dose^{-1}, ec is the airborne pathogen concentration (cells m^{-3} of air), br is the breathing rate (m^3 h^{-1}), t is hours of exposure, and ag is the aerosol ingestion rate. The breathing rate was set to 0.61 m^3 h^{-1}, as that is the volume of air inhaled by an average person (male and female combined, ≥ 21 years) in 1 h.[50] Because the dose−response model used in this study is specific to pathogen ingestion, a value of 0.1 was used for ag, which assumes that 10% of inhaled airborne pathogens was ingested into the intestinal tract.[51] After correcting for inactivation, each airborne pathogen was considered to be capable of causing infection in the downwind receptors.

The β-Poisson dose−response model was used to estimate the infection process for the bacterial pathogens.[52] The simplified version of the model, as used in this study, is presented as eq 3:

$$P_i = 1 - (1 + d/\beta)^{-\alpha} \quad (3)$$

where P_i is the probability of infection based on a one-time pathogen exposure, d is the pathogen dose as calculated in eq 1, and α and β are dose−response factors obtained from the literature (*C. jejuni*;[53] *E. coli* O157:H7;[54,55] non-O157 (enteropathogenic *E. coli* serogroups O55 and O111);[52] *L. monocytogenes*;[56] *Salmonella* spp.[52]). In cases where only the median infectivity parameter, N_{50}, was available, β was calculated using eq 4:

$$\beta = N_{50}/(2^{1/\alpha} - 1) \quad (4)$$

The probability of infection over a multiday event was determined using eq 5:

$$P_{ann} = 1 - (1 - P_i)^n \quad (5)$$

where P_{ann} is the probability of infection based on the number of days or events, n, per year. The multiday event was based upon 1 h of daily exposure to the airborne pathogens over a 7 d period.

RESULTS AND DISCUSSION

Risk of Infection from Inhalation Exposures to Airborne Pathogens. Figure 1 presents the log probability of infectious risk from inhalation exposure to bacterial pathogens aerosolized during spray irrigation of diluted dairy wastewater. Accounting for a low rate of pathogen inactivation,

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Table 3. Risk of Infection from Inhalation Exposures to Airborne Bacterial Pathogens in Residential Populations 1 Km Downwind from a Center Pivot Spraying Diluted Dairy Wastewater[a]

		log risk of infection[c]											
		duration of one-time exposure events									multiday exposure event[d]		
		1 h			8 h			24 h			7 days		
organism	scenario[b]	min	med	max	min	med	max	min	med	max	min	med	max
Campylobacter jejuni	A	−6.5	−5.9	−5.5	−5.6	−5.0	−4.6	−5.1	−4.5	−4.1	−5.6	−5.1	−4.6
	B	−4.0	−3.5	−3.1	−3.1	−2.6	−2.2	−2.7	−2.1	−1.7	−3.2	−2.6	−2.2
	C	−3.0	−2.5	−2.1	−2.2	−1.6	−1.3	−1.7	−1.2	−0.9	−2.2	−1.7	−1.2
	D	−1.5	−1.1	−0.8	−0.8	−0.6	−0.5	−0.6	−0.4	−0.4	−0.7	−0.3	−0.1
E. coli O157:H7	A	−6.7	−6.2	−5.8	−5.8	−5.3	−4.9	−5.4	−4.8	−4.4	−5.9	−5.3	−4.9
	B	−4.3	−3.8	−3.3	−3.4	−2.9	−2.4	−2.9	−2.4	−2.0	−3.5	−2.9	−2.5
	C	−3.3	−2.8	−2.3	−2.4	−1.9	−1.5	−2.0	−1.4	−1.1	−2.5	−1.9	−1.5
	D	−1.7	−1.3	−0.9	−1.0	−0.7	−0.5	−0.7	−0.5	−0.4	−0.9	−0.5	−0.2
non-O157	A	−10.2	−9.7	−9.2	−9.3	−8.7	−8.3	−8.8	−8.3	−7.8	−9.4	−8.8	−8.4
	B	−7.8	−7.2	−6.8	−6.9	−6.3	−5.9	−6.4	−5.8	−5.4	−6.9	−6.4	−5.9
	C	−6.8	−6.2	−5.8	−5.9	−5.3	−4.9	−5.4	−4.8	−4.4	−5.9	−5.4	−4.9
	D	−5.2	−4.6	−4.2	−4.3	−3.7	−3.3	−3.8	−3.2	−2.8	−4.3	−3.8	−3.3
Listeria monocytogenes	A	−6.6	−6.1	−5.6	−5.7	−5.2	−4.7	−5.2	−4.7	−4.3	−5.8	−5.2	−4.8
	B	−4.2	−3.6	−3.2	−3.3	−2.7	−2.3	−2.8	−2.3	−1.8	−3.4	−2.8	−2.4
	C	−3.2	−2.6	−2.2	−2.3	−1.8	−1.4	−1.8	−1.3	−1.0	−2.4	−1.8	−1.4
	D	−1.6	−1.1	−0.8	−0.9	−0.5	−0.4	−0.6	−0.4	−0.3	−0.8	−0.4	−0.2
Salmonella spp.	A	−8.7	−8.2	−7.7	−7.8	−7.3	−6.8	−7.3	−6.8	−6.4	−7.9	−7.3	−6.9
	B	−6.3	−5.7	−5.3	−5.4	−4.8	−4.4	−4.9	−4.4	−3.9	−5.4	−4.9	−4.5
	C	−5.3	−4.7	−4.3	−4.4	−3.8	−3.4	−3.9	−3.4	−2.9	−4.4	−3.9	−3.5
	D	−3.7	−3.1	−2.7	−2.8	−2.2	−1.8	−2.3	−1.8	−1.4	−2.8	−2.3	−1.9

[a]Assuming a low rate of pathogen inactivation, where $\lambda = 0.002$. [b]A, B, C, and D represent low, medium, high, and very high risk scenarios, respectively. For complete details, see Table 1. [c]Risk of infection calculated using the first highest 3 h concentrations from AERMOD. [d]Assumes 1 h of daily exposure to aerosolized pathogens over a 7 d period; calculated using eq 4.

one-time infectious risk for a 1 h exposure event was highest for all pathogens under scenario D (very high risk), while the risk was estimated to be the lowest under scenario A (low risk). At 1 km in scenario D, the median risks for *C. jejuni*, *E. coli* O157:H7, non-O157, *L. monocytogenes*, and *Salmonella* spp. were approximately 10^{-1}, 10^{-1}, 10^{-5}, 10^{-1}, and 10^{-3}, respectively. Conversely, at a receptor distance of 10 km from the center pivot, median infectious risks were about 3 orders of magnitude lower. Under scenario A conditions at 1 km, respective median risks for the pathogens were estimated to be 10^{-6}, 10^{-6}, 10^{-10}, 10^{-6}, and 10^{-8}. This is a 5 order of magnitude decrease in risk compared to the same distance under scenario D conditions, where the pathogen emission rate from the center pivot was 1.2×10^5 times higher.

On the basis of a one-time pathogen exposure, the probability of infection (P_i) at 10^{-6}, for example, means that for every 1×10^6 times an individual is exposed to this dose, they will likely become infected once. For the purpose of this risk assessment, a conservative risk threshold of 10^{-6} was considered to be protective of public health. This value is not from current regulations but is based on the fact the 10^{-4} to 10^{-6} thresholds are commonly cited for infectious and lifetime cancer risks.[34]

Assuming a high rate of pathogen inactivation, the risk of infection for 1 h of exposure at distances of ≥2 km were calculated to be near zero and are not presented (1 km results are presented in Table 2). Additional infectious risk estimates for one-time and multiday exposure events (only 1 km from the center pivot) for low and high microbial die-off factors are presented in Tables 2 and 3, respectively. In both cases, compared to the one-time exposure event of 1 h, increasing the exposure times to 8 or 24 h generally led to an order of magnitude increase in risk, with the highest estimated risk for a 24 h event. For example in Table 2 under scenario A, the median risk of infection for *C. jejuni*, *E. coli* O157:H7, and *L. monocytogenes* was approximately 10^{-13}, 10^{-12}, and 10^{-11} for 1, 8, and 24 h exposure events, respectively. The risk of infection associated with the multiday exposure event (i.e., 1 h d^{-1} for 7 d) was similar to a one-time 8 h event for all bacterial pathogens.

With use of a high die-off factor for airborne pathogens (Table 2), the median infectious risks for the 8 h event were very low and ranged from 10^{-14} to 10^{-12} under scenario A and from 10^{-10} to 10^{-7} under scenario D. When the die-off factor was low (Table 3), the overall risk of infection was about 6 to 7 orders of magnitude higher than when high microbial die-off was assumed as presented in Table 2. For comparison, the median risk of infection for an 8 h exposure event ranged from 10^{-9} to 10^{-5} and 10^{-4} to 10^{-1} for scenarios A and D, respectively. Overall, risk of infection under scenarios B and C fell between scenarios A and D. Because of the specific infectious parameters used in the dose−response model, the pathogens that present the most risk regardless of irrigation scenario and exposure event were *C. jejuni*, *E. coli* O157:H7, and *L. monocytogenes*.

Understanding the Risks. In the dairy production environment, it is commonplace to land apply liquid and solid manures and wastewaters, thus potentially exposing downwind individuals to airborne pathogens. The exposures can occur through ingestion after airborne pathogens are deposited on fomites and food crops or possibly when aerosols are trapped in nasal mucosa and then ingested. The ingestion of

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contaminated produce was not considered in this QMRA since subterranean food crops (e.g., potato, sugar beet, onion) commonly grown in southern Idaho will not be exposed to airborne pathogens, while above ground crops (e.g., wheat, barley, dry beans) are processed which should theoretically minimize contamination. Although airborne pathogens could potentially contaminate exposed fruits and vegetables within a residential or community garden, this topic was deemed to be beyond the scope of this QMRA but should be addressed in future studies. Other than plant pathogens,[57] it is not known whether the airborne transmission of enteric pathogens has any role in the contamination of plants in the field.[58] Since the dairies and associated croplands are generally located away from residential communities and city centers, subsequent risk of infection after pathogen deposition on home garden crops is expected to be low. This assumption is supported by results from Hutchison and co-workers[59] who did not detect airborne *E. coli* at 250 and 500 m downwind from a rain gun spraying pig slurry. Even when the phylloplane of spinach and lettuce was directly irrigated with water containing high concentrations of *E. coli* O157:H7, *C. jejuni*, and *Salmonella*, the zoonotic pathogens were not detected after 2 to 3 weeks.

Regardless of the bioaerosol source such as animal feeding operations, composting facilities, municipal treatment plants, and biosolids (sewage sludge), manure, and wastewater land application sites, the general trend is that airborne microorganism concentrations have been shown to decrease with increasing distance from the source.[60] A decrease in bioaerosol concentration with distance is associated with a decrease in infectious risks from pathogens as determined by the results from this QMRA, which is also supported by risk assessments that considered aerosol generation and transport during land application of biosolids.[26,34,61] Dilution clearly plays a key role in reducing airborne microorganism concentrations, but meteorological factors such as temperature, solar radiation, and humidity can affect the viability of microorganisms during their dispersion. While there is very little pathogen-specific information, laboratory and field studies have shown that viability of airborne microorganisms generally decreases with increases in temperature and solar radiation or decreases in relative humidity.[62] Some viability decay rates (λ) have been determined by the U.S. EPA for fecal indicator organisms during spray irrigation studies of municipal wastewater.[29] Median decay rates for total coliforms, fecal coliforms, and coliphage were estimated to be 0.032, 0.023, and 0.011 s^{-1}, respectively. Teltsch et al.[47] determined λ for airborne *E. coli* to be 0.0088 and 0.066 s^{-1} during the early morning and afternoon runs, respectively. In aerosol chamber studies conducted by Paez-Rubio and Peccia,[49] respective inactivation rates for *E. coli* under high humidity conditions were estimated to be 0.002 and 0.02 s^{-1} during nonsolar and solar conditions. Because there are no known decay rates for airborne pathogens targeted in this QMRA, the selected λ values (i.e., 0.002 and 0.07 s^{-1}) were used for all pathogen die-off corrections even though they were originally determined for *E. coli*. Despite potential limitations with this approach, it is essential to account for some degree of inactivation that inevitably occurs with most airborne microorganisms. On the basis of an average wind speed of 4.4 m s^{-1} (from April to October, 2004), the aerosol age downwind of the center pivot was estimated to range from 3.8 to 37.9 min, allowing sufficient time for the airborne microorganisms to be affected by the ambient atmospheric conditions. Research with indicator organisms[63,64] suggests that downwind concentrations of viable airborne pathogens would be lowest during the day when wind speeds, temperature, and solar radiation are highest and relative humidity is lowest.

Risk of infection to the public from inhalation exposure to bacterial pathogens was only considered in this QMRA (Figure 1; Tables 2 and 3). Occupational infectious risks to dairy farm workers were not evaluated, since they were considered to be beyond the scope of this risk assessment. It was speculated, however, that it would be more likely to have an individual at a residential dwelling ≥1 km downwind from a center pivot operation, than for a farm worker to intentionally or accidentally subject themselves to land applied wastewaters and associated aerosols at anytime during the irrigation season. Although it is possible that wastewater application sites could be immediately upwind from a dairy operation, there are a number of complicating factors in determining infectious risks in dairy workers since they are also exposed to various nonaerosolized and aerosolized pathogens during handling of livestock and manures.[65−68] Occupational exposures are typically calculated assuming daily exposures for a working year; thus, infectious risks would certainly be higher when compared to public exposures. Once again, it is very unlikely that dairy farm workers would be exposed to airborne pathogens from center pivots spraying wastewater for such a prolonged period of time; this scenario would undoubtedly overexpress risk.

Many of the studies attempting to quantify microbial health risks (public and occupational) have been associated with land-applied biosolids,[26,34,61,69−71] not manures or wastewaters. Despite the fact that biosolids are generally dewatered, the results from these risk assessments should certainly be considered for comparative purposes. Risks were reported to range from low (10^{-12} per year) for consumption of root crops contaminated with *E. coli* O157 to high (10^{0} or 100%) for 8 h of continuous exposure to airborne coxsackievirus under high wind conditions 100 m downwind from a biosolids pile. Studies that have addressed inhalation exposures to various airborne pathogens (e.g., *Salmonella* spp., coxsackievirus, norovirus) found that infectious risks were highest in individuals immediately downwind (i.e., 5 to 100 m) of biosolids application sites,[26,34,61,70] whereas risks were substantially lower with increasing distance from the application sites. In a QMRA by Brooks and co-workers,[4] the greatest public and occupational one-time risks ($<10^{-1}$) were associated with intentional soil ingestion (pica child) and fomite exposures, respectively. At 100 m downwind from the land application of bovine, poultry, and swine manures, risk of infection to the public from aerosol exposures (1 h d^{-1} for 6 d) to *C. jejuni*, *E. coli* O157:H7, *L. monocytogenes*, and *Salmonella* spp. was estimated to range from 10^{-11} to 10^{-4}.

Infectious risk estimates from inhalation exposures to enteric pathogens in this QMRA, at 1 km from the center pivot (during high microbial die-off; Table 2), ranged from 10^{-15} to 10^{-6} for one-time (1, 8, and 24 h) exposures and 10^{-14} to 10^{-6} for a 7 day exposure event. The risk of infection was much higher (6 to 7 orders of magnitude) when low microbial die-off was assumed (Table 3). In all cases, the risk of infection was found to be the highest under scenario D, where the wastewater percentage, pathogen concentration, and aerosolization efficiency was the greatest. Clearly, risk to downwind individuals could be substantially higher if more than 20% wastewater (v/v) was used; a result of more pathogens being aerosolized

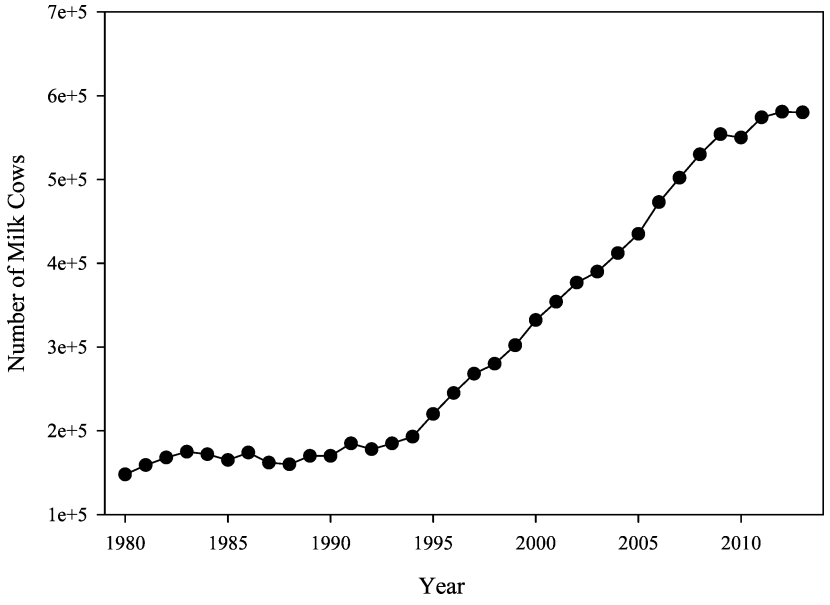

Figure 2. Annual number of milk cows in Idaho from 1980 to 2013 (Source: USDA, National Agricultural Statistics Service).

during irrigation events. The 20% wastewater value was chosen as an upper boundary level because it was determined to be most representative of irrigation practices by dairymen in southern Idaho. The results also indicate that increasing one's exposure time to the airborne pathogens increases the chance of infectious risk. However, because wind speed and direction are constantly changing, it is very unlikely that an individual would be exposed to some of the modeled airborne pathogen concentrations for an extended period of time (e.g., 24 h). Distance from the center pivot is also an important factor, as infectious risks were determined to be substantially lower at ≥5 km due to dilution and inactivation of the airborne pathogens at these extended distances (Figure 1). Once again, this only applies to when the microbial die-off factor was low, as the risk of infection at distances >1 km under high microbial die-off was estimated to be near zero (data not shown).

Risk Assessment Caveat. Although estimates from this QMRA do suggest that inhalation exposures to airborne bacterial pathogens from center pivots that spray diluted dairy wastewaters can potentially cause infection in downwind individuals, no empirical and clinical data were available for comparison. Therefore, validation of the dispersion model results and infectious risk estimates was not possible. Any attempt to validate bioaerosol measurements over such a large area (10 km radius), as simulated in the four scenarios, is certainly not feasible from a logistical standpoint. The difficulty in performing such a task would be further compounded by the many issues associated with capturing and quantifying airborne pathogens. Considering such complexities, it is understandable why models have been used to estimate the dispersion of bioaerosols under various situations.[28,33,72,73] As with most risk simulations, there is variability and uncertainty associated with many of the parameters, such as pathogen levels, viability, infectivity and dose, dose−response model, health status of affected populations, ambient environmental conditions, and exposure time. Accordingly, model inputs and settings were chosen through careful selection of data (from the literature and the author's personal research) and sensitivity analysis, respectively.

Although every effort was made to ensure that the infectious risk estimates did not underestimate actual risks in downwind individuals, the results should still be used cautiously for the reasons outlined above. For example, large variations in the wastewater pathogen concentrations could have a substantial influence on the estimated airborne concentrations and infectious risks at each receptor. It should be noted that the pathogen concentrations used in this study were taken from the best available data set for dairy wastewaters in Idaho. However, because the concentrations were determined via qPCR, the mere detection of a nucleic acid target does not mean the organisms were viable and capable of causing infection. Essentially all data and assumptions used in the simulations accounted for a wide value range to produce reasonable estimates, but determining if the final results are a realistic representation of actual infectious risks is not possible without model validation. While it is possible to assess variability and uncertainty of some data sets through the use of Monte Carlo analyses, applying a realistic range of values for a given parameter in the simulations should help to address these limitations. Some recently published risk analyses addressing biosolids land application scenarios have utilized this approach.[4,61,71]

Similar to the approach used in this paper, QMRA has been used to understand human health effects from exposures to biosolids and related pathogens released during land application.[26,71,74] Regardless of the context in which these exposures were being assessed, there is much uncertainty associated with inhalation of airborne pathogens and their ability to cause infection after subsequent ingestion. Additionally, there is little or no respective epidemiological-based evidence of dairy wastewater health effects on the public. Although it is possible to determine dose−response relationships for humans exposed to airborne pathogens from dairy wastewaters, there are a number of factors that would have to be overcome to generate accurate estimates, such as understanding the viability/infectivity of pathogens under a range of ambient environmental conditions and assessing susceptibilities across a representative population of humans. In the latter case,

the ethical issue of exposing humans to pathogens is certainly a large barrier that might prevent such dose−response studies.

Dairying and Public Health in Idaho. Since this QMRA is specific to wastewater applications in Idaho, a discussion of the dairy herd, manure production, and historical rates of disease for manure-borne pathogens is warranted. Idaho is currently the third largest dairy state in terms of milk production, following Wisconsin (no. 2) and California (no. 1), with a total of 580,000 lactating cows.[75] Since 1980, the total herd size in Idaho has increased 3.9-fold (Figure 2), which means that approximately 3.4×10^7 kg of manure is being generated on an annual basis (assuming 58 kg of manure lactating cow^{-1} d^{-1}). Most of the dairy production (~69%) is currently located in the south-central part of the state known as the Magic Valley. Because the amount of dairy manure and wastewater being land applied has also increased during the last three decades, public interest in the offsite transport of airborne fecal pathogens has increased, thus prompting the need for a QMRA to address this issue.

While not all disease incidents are diagnosed, trends in Idaho during 1987 to 2012 indicate that the rate of disease per 100,000 population for campylobacterosis and salmonellosis has remained largely unchanged and is below or near national rates.[76] The disease rate for Shigatoxigenic *E. coli* (O157:H7 and non-O157) in Idaho (8.7 per 100,000 in 2012) is above the national rate (2.3 per 100,000), and from 2001 to 2012, the annual rate fluctuated between 4 and 9 cases per 100,000 with no discernible trend. With respect to listeriosis, reportable disease incidents have occurred in 12 out of 26 years since 1987, with the total number of cases in Idaho ranging from 1 to 5 per year (national rate is 0.25 cases per 100,000). Despite some annual reporting of spikes in Idaho during the last two decades, rates of disease for giardiasis and cryptosporidiosis have tracked national rates since 2002 and 1998, respectively.

The Idaho public health records indicate that overall disease rates for fecal pathogens have declined or remained relatively stable over the same time period under which there was rapid growth of the dairy industry.

CONCLUSIONS AND RECOMMENDATIONS

This QMRA, which takes advantage of published scientific data, provides a useful starting point to understand and manage infectious risks associated with the airborne transmission of bacterial pathogens during center pivot irrigation of dairy wastewaters. It can also be used to simulate alternative irrigation scenarios and be expanded to accommodate other routes of transmission, as well as be refined. To reduce uncertainty associated with the risk estimates, generation of data that can aid in its refinement should be pursued. Refinements or knowledge are required in the following areas: (1) aerosolization efficiency of pathogens during spray irrigation of wastewaters; (2) inactivation and deposition rates for airborne pathogens under various environmental conditions; (3) inhalation transmission and dose−response of enteric pathogens in humans; and (4) exposure frequency and duration of affected populations. While not assessed in this QMRA, variability and uncertainty of future and improved data sets and associated model outputs should be addressed (e.g., Monte Carlo analyses) where appropriate to improve confidence in the risk estimates.

During daytime applications of dairy wastewater, the simulations indicate that residential populations have a very low ($<10^{-6}$) to near zero risk of infection from the bacterial pathogens if they are located ≥1 km downwind from a center pivot. In contrast, infectious risks were dramatically higher if inactivation of airborne pathogens was assumed to be low. If infectious risks $>10^{-6}$ are not considered acceptable, then residential populations could be exposed to unsafe levels of airborne pathogens at distances of 1 to 10 km from a center pivot during nighttime applications of dairy wastewaters, though one must consider that this risk might be reduced or negated, as residents are likely to be indoors during night hours. Regardless, on the basis of the information presented in this QMRA, it is recommended that irrigation events of dairy wastewaters be scheduled to occur during daylight hours to reduce exposures of nearby residential populations to viable airborne pathogens. Wind speeds are typically higher during the day, which enhance dilution, and inactivation rates are higher as well due to desiccation and ultraviolet light exposure. Organic matter in wastewaters, however, may protect microorganisms from the latter items, which should be addressed in future research endeavors. Dairymen should also consider applying the lowest possible percentage of dairy wastewater through center pivots to minimize the number of pathogens that can be aerosolized. Ancillary information from the literature also suggests that application of wastewaters through sprinkler heads that produce larger droplets might limit the aerosolization of microorganisms.

ASSOCIATED CONTENT

Supporting Information

Table S1 is a summary of the AERSURFACE results, while Figure S1 shows wind rose plots for years 2000 to 2004 at Minidoka, Idaho. The text and additional figures detail the results of the AERMOD sensitivity analysis: Figure S2) modeled pathogen concentrations as influenced by a) averaging period and b) emission rate; Figure S3) influence of bioaerosol release height and receptor height on the pathogen concentrations; Figure S4) relationship between the pathogen emission rate and average concentration at the receptor rings; Figure S5) pathogen concentrations as influenced by the a) monthly and b) yearly meteorological datasets; Figure S6) pathogen concentrations as influenced by the highest value and a) averaging period and b) meteorological year; and Figure S7) influence of center pivot orientation on the average pathogen concentrations. This material is available free of charge via the Internet at http://pubs.acs.org.

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Notes

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The authors declare no competing financial interest.

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