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Quantitative microbial risk assessment (QMRA) of workers exposure to bioaerosols at MSW open dumpsites

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Abstract

The bioaerosol exposure data from the study by Akpeimeh, Fletcher, and Evans (2019) was used to compute the risk of infection from the exposure of dumpsite workers to *A. fumigatus* and *E. coli* O157:H7. A stochastic (Markov Chain) model was used to model the transport of the inhaled dose through the human respiratory system and then integrated into the beta-Poisson dose-response model to estimate workers risks of respiratory and gastrointestinal (GI) infection. The infection risk was computed based on workers exposure to *E.coli* O157:H7 at 10-50% pathogen ingestion rate and pathogen-indicator ratio (P:I) of $1{:}10^3$ and $1{:}10^4$, while exposure to *A. fumigatus* was based solely on the average initial exposure dose.

The results showed that after 11 hrs of exposure, workers engaged in scavenging, waste sorting and site monitoring were at risk of respiratory and GI infection in the magnitude of 10^{-1}. However, the risk estimates associated with specific areas of the dumpsite showed that, the risk of GI infection at the active area ranged between 3.23×10^{-3}-1.56×10^{-2} and 3.25×10^{-4}-1.62×10^{-3}; dormant area 2.06×10^{-3}-1.01×10^{-2} and 2.09×10^{-4}-1.04×10^{-3}; entrance 1.85×10^{-3}-9.09×10^{-3} and 1.87×10^{-4}-9.27×10^{-4}; boundary 1.82×10^{-3}-8.82×10^{-3} and 2.09×10^{-4}-8.94×10^{-4} for P:I=$1{:}10^3$ and $1{:}10^4$ respectively, while the risk of respiratory infection risks were in the magnitude of 10^{-1} for all four locations.

The estimated risk of workers developing respiratory and gastrointestinal infections were high for all activities assessed at the dumpsite.

Summary: MSW dumpsite workers are exposed daily to bioaerosols from dumpsite activities. Risk of respiratory and gastrointestinal infection from exposure was estimated using QMRA modelling. The result shows high infection risk of workers.

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KEYWORDS: Bioaerosols; *Aspergillus fumigatus*; *E. coli*; QMRA; Open dumpsite

1. INTRODUCTION

The public health and environmental hazards that result from the mismanagement of municipal solid waste (MSW) are a global issue that cannot be ignored. The most severely impacted are developing and transition countries where the rate of solid waste generation has been on the rise due to urbanization, but without corresponding infrastructure developments to treat such volumes of waste (UN-HABITAT, 2009). For instance, sub-Saharan Africa alone is estimated to generate 62 million tonnes of MSW per year, with a corresponding annual urban population growth rate of 2.27 percent per year, yet lacks a sustainable system of managing MSW (Akpeimeh et al., 2019; Hoornweg & Bhada-Tata, 2012). This results in the uncontrolled dumping of the excess MSW on open land areas, forming large waste hills over time known as open dumpsites. Open waste dumps are a major source of environmental pollution and a huge public health risk in vicinities where they are located. They generate heavy metals, polluting the soil and nearby water bodies; emit toxic chemicals such as dioxins due to uncontrolled burning; bioaerosols, organic dust and methane gas which is a potent greenhouse gas (Minh *et al.* 2003; Karakurt *et al.* 2012; Han *et al.* 2016; Akpeimeh *et al.* 2019; Vongdala *et al.* 2019). Respiratory diseases are one of the most commonly reported health symptoms by dumpsite workers and residents living near dumpsites, and have been attributed to exposure to aerosolized aetiological agents from these dumpsites (Ray *et al.* 2005; Garrido *et al.* 2015). Although a lot of information has been reported on the respiratory health impact from exposure to toxic particulate matter (Hamra et al., 2014; Kim, Kabir, & Kabir, 2015), reports exclusively associating respiratory disease to exposure to bioaerosols are limited. Moreover, empirical data supporting infection resulting from exposure to bioaerosols are scarce and only available for a few microorganisms (Haas, Rose, & Gerba, 2014). Thus the use of analytical models such as Quantitative Microbial Risk Assessment (QMRA) by Haas, Rose, and Gerba (1999) for the evaluation of public risk from exposure to bioaerosols have become widely accepted. The main advantage of QMRA is that it provides

63 researchers with readily available analytical models that can mimic the human response to
64 pathogen exposure without over reliance on existing animal models, which are expensive to run
65 and may have ethical implications.

66 QMRA as a mathematical model for evaluating risks associated with exposure to pathogenic
67 microbial agents have been widely used as an invaluable tool in decision and policy making in
68 the areas of food safety, recreational water safety and wastewater reuse (McBride *et al.* 2013;
69 Romero-Barrios *et al.* 2013; Pielaat *et al.* 2014). However, given the rise in global concerns about
70 infectious diseases (e.g SARS in 2003) and bioterrorism threats, government agencies and public
71 health experts have developed a keen interest in infection risk modelling and quantification of
72 exposure to aerosolised pathogenic microbial agents (Ksiazek *et al.* 2003; Bartrand *et al.* 2008;
73 Huang and Haas 2009). The QMRA framework is such that it utilizes mathematical models and
74 quantitative data to examine the exposure, characterize the spread of the pathogenic agents and
75 assesses the infection risk from such exposure. The four-tiered approach commonly used are
76 hazard identification (HAZ ID), dose-response assessment, exposure assessment and risk
77 characterization. The dose-response assessment phase in the QMRA model is the quantitative
78 yardstick for estimating infection risk. In previous studies of respiratory health risks from
79 bioaerosol exposure, most often, the average exposure dose were used in this phase to estimate
80 the workers risk of respiratory diseases from exposure to bioaerosols. However, in reality, when
81 bioaerosols considered infectious are inhaled, they are transported to specific regions of the lungs
82 and would have to be deposited for an infection to take place (Weir & Haas, 2011). Thus, the
83 average exposed dose does not account for the required particle transport through and losses in
84 initiating infection in the respiratory system. Bartrand et al. (2008) demonstrated that the host's
85 response to bioaerosol particle dose was a function of the particle diameter, leading to the need
86 to develop an effective dose-response model based on the understanding of this behaviour. Weir
87 and Haas (2011) attempted to model a physical system incorporating the Markov Chain

88 stochastic principles to estimated particle transport and deposition in the various stages of the
89 respiratory system based on the particle size. In this study however, ingestion of pathogenic
90 bacteria particles was coupled to the model by Weir and Haas (2011), further stretching the
91 applicability of the model to include gastrointestinal (GI) infections exclusively from swallowing
92 of particles deposited in the nasopharynx religion of the lungs.

93 The data on bioaerosol concentration used in this study has already been published in a previous
94 report by Akpeimeh et al. (2019). They reported the ambient concentration for total bacteria,
95 gram-negative bacteria and *Aspergillus fumigatus* at Olusosun open dumpsite, Nigeria. The
96 dumpsite workers were reported to be exposed to bioaerosols at concentrations up to 10^6 cfu m^{-3}
97 depending on the activities they were involved in. These workers spent on average 11 hours
98 daily on the dumpsite and would have been working on the dumpsite for 5 years (median). The
99 authors also reported that only 11% of the workers used nose mask at least twice during work in
100 the last 6 months prior to the study, while 89% used nose mask only once or not at all for the
101 same period. High prevalence of respiratory symptoms were also reported among the dumpsite
102 workers, and was attributed to the prolong exposure to aetiological agents including bioaerosols.
103 Because these respiratory symptoms was partly as a result of exposure to bioaerosols, it was
104 necessary to compute the probable health risk associated with such exposure by running a QMRA
105 with the dataset. It is worthy of note that hitherto, QMRA reports on bioaerosols isolated from
106 solid waste dumpsites either do not exist or are extremely scarce. As such, this study aims to
107 estimate the probable risk of infection of the dumpsite workers from exposure to gram-negative
108 bacteria and *Aspergillus fumigatus*.

2. METHODOLOGY

2.1 Markov Chain Model

A Markov chain model is a probabilistic tool that uses stochastic processes to model physical systems (Privault, 2013). Fig. 1 shows the schematics of the Markov chain applied in this study where the physical element in each region is represented as 'states' and the loss rates from each associated state is signified as λ. The loss rate (λ) is the function that describes the rate of change of the pathogen from state *i* to state *j*, or pathogens being removed from state *i* to state *j*. It can be seen that in this study the Markov chain model consists of 8 states. Described in order, the model starts from the nasopharynx region R_1 with the bulk fluid in state 1 (air) and deposition on the surface of the respiratory system in state 2 (deposition). As flow passes from R_1 to R_2 starting with the bulk fluid in state 3 (air) and deposition on surface of the respiratory system in state 4 (deposition). Then from R_2 to R_3 starting with the bulk fluid in state 5 (air) and deposition on the surface of the respiratory system in state 6 (deposition). Inactivation of the pathogen from natural causes is defined as state 7 (applicable to R_1, R_2 and R_3) and exhalation is state 8.

2.1.1 *The Markov Transitional Matrix*

The Markov transition probability matrix (P) (e.q 1) contains probabilities (*p*) that predict the transitioning of the pathogens from one state to another, either within the same region or to another region of the respiratory system. Consider an inhaled pathogen in state *i* (air), in the next time step Δt, the pathogen has an unconditional probability of remaining in the same state *i*, denoted as p_{ii} and an unconditional probability of transitioning to another state *j*, denoted as p_{ij}. The sum of p_{ij} (j = 1, 2…, 8) equals one. Equation 1 shows the first order transition probability matrix **P** for the system in Fig. 1. The values of p_{ij} are entered with each row representing a state in the system. The zero entry, i.e. $p_{ij} = 0$, signifies that the pathogen cannot move between the two states in one-time step (1 min), e.g. P_{51}, P_{36}. For absorbing states such as states 7 and 8, $p_{ij} = 1$.

Furthermore, considering the Markov chain at time zero, a pathogen is introduced into the state *i*, and after n × Δ*t* time steps, the probability that the introduced pathogen is in state *j* at n × Δ*t* is the entry in *i*th row and *j*th column of **P** multiplied by itself *n*th times. The probability is designated p_{ij}^{n}, while the latter matrix is designated as $\mathbf{P}^{(n)}$, with *n* being the number of multiplications.

$$\mathbf{P} = \begin{bmatrix} p_{11} & p_{12} & p_{13} & 0 & 0 & 0 & p_{17} & p_{18} \\ 0 & p_{22} & 0 & 0 & 0 & 0 & p_{27} & 0 \\ p_{31} & 0 & p_{33} & p_{34} & p_{35} & 0 & p_{37} & 0 \\ 0 & 0 & 0 & p_{44} & 0 & 0 & p_{47} & 0 \\ 0 & 0 & p_{53} & 0 & p_{55} & p_{56} & p_{57} & 0 \\ 0 & 0 & 0 & 0 & 0 & p_{66} & p_{67} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \end{bmatrix} \quad \textbf{[1]}$$

2.1.2 *Loss rates and Probabilities*

Given the sum all the loses from state *i* (λ_i), the probability of remaining in state *i* or p_{ii} is the exponential survival probability in eq. 1 (Nicas & Sun, 2006).

$$p_{ii} = \exp(-\lambda_i \cdot \Delta t) \quad \textbf{[2]}$$

Since the Markov chain model is based on a flow through the system, pathogenic particles that are not deposited and have survived inactivation in a previous region (e.g. from R1 to R2) are assumed to have moved to the next region. Hence, the unconditional probability of the pathogen transitioning from state *i* to state *j* in Δ*t* is the product of the probability that the pathogen in states *i* moves to *j,* i.e. (1- p_{ii}), and the ratio of the loss rates associated with transitioning from state *i* (λ_i) to state j(λ_{ij}), shown in eq. 3 (Weir & Haas, 2011).

$$p_{ij} = \frac{\lambda_{ij}}{\lambda_i} \cdot [1 - p_{ii}] \quad \textbf{[3]}$$

Where $\lambda_i > 0$. If $\lambda_i = 0$, state *i* is an absorbing state and $p_{ij} = 0$ for $i \neq j$

The loss rate associated with inhaled pathogens moving deeper into the respiratory system from a region of higher R_x air volume to lower R_y, is generalised in eq. 4 (Weir & Haas, 2011).

$$\lambda_{xy} = \frac{Q + B}{V_{R_x}} \quad \textbf{[4]}$$

Where V_{R_x} = the volume of the higher region (cm^3), λ_{xy} = the loss of a spore in the higher region transitioning from region *x* to region *y*, *Q*= the volumetric flow rate of the inhaled air and *B* = the volumetric flow rate of exhaled air. Both *Q* and *B* are assumed to be constant throughout the lungs (i.e. inflow is equal to outflow) and has the value of 125 cm^3 min^{-1} (Weibel, Cournand, & Richards, 1963).

The loss rate associated with spores transitioning from lower regions to the higher regions of the respiratory system via exhalation, is expressed in eq. 5

$$\lambda_{yx} = \frac{B}{V_{R_y}} \quad \textbf{[5]}$$

Where V_{R_y} = the volume of the lower region, λ_{yx} = the loss of a spore in the lower region transitioning from region *y* to region *x*.

Bulk transport or phagocytosis is the main mechanism for the loss of pathogens in the human body, including the respiratory system (Clarke et al., 2010). In addition to phagocytosis, deposition can occur on the respiratory system surface. The resuspension of the deposited pathogens is prevented by mucociliary escalators, and they are eventually expectorated within 12 h (Koblinger, 1985). The loss due to deposition is accounted for by impaction, sedimentation and diffusion (Weir & Haas, 2011). For sedimentation, the rate is determined by the terminal settling velocity of the particle (v_{ts}) and is expressed in eq. 6

$$v_{ts} = 0.0018 \cdot d_{\mathrm{p}}^2 \cdot \left[1 + \frac{0.166}{d_{\mathrm{p}}}\right] \quad \textbf{[6]}$$

Where d_p is the particle size and hold accurate for particle up to 50 µm in diameter.

Therefore, the loss of pathogen from deposition accounting for sedimentation, impaction and diffusion can be estimated in eq. 7

$$\lambda_{deposition} = \frac{v_{ts}}{d_{R_x}} + DI_{R_x} \quad \textbf{[7]}$$

Where: DI_{R_x} = diffusion deposition rate in associated region, d_{R_x} = diameter of the associated region. The estimated values of the loss rates in the Markov chain model and the physiological parameters for humans used in the computation are found in S1 Table and S2 Table in supporting information.

2.1.3 Effective dose from inhalation

Effective dose is the fraction of the viable pathogens that would have been deposited on the target organ, survived inactivation and has the potential of germination that results in infection. Once the probabilities of the transition matrix P (eq. 1) were assigned, the estimate of the viable pathogens in any given state at time Δt is the product of the sum total of the probabilities associated with that state in each time step as seen in eq. 8

$$E[D_i] = N_i \cdot \sum_{n=1}^{\infty} p_{ij}^n \quad \textbf{[8]}$$

Where n is the number of multiplications associated with the time step in the model, Ni = initial pathogen load either transitioned or remaining in the same state.

Subsequently, the initial pathogen load for the next state or region in turn equals the effective dose E [D_e] of the previous state or region. For example, in order to compute the effective dose of the particle deposited in the surface at state 6, let's consider E [D_1] which denote viable pathogens in state 1 (Air), E [D_3] denotes viable pathogens in state 3 (Air), E [D_5] denotes viable

pathogens in state 5 (Air) and E [D_6] denotes viable pathogens to state 6 (Respiratory surface), the doses are quantified as follows:

$$E\,[D_1] = N_1 \times (p_{11}^n + p_{13}^n) \quad \textbf{[9]}$$

$$N_2 = N_1 \times p_{12}^n \quad \textbf{[10]}$$

$$E[D_2] = N_2 \times p_{22}^n \quad \textbf{[11]}$$

$$E\,[D_3] = E[D_1] \times (p_{33}^n + p_{35}^n) \quad \textbf{[12]}$$

$$E\,[D_5] = E[D_3] \times (p_{55}^n) \quad \textbf{[13]}$$

$$N_6 = E[D_3] \times p_{56}^n \quad \textbf{[14]}$$

$$E\,[D_6] = N_6 \times p_{66}^n \quad \textbf{[15]}$$

2.1.4 Effective dose from the swallowing of pathogens

The effective internal swallowed dose (d_i) was calculated from considering two major sources:

i. The estimated internal dose from particles with an aerodynamic diameter < 3.3 µm, which may be deposited in the Nasopharynx region of the respiratory system, i.e. E [D_2].

ii. The estimated internal dose of viable pathogens > 3.3 µm in diameter that may be deposited in the Nasopharynx region of the respiratory system, E_c. For gram-negative bacteria of this size range, it was assumed that all inhaled pathogen particles were deposited in the upper respiratory track or Nasopharynx region of the respiratory system.

The sum total of the inhaled dose *(d_i)* of viable gram-negative bacteria deposited in the Nasopharynx region can be estimated in eq. 16

$$d_i = E\,[D_2] + E_c \quad \textbf{[16]}$$

Where $E_c = ec \cdot \lambda_7$, ec being the initial exposure concentrations per day of particles with an aerodynamic diameter > 3.3 µm.

Fig. 2 shows a schematic representation of the GI infection pathway of inhaled particles that were eventually swallowed. The entrapped particles (or pathogens) on the surface of the respiratory system are prevented from resuspension by the actions of the mucociliary escalators, and they are eventually removed by expectoration or swallowed, with the latter increasing the gastrointestinal (GI) pathogen load (Koblinger, 1985; Pillai, 2007). The ingestion rate *ag*, is estimated to be between 10-50% of the inhaled pathogens (Medema *et al.* 2004; Brooks *et al.* 2005). Pathogen ingestion is accounted for by multiplying eq. 16 with ingestion rate *ag*, as shown in eq. 17:

$$d_{sw} = d_i \cdot ag \quad \textbf{[17]}$$

The effective gastrointestinal pathogen dose is expressed in the eq. 18:

$$E[d_{sw}] = d_{sw} - (d_{sw} \cdot \lambda_s) \quad \textbf{[18]}$$

Where d_{sw} is the ingested pathogen load; *ag* = ingestion rate (%); $E[d_{sw}]$ = effective gastrointestinal pathogen dose; λ_s (min^{-1}) is the rate of inactivation of *E. coli* from stomach acid (Lindqvist & Barmark, 2014).

The estimated values of the loss rates in the Markov chain model and the swallowing of *E. coli* used in the computation of the GI load can be seen in S1 Table and S2 Table (supporting information).

2.2 Dose-response (D-R) Assessment

The beta-Poisson dose-response model was used in this study because the model has been widely used from inhalation and ingestion of *Aspergillus fumigatus* and *E.coli* respectively (Teunis *et al.*

2008; Leleu *et al.* 2013; Dungan 2014). The dose-response assessment establishes a mathematically relationship between the inhaled pathogen dose and the probability of infection in exposed waste workers at Olusosun dumpsite. The beta-Poisson D-R model by Haas et al. (1999) was used to estimate the risk of infection from exposure to both respiratory and GI pathogens as described in eq. 19:

$$P_i = 1 - \left[1 + \left({}^{d_e}/_{\beta}\right)\right]^{-\alpha} \quad \textbf{[19]}$$

Where P_i is the probability of infection, d_e is the effective infective dose (either as $E\,[D_6]$ or $E[d_{sw}]$ for respiratory or gastrointestinal respectively), α and β are the slope parameters related to the pathogen, and their values can be found in Table I.

2.3 Risk characterization

The risk characterization combined the dose-response results and exposure information to estimate the magnitude of the risk to the exposed waste workers. The infection probability was calculated based on a one-time (one-minute), daily (11 hours/day) and annual exposure duration (Akpeimeh et al., 2019). A Pathogen to Indicator ratio (P:I) ranging from conservative 1: 1000 to a least conservative 1: 10,000 for the ratio of *E. coli* O157:H7 to gram-negative bacteria was used to calculate the infection risk from exposure to gram-negative bacteria. Brooks et al., (2005) used similar ratios in modelling of infection risks from aerosolized *Salmonella* spp. and coxsackievirus A21 from the spreading liquid biosolids. Risk combination using the inclusion-exclusion principle estimated the overall risk estimate in different scenarios combining several risk estimates.

2.3.1 Combining Risk

The mathematical principle of inclusion-exclusion was used to calculate the overall expected infection risk ($E[R]$) in any particular scenario. This assumes that infection can occur only once,

as described in eq. 20,21 and 22 for two, three and four risk combination respectively (Nicas & Sun, 2006):

$$E[R] = |R_A| + |R_B| - |R_A R_B| \quad \textbf{[20]}$$

OR

$$E[R] = |R_A| + |R_B| + |R_C| - |R_A R_B| - |R_A R_C| - |R_B R_C| + |R_A R_B R_C| \quad \textbf{[21]}$$

$E[R]$ = Overall expected risk, R_A, R_B, R_C, are the risk variables

2.4 Data analysis and model testing

The Markov chain model was developed as a steady state model. A one-minute time-step was used, as the model was expected to estimate pathogen deposition in human lungs based on the number of breaths taken per minute. The model was developed in MS Excel 2013 (Microsoft Inc.) and in R-project (by the R Foundation). The Monte Carlo simulation for the β- Poisson dose-response model was run on Minitab 18 statistical software. The Monte Carlo simulation was used to account for the natural variability in the model parameters and to reduce the level of uncertainty in the model results (Soller, Schoen, Bartrand, Ravenscroft, & Ashbolt, 2010). The technique works by sampling values at random from the probability distribution of the input data, in this case, the bioaerosols exposure data (Kottegoda & Rosso, 2008). Thus, it was important that, prior to running the Monte Carlo simulation, a goodness-of-fit test was conducted to determine the kind of distribution that best fits the input data for this study. A one-sample Kolmogorov-Smirnov (K-S) test was carried out on the bioaerosol exposure data to determine the distribution of best-fit for gram-negative bacteria and *Aspergillus fumigatus* (Sunger & Haas, 2015). The data for gram-negative bacteria was fit to a normal distribution (p = 0.59) and *Aspergillus fumigatus* fit to an exponential distribution (p = 0.49). Randomised data were subsequently generated based on the result of the one-sample K-S test for *Aspergillus fumigatus*

($E[D_6]$) and *E. coli* O157:H7 ($E[d_{sw}]$) and subsequently use to run the Monte Carlo simulation for β- Poisson dose-response model. The Monte Carlo simulation was run for 10,000 iterations and the median was considered to present the most likely scenario for estimating the infection risk.

3. RESULTS AND DISCUSSION

3.1 Risk of infection inherent to location on the dumpsite *(Aspergillus fumigatus)*

The results of the QMRA have shown the potential health risk of the poor microbial air quality at Olusosun dumpsite. The risk from the one-time exposure (1.4×10^{-5}) to *Aspergillus fumigatus* increased by 5-log (combined risk: 5.33×10^{-1}) after 11 hours of exposure from passive activities (e.g. Middlemen, visitors and small business owners) at the dumpsite (Table II). This implies that overall; there is at least a 53.3% chance of an individual involved in passive activities at the dumpsite to develop a respiratory ailment from inhalation of the spores of *Aspergillus fumigatus* from merely being present at Olusosun dumpsite for 11 hours. *Aspergillus fumigatus* is one of the common moulds present in the ambient air at compositing sites and landfill sites (Persoons, Parat, Stoklov, Perdrix, & Maitre, 2010; Schlosser, Robert, & Debeaupuis, 2016). Though the respiratory pathologies associated with the inhalation of its spores have been thoroughly investigated, the probable estimate of the risk of infection from inhalation of the spores have received limited attention. In this study, the results of the D-R model suggest that, based on the concentration of spores of *Aspergillus fumigatus* in the air samples, the risk to the individuals actively working on the dumpsite per day might be between 1.01×10^{-1} to 3.01×10^{-1}, which 1.24 times higher overall compared to those who are not involved in activities at the dumpsite (Table II). The differences between the two infection risk estimates (passive and active workers) was marginal, suggesting that the aetiology of the infection would be the same once the pathogen is inhaled whether or not people are active at the dumpsite.

306 Fig. 3 shows a trend that suggests an overall reduction in risk levels with distance from the active
307 area to the site boundary. Although the risk magnitude remained the same across locations i.e.
308 10^{-1}, the result otherwise suggests that workers working at the active area may be at greater risk
309 of infection from *Aspergillus fumigatus* than those located further away.

310 The combined infection risk indicates adjusted overall expected risk for Olusosun open dumpsite,
311 considering the risk levels inherent to each sampling location. Because the waste workers and
312 food vendors spend their time moving from one part of the dumpsite to the other during the day
313 (11-hour exposure), the minimum expected infection risk for these group of workers is the
314 combined risk of 5.90×10^{-1} (Annual risk = 9.58×10^{-1}). In other words, on the one-time pathogen
315 exposure, for every 10 times during the day they are exposed at the dumpsite, they will likely be
316 infected 6 times from inhaling spores of *Aspergillus fumigatus*. Owners of small businesses and
317 middlemen are usually stationed at the dormant area and the boundary, which are 'relatively'
318 lower risk compared to the active area where scavenging is the predominant activity. However,
319 by combining the inherent risk from each activity with their associated locations, the chances of
320 infection increases to the range of 66-78% (see S4 Table, supporting information). Taking the
321 dormant area as an example; the result of the combined risk for waste sorting (which is the
322 predominant activity) estimates the chances of infection at 68% and 90% as daily and annual
323 infection risk respectively, which is a 5 and 4 percentage points increase, assuming the individual
324 was not engaged in waste sorting at the dormant area. The trend suggests that the kind of activity
325 undertaken at the dumpsite can play an important role in heightening the risk of infections for
326 the workers irrespective of the location they take place.

327 ### 3.2 Risk of GI infection inherent to location on dumpsite (*E.coli* O157:H7)

328 The risk of GI infection from an 11-hour exposure to bioaerosols containing *E.coli* O157:H7 at
329 the active area was only 1-log greater than the boundary for P: I = $1:10^3$ and $1: 10^4$ (Table III).
330 The decrease in bioaerosol concentration with distance as reported by Akpeimeh et al. (2019)

may explain the decrease in GI infection risk from the results of the QMRA. A similar trend was observed by Dungan (2014), where the decrease in GI infection risk from enteric pathogen during land application of dairy wastewater was associated with the decrease in the concentration with distance, owing primarily to wind dilution. There are currently no guidelines for the acceptable risk threshold from exposure to aerosolized enteric bacteria in occupational environments, however, the range 10^{-6}-10^{-4} (conservative to a less-conservative) have been commonly cited in the literature for GI infection risk, and have been adopted in this study for comparison purposes (Dungan, 2014; Regli, Rose, Haas, & Gerba, 1991). Considering the results of the QMRA, only the estimates of GI infection risk for P: I = 1:10^4 were within acceptable limit (upper boundary). For individuals involved in passive activities at the entrance (1.31×10^{-4}- 6.57×10^{-4}) dormant area (1.46×10^{-4}- 7.32×10^{-4}) and the boundary (1.46×10^{-4}- 5.72×10^{-4}) would do so within the acceptable GI infection risk threshold. Furthermore, only individuals involved in active activities at the entrance (1.87×10^{-4}-9.27×10^{-4}) and the boundary (2.09×10^{-4}-8.94×10^{-4}) would do so within the acceptable GI infection-risk threshold. Furthermore, the data for P: I = 1:10^3 showing an 11-hour combined risk for all four sampling locations indicates that workers who are physically active (lifting, climbing the waste hill, pulling etc.; breathing rate = 17 breath per min) at the dumpsite will have a risk range of 8.93×10^{-3} – 4.29×10^{-2}, while the infection risk for those who are passively active (breathing rate = 12 breath per minute) will range from 6.19×10^{-3}-3.05×10^{-2} (Table III). Interestingly, the differences in the risk estimates for the two levels of activities is only marginal, thereby indicating that, not engaging in physical activities does not necessarily decrease the magnitude of the risk. Jahne, Rogers, Holsen, Grimberg, and Ramler (2015) reported a GI infection risk from *E. coli* O157:H7 aerosolized during manure application to be 10^{-3}-10^{-2} for an 8-hour exposure, values comparable to the prediction in this study. Although ranked as a medium-risk scenario, they however cautioned that that the risk level could easily escalate to high should there be any outbreak of *E. coli* O157:H7 from the sources feeding the point of exposure. A similar threshold (5×10^{-3}) was also reported by Seto, Soller, and Colford Jr (2007)

and Brooks, McLaughlin, Gerba, and Pepper (2012) to have caused the *E.coli* O157:H7 outbreak in 2006, with 205 reported illnesses and 5 death in the United States.

3.3 Risk of respiratory infection inherent to activities at dumpsite *(Aspergillus fumigatus)*

The annual respiratory infection risk inherent to activities like scavenging, waste sorting and site supervision are as high as 10^{-1} (Table IV). The result further indicates that by engaging in these activities in the active area (infection risk = 3.01×10^{-1}), the risk of infection increases by 3.11×10^{-1}, 3.16×10^{-1} and 3.70×10^{-1} points for scavenging, waste sorting and site monitoring respectively. The annual risk of respiratory infection from exposure to *Aspergillus fumigatus* during scavenging, waste sorting and site monitoring ranged from 7.93×10^{-1}-8.25×10^{-1}. For such estimates, it can be assumed based on a one-time exposure, for every 10 times the workers are exposed during the year to this dose at the dumpsite, they will likely become infected 8 times, especially those with suppressed immune systems. By implication, the workers are likely to be infected several times in a year from inhaling the spores of *Aspergillus fumigatus*. The risk estimates are very high considering that the workers are exposed 6 days per week and may be exposed to other pathogenic agents that may take a toll on their immune systems.

For healthy individuals, the inhaled spores are either removed by the mucociliary clearance mechanism or killed by the alveolar macrophages. Those that evade macrophage killings may germinate in the bronchioles or alveolar spaces; and at this point are targeted by infiltrating neutrophils capable of destroying their hyphae (Dagenais & Keller, 2009). The risk associated with developing any form of invasive aspergillosis is primarily the breakdown or dysfunction of the hosts defence system and the survival ability of the pathogen in the target growth environment of the hosts (Schaffner, Douglas, & Braude, 1982). Moreover, the combination of smoking and exposure to other aerosolized environmental pollutants can impair mucociliary clearance even in healthy individuals, thereby increasing the chances of deposition and possible growth of inhaled

spores of *Aspergillus fumigatus* (Wolff, 1986; Xavier et al., 2013). In the case of the study by (Akpeimeh et al., 2019) where 41% of the participants were smokers and 89% had never used nose masks for nasal protection during their work at the dumpsite, the respiratory risk estimates modelled in this study may be consistent with the reality of the respiratory health risk associated with working in environments such as Olusosun dumpsite.

3.4 Risk of GI infection inherent to activities at dumpsite (*E.coli* O157:H7)

Workers engagements in activities at the dumpsite, depending on the kind of activity, are at a high risk of GI infection, i.e. risk estimates higher than the inherent risk associated with the location where the activity took place. The risk of GI infection from scavenging at the active area (5.03×10^{-1}-6.63×10^{-1}) for example, is two-threefold greater than the inherent risk at the active area (3.23×10^{-3}- 1.56×10^{-2}) for the same exposure duration (Table III and V). A similar trend was also observed for the category of P:I = 10^4 where risk levels were higher by three-four orders of magnitude for scavenging, waste sorting and site supervision compared to the inherent risk levels at the active area where the sampling took place. Furthermore, the combined risk showed an even higher risk estimate overall than if the inherent risk for the locations and activity were measured as stand-alone (S4 Table, supporting information). Combining the risk of the active area and scavenging increased the overall adjusted risk by 2-3 orders of magnitude to 5.05×10^{-1}-6.68×10^{-1} for P:I=10^3 and 3-4 orders of magnitude to 2.10×10^{-1}-4.21×10^{-1} for P:I=10^4. The proximity of these activities to the exposure source and the reduced effect of dilution during these activities might explain the high-risk values in the dose-response model. Occupational risk studies accounting for enteric bacterial risk is very limited. Some notable exceptions are healthcare workers, wastewater treatment plant personnel and in concentrated animal feeding operations (CAFOs) (Medema *et al.* 2004; Bobo and Dubberke 2010; Brooks *et al.* 2012). Notably, Medema et al. (2004) reported the predicted annual risk from a wastewater treatment plant to be as high as 2×10^{-1} from a one-time exposure to enteric pathogens. Tanner et al. (2008)

on the otherhand, simulated an annual risk range of 2×10^{-2} (use of protective equipement) to 3×10^{-1} (no use of protective equiptment) during CAFOs. Brooks et al. (2012) reported similar risk values to Tanner et al. (2008), ranging from 1×10^{-2} to 5×10^{-1}, values comparable what is predicted in this study (Table V). As it currently stands, there are no epidemiological or clinical studies establishing the inhalation-ingestion route of transmission of enteric bacterial pathogens in humans (Brooks et al., 2012). This is because in most of the cases considered, there also exist faecal-oral route of transmission (from fomite, waterborne or foodborne) in the same environment. It is also worthy of note that because the detection procedure for the faecal-oral transmission has been established over time, it is common to ignore the inhalation-ingestion route of transmission. However, there is mounting evidence from animal trials that inhalation-ingestion routes of transmission exist and can pose a high risk of GI infection in a population exposed to aerosolized enteric bacteria (Clemmer *et al.* 1960; Fedorka-Cray *et al.* 1995; Darlowa *et al.* 2009).

3.5 Risk management options

3.5.1 Use of PPEs an RPEs

Workers at Olusosun dumpsite generally did not use personal protective equipment (PPE), including respiratory protective equipment (RPE) because they were expensive, and they could not afford them (Akpeimeh et al. (2019). This reflects the economic status of the workers, as most of the recycled materials are sold to intermediaries at cheap rates; barely enough to cover their daily upkeep let alone afford a personal protective equipment. To this end, intervention by the authorities is necessary to protect the health of the workers. PPE's should be subsidised for the workers, and the workers monitored for effective usage of the PPE's. Routine, but compulsory respiratory health checks (however rudimental) should be carried as part of the requirement to work on the dumpsite. This will help the authorities keep on top of the health conditions of the workers and incentivise record keeping.

3.5.2 Reduction of the number of waste scavengers in dumpsites

Scavengers composed of the highest proportion (61%) of workers at Olusosun dumpsite (Akpeimeh et al. (2019). By the nature of their activity, they are the most exposed to bioaerosols and have the

highest risk of getting infected. It is therefore recommended that by systematically reducing the number of scavengers picking at the dumpsite, it is possible to reduce the overall health impact on population at the dumpsite. If city authorities implement programmes that reduces to the barest minimum the amount of recyclables reaching dumpsites, the population of scavengers on the dumpsite will consequently reduce. The UK's waste hierarchy for example is core to the waste directive (Directive 2008/98/EC), which prioritizes waste prevention, then re-use, then recycling, then recovery and last of all, disposal (e.g. in landfill). Another example is described by Asim, Batool, Chaudhry, and Recycling (2012) where informal recyclers are integrated into the mainstream of the waste management system of Lahore city, Pakistan. They go door-to-door collecting household recyclable waste, and then take them to waste transfer points across the city where itinerant buyers buy the waste at higher value than they would at the dumpsite. Moreover, the approach of using local expertise (like above) to proffer sustainable low-cost solutions to solid waste management problems will directly or indirectly impact positively on the social-economic status of the people in that society (Zurbrügg, Gfrerer, Ashadi, Brenner, & Küper, 2012). Conclusively, the informal waste workers will earn more money from their enterprise while reducing exposure to pathogens and improving their overall health.

3.6 Research limitations

In carrying out this research, there were sources of uncertainty inherent to the simulation such as the sample collection, effective dose dose-response model and the population type and these may have cascaded through the model, widening the 'cone of uncertainty' through the various steps of the modelling process. Firstly, the method of sample collection was a potential source of uncertainty in the risk calculation, as *E.coli* O157:H7 was not originally isolated in the air samples at the dumpsite. However, one of the approaches used to address this was to assume a pathogen-indicator ratio in the exposure dose. This approach has been applied by Brooks et al. (2005) representing the risk estimate as a range of values of the pathogen doses and this was adopted in this study. Secondly, because inactivation rates vary by microbial specie and the

459 environment, applying the same inactivation rates for both indicator microorganisms as used in
460 this study, may have increased the uncertainty in the model. However, the use of a Monte Carlo
461 simulation to estimate the natural variability of the indicator organisms as they are inhaled
462 mitigates this uncertainty to some extent.

463 ## 4. CONCLUSION

464 The QMRA simulation presented here involved the first application of a stochastic model to
465 predict the transport of bioaerosols in the human respiratory system (Markov Chain Model), and
466 to estimate the risk of infection specific to dumpsite workers from the settlement of those
467 pathogens in the respiratory and gastrointestinal tracks. The overarching trends suggest that the
468 infection risk from inhaling contaminated air containing spores of *Aspergillus fumigatus* at all
469 locations were of the same magnitude (10^{-1}) irrespective of whether the individual was involved
470 in activities in the dumpsite or not. The combined risk of exposure from activities and ambient
471 exposure to *Aspergillus fumigatus* increases the daily chances infection. At the active area, the
472 risk of infection ranged between 73-78%, while at the boundary the range was 66-70% for all
473 activities associated with the locations. The daily estimates of the risk of infection from ingestion
474 of *E.coli* O157:H7 ranged from 10^{-3}-10^{-2} for the conservative and 10^{-4}-10^{-3} for the least
475 conservative pathogen to indicator ratio and was classified as a medium-high and low-medium
476 risk respectively. The probable outcome from ingesting inhaled *E.coli* O157:H7 during
477 scavenging, waste sorting and site monitoring was high (10^{-1}), with similar magnitude
478 comparable to the annual infection risk.

479 Overall, the trends in the risk estimates suggest that the activities at the dumpsite may contribute
480 more to the likelihood of workers developing either respiratory infection or GI infection than any
481 other factor.

Conflict of Interest

Each named author has contributed substantially to conducting the underlying research and drafting this manuscript. Additionally, to the best of our knowledge, the named authors have no conflict of interest, financial or otherwise.

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Ethics approval

Ethical approval was obtained from the Engineering Faculty Research Ethics Committee, University of Leeds, UK.

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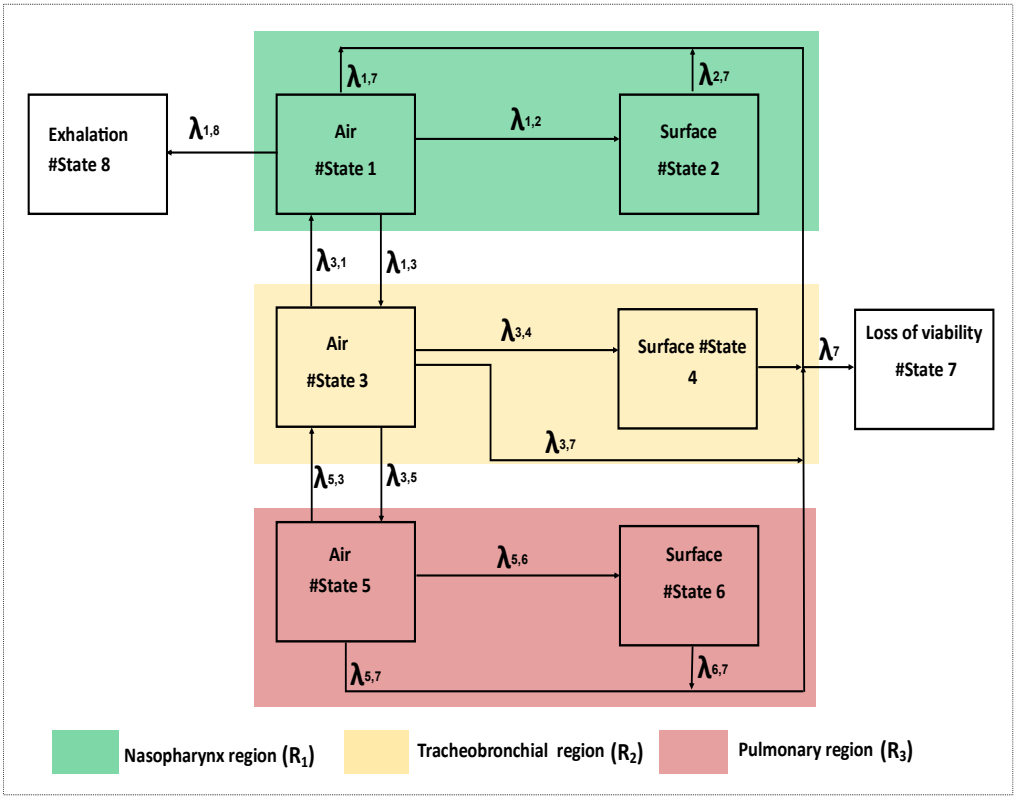

Fig. 1. Schematics showing the connection between the eight states in the Markov Chain model used to model transport and deposit in the respiratory system

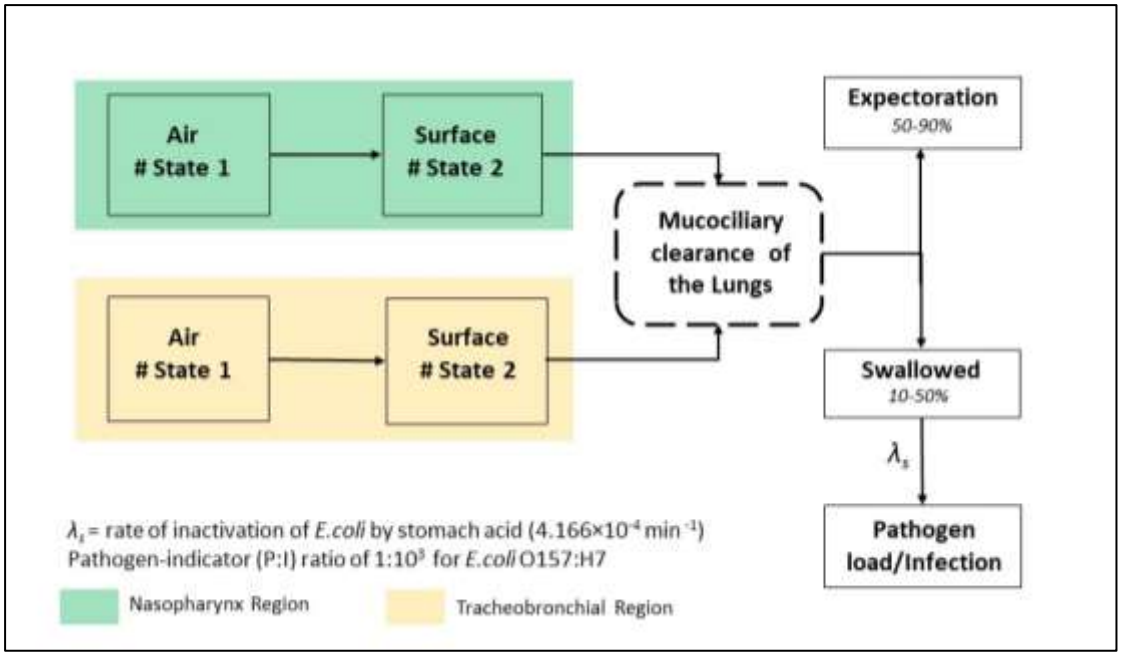

Fig. 2. Schematic representation of the GI infection pathway

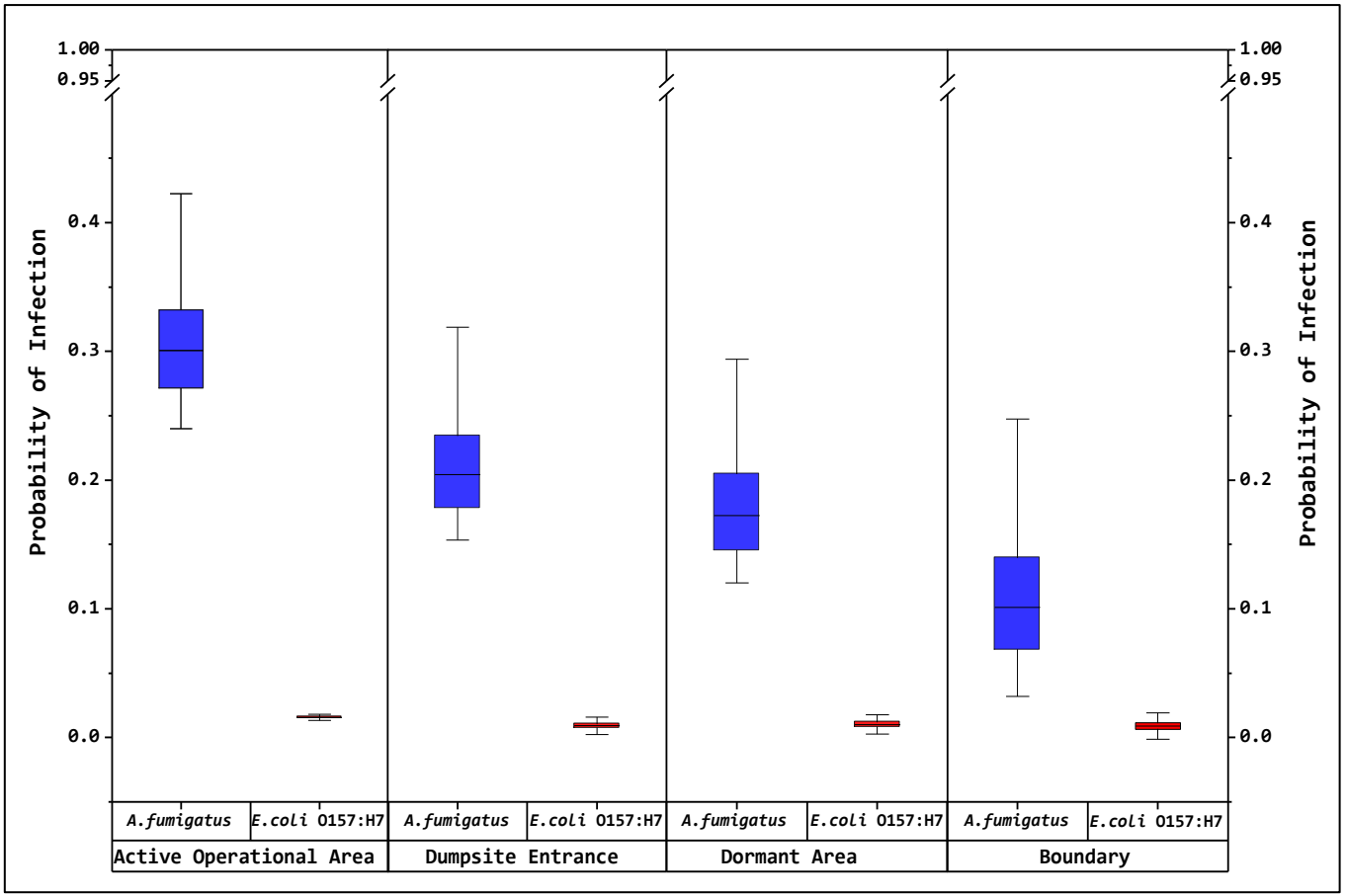

Fig 3. Eleven-hour risk of infection from bioaerosol containing *Aspergillus fumigatus* and *E. coli* O157:H7 from the four sampling locations at Olusosun dumpsite. Boxplots indicates upper/lower quartiles and median; Whiskers indicates 95th percentiles.

Table I Slop parameters used in the beta-Poisson D-R model and assumptions

Pathogen	D-R Model	Parameter	Conditions of development	References
A. fumigatus	β-Poisson	$\alpha = 1.1,\ \beta = 20$	Developed from animal model of immunosuppressed mice.	Leleu et al. (2013)
E.coli	β-Poisson	$\alpha = 0.248,\ \beta = 48.8$	Developed from fitting data from 8 out breaks from *E. coli* O157:H7	Jahne et al. (2015); Teunis et al. (2008)

Table II: Risk of infection (median) from inhalation of spores of *Aspergillus fumigatus* at the four sampling locations

Variable	Risk of infection	
	11 h	1 year*
Risk associated with active involvement at sampling location (Breathing rate = 17 breathe per min)		
Active Area	3.01×10^{-1}	6.27×10^{-1}
Entrance	2.04×10^{-1}	5.71×10^{-1}
Dormant Area	1.72×10^{-1}	5.50×10^{-1}
Boundary	1.01×10^{-1}	4.96×10^{-1}
Combined risk	5.9×10^{-1}	9.64×10^{-1}
Risk associated with passive involvement at the sampling location (Breathing rate = 12 breathe per min)		
Active Area	2.75×10^{-1}	6.12×10^{-1}
Entrance	1.77×10^{-1}	5.54×10^{-1}
Dormant Area	1.48×10^{-1}	5.34×10^{-1}
Boundary	8.12×10^{-2}	4.77×10^{-1}
Combined risk	5.33×10^{-1}	9.58×10^{-1}
One-time exposure (min^{-1})	1.4×10^{-5}	-

*Annual risk of infection based on exposure for 6 days per week for 52 weeks.

Table III: Risk of infection (median) from inhalation-ingestion exposure to *E.coli* O157:H7 at the four sampling location at Olusosun dumpsite

	Risk of infection for 10-50% ingestion rate			
	1:1000‡		1:10000‡	
Variable	11 h	1 year*	11 h	1 year*
Risk associated with active involvement at sampling location (Breathing rate = 17 breathe per min)				
Active Area	3.23×10^{-3}- 1.56×10^{-2}	3.32×10^{-1}-5.32×10^{-1}	3.25×10^{-4}-1.62×10^{-3}	8.16×10^{-2}- 2.41×10^{-1}
Entrance	1.85×10^{-3}- 9.09×10^{-3}	2.58×10^{-1}-4.68×10^{-1}	1.87×10^{-4}-9.27×10^{-4}	5.12×10^{-2}-1.75×10^{-1}
Dormant Area	2.06×10^{-3}- 1.01×10^{-2}	2.72×10^{-1}- 4.81×10^{-1}	2.09×10^{-4}-1.04×10^{-4}	5.64×10^{-2}-1.87×10^{-1}
Boundary	1.82×10^{-3} - 8.82×10^{-3}	2.56×10^{-1}-4.64×10^{-1}	2.09×10^{-4}-8.94×10^{-4}	5.01×10^{-2}-1.71×10^{-1}
Combined Risk	8.93×10^{-3} - 4.29×10^{-2}	7.32×10^{-1}-9.31×10^{-1}	9.32×10^{-4} -4.47×10^{-3}	2.19×10^{-1}-5.78×10^{-1}
Risk associated with passive involvement at the sampling location (Breathing rate = 12 breathe per min)				
Active Area	2.28×10^{-3}- 1.11×10^{-2}	2.86×10^{-1}- 4.90×10^{-1}	2.29×10^{-4}- 1.14×10^{-3}	6.09×10^{-2}- 1.99×10^{-1}
Entrance	1.31×10^{-3}- 6.46×10^{-3}	2.15×10^{-1}- 4.24×10^{-1}	1.31×10^{-4}- 6.57×10^{-4}	3.71×10^{-2}- 1.39×10^{-1}
Dormant Area	1.45×10^{-3}- 7.19×10^{-3}	2.27×10^{-1}- 4.43×10^{-1}	1.46×10^{-4}- 7.32×10^{-4}	4.11×10^{-2}- 1.49×10^{-1}
Boundary	1.15×10^{-3}- 5.73×10^{-3}	1.99×10^{-1}- 4.09×10^{-1}	1.46×10^{-4}- 5.72×10^{-4}	3.31×10^{-2}- 1.25×10^{-1}
Combined Risk	6.19×10^{-3}- 3.05×10^{-2}	-	6.52×10^{-4}- 3.11×10^{-3}	-
One-time exposure (min^{-1})	1.39×10^{-5}- 6.97×10^{-5}	-	1.40×10^{-6}- 7.00×10^{-6}	-

*Annual risk of infection based on exposure for 6 days per week for 52 weeks.

‡ Pathogen – indicator ratio at $1:10^3$ and $1:10^4$

Table IV: Risk of infection (median) from inhalation of spores of *Aspergillus fumigatus* during activities at the Olusosun dumpsite

	Risk of infection	
Exposure Activity	11 h	1 year*
Scavenging	6.11×10^{-1}	7.93×10^{-1}
Waste sorting	6.17×10^{-1}	7.96×10^{-1}
Site monitoring/ supervision	6.71×10^{-1}	8.25×10^{-1}

*Annual risk of infection based on exposure for 6 days a week for 52 weeks.

Table V: Risk of infection (median) from inhalation-ingestion exposure to *E.coli* O157:H7 during activities at Olusosun dumpsite

Exposure Activity	Risk of infection for 10-50% (low-high) ingestion rate (*ag*)			
	1:1000‡		1:10000‡	
	11 h	1 year*	11 h	1 year*
Scavenging	5.03×10^{-1}-6.63×10^{-1}	8.79×10^{-1}-9.19×10^{-1}	2.10×10^{-1}-4.20×10^{-1}	7.86×10^{-1}-8.56×10^{-1}
Waste sorting	4.54×10^{-1}-6.27×10^{-1}	8.66×10^{-1}-9.11×10^{-1}	1.63×10^{-1}-3.65×10^{-1}	7.62×10^{-1}-8.40×10^{-1}
Site monitoring/ supervision	1.89×10^{-1}-3.96×10^{-1}	7.76×10^{-1}-8.49×10^{-1}	3.04×10^{-2}-1.18×10^{-1}	6.05×10^{-1}-7.34×10^{-1}

*Annual risk of infection based on exposure for 6 days a week for 52 weeks.

‡ Pathogen – indicator ratio (P:I) at $1:10^3$ and $1:10^4$