Human Health Risk Assessment of Potentially Hazardous Agents in Land-Applied Sewage Sludge

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The impacts on human health and environment arising from the spreading of sewage sludge to land (CR/2016/23)

Human Health Risk Assessment of Potentially Hazardous Agents in Land-Applied Sewage Sludge

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List of Abbreviations

ADD – Average Daily Dose [mg kg^{-1} d^{-1}]

ARB – Antibiotic Resistant Bacteria

BBP - Benzyl butyl phthalate

BBN – Bayesian Belief Network

BCF – Bioconcentration Factor [unitless]

BFR – Brominated flame retardants

BPA – Bisphenol A

CPT – Conditional Probability Table

DEFRA – Department of Environment, Food and Rural Affairs

DEHP - Di(2-ethyhexyl)phthalate

ED – Expected Dose

H – Henry's Law constant [unitless]

Kow – Octanol:Water partition coefficient [unitless]

LASs - Linear alkylbenzene sulphonates

NOAEL – No Observed Adverse Effect Level

NOEL – No Observed Effect Level

OTs – Organotins

PAEs - Phthalate acid esters

PAH – Polyaromatic Hydrocarbons

PBDE - Polybrominated diphenyl ethers

PCAs – Polychlorinated alkanes

PCB – Polychlorinated Biphenyls

PCDD/Fs – Polychlorinated dibenzodioxins and furans (often termed “Dioxins and Furans”)

PCNs – Polycholrinated naphthalenes

PDMSs - polydimethylsiloxanes

PFCs – Perfluorochemicals

PFOA - Perfluorooctanoic acid

PFOS - Perfluorooctanesulfonic acid

PPCP – Pharmaceutical and Personal Care Products

QAC - Quaternary Ammonium Compounds

QRA – Quantitative Risk Assessment

RfD – Reference Dose [mg kg^{-1} d^{-1}]

SC – Steering Committee

SNIFFER - Scotland and Northern Ireland Forum for Environmental Research

SSRI – Selective Serotonin Reuptake Inhibitors

TCC – Triclocarban

TCPP - Tris (1-chloro-2-propyl) phosphate

TCS – Triclosan

TDCP - (Tris[2-chloro-1-(chloromethyl)ethyl]phosphate)

TEF – Toxic Equivalency Factor

TEQ – Toxic Equivalents

TPP - Triphenyl phosphate

UF – Uncertainty Factor

WRAP – Waste and Resources Action Programme

WWTP – Waste Water Treatment Plant

1. Introduction

Treated sewage sludge, also known as biosolids, has been beneficially recycled to agricultural land for many decades in the UK, Europe, the USA and other countries throughout the world. Recent data suggest that around 78% of the 3.6 million tonnes (fresh weight) of biosolids produced in the UK is currently recycled to agricultural land (Black et al., 2016). Biosolids are a valuable source of nitrogen and phosphorus, as well as other major and minor plant nutrients, and because of some conditioning processes have value as a liming material (SRUC, 2013). Biosolids also contain valuable quantities of organic matter and are therefore an important means of replenishing soil organic matter levels that can provide long-term benefits to soil structure and fertility. As such, the recycling of biosolids to land is recognised as being the best practicable environmental option by the European Union (EU) and UK Government in most circumstances.

However, partly due to complaints and concerns from communities reporting potential issues associated with the recycling of biosolids to land, a review of the existing legislation and procedures relating to the spreading of sewage sludge to land in Scotland was commissioned in 2014. This work was overseen by Scottish Government, SEPA and Scottish Water and brought forward several recommendations.

One key recommendation focused on the potential health impacts associated with sludge spreading and the lack of up to date, robust evidence. Most recently, a SNIFFER (Scotland and Northern Ireland Forum for Environmental Research) report in 2008 looked at human health and environmental impacts of the use of sewage sludge in land restoration and forestry and found no proven elevated health risks associated with the spreading of this material. The report also stated that incineration of sludge potentially presented “a higher risk to workers and the public.” The SNIFFER 2008 report did not however cover storage or spreading to agricultural land or include many emerging issues such as the presence and spread of antimicrobial resistance genes (AMR) through the use of sewage sludge. It was therefore considered, in light of recent concerns expressed by the general public into odour, bioaerosols, diffuse pollution etc. that this topic should be re-visited.

Since the publication of SNIFFER 2008, several studies have been undertaken looking at potential health impacts associated with the use of sewage sludge. Arguably, of most relevance to this project has been work undertaken in the Unites States by the EPA (2014) “Bioscience Resource Project: Sewage Sludge (Biosolids) — land application, health risks, and regulatory failure”. There have also been a large number of academic studies since 2008 (the search terms “sewage sludge OR biosolids AND human health” return almost 19,000 studies published since 2009 on Google Scholar). While the number of studies relevant to this project is far less (and many studies that seem relevant will not be of appropriate design), this does provide an indication of the level of new knowledge.

It is therefore now imperative that the evidence base provided in SNIFFER 2008 is not only updated, but also extended. In particular it needs to include common causes of complaint and emotive issues such as odour control, as well as ‘emerging’ contaminants including pharmaceuticals, personal care products, and AMR.

1.1. Objectives

The project aims to undertake the following objectives with oversight from a Steering Group consisting of Scottish Government, SEPA and Health Protection Scotland:

- Update the SNIFFER 2008 report with a focus on re-assessing and quantifying the human health implications and risks from sewage sludge storage, transportation and spreading on land (agricultural and non-agricultural land).

- Develop robust and effective evidence for potential impacts associated with spreading of sewage sludge on land, with emphasis on the potential effects strong odour can have on human health and well-being.

2. General Methodology

This section provides an overview of the methodology used within this generalised Quantitative Risk Assessment (QRA). Specific details and assumptions for individual potentially hazardous agents are described further in Section 3 (Results).

The aim of this work was to undertake a quantitative risk assessment that establishes the potential for harm to human health or well-being, resulting from the use of sewage sludge products, including the manner in which they were processed and used as shown in Figure 2-1.

Table 2-1 Summary of sewage sludge products and uses covered by this report

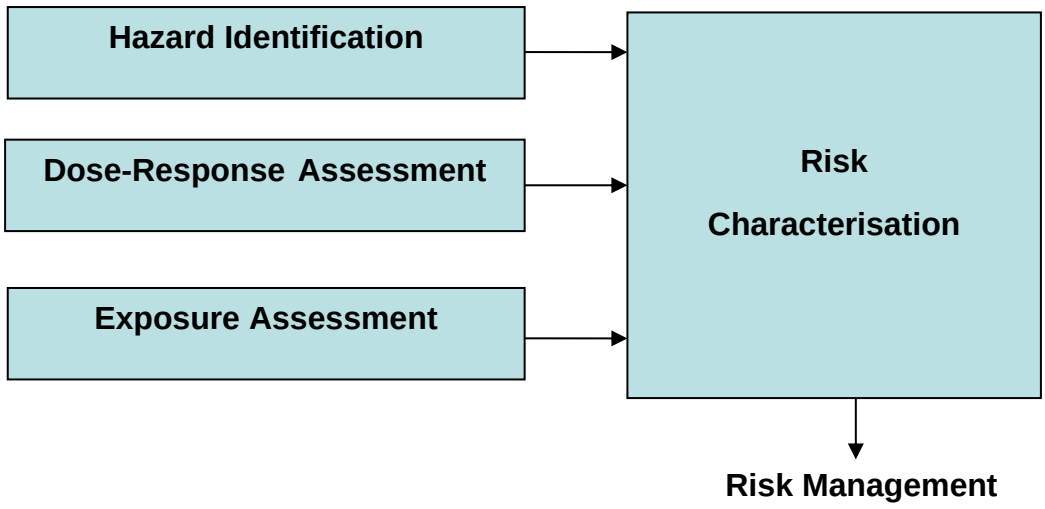
Input Materials	Treatment Method	End Use Parameters
• Thickened sludge from primary and secondary water treatment	• Conventionally (anaerobically) digested & dewatered cake • Dewatered raw cake with limed pasteurisation (lime caked) • Thermal hydrolysis pasteurisation (THP) digested and dewatered cake	• Agriculture (grazing land) • Agriculture (land used to grow grain crops for animal consumption) • Agriculture (land used to grow root crops for animal consumption) • Agriculture (land used to grow leaf crops for animal consumption)

It must be emphasised that this assessment only considered sewage sludge that has been produced under (regulatory) control. Activities outside of this specification, including unauthorized contamination of feedstocks and illegal use of sewage sludges have not been considered. This assessment examines potential risks associated with a specific product, and therefore does not make predictions about system failure, bypass of processing systems, or illegal activities.

The work was undertaken using the ‘classical model’ for QRA. This approach has been adopted by several agencies including Department of the Environment, Food & Rural Affairs (DEFRA) and the Institute of Environment and Health (Defra 2002).

The standard QRA model involves four key stages, namely hazard identification, dose-response assessment, exposure assessment, and risk characterisation (Figure 2-1). Briefly, the hazard identification comprises a literature-based review to identify which hazards, if any, are of most concern/most likely to be a risk; the dose-response assessment to characterise the magnitude of effect caused by specific doses of specific hazards, the exposure assessment to determine to what extent receptors are exposed to the hazards of concern, and finally, the risk characterisation to quantify the level of risk, i.e. the probability that a specific hazard will result in a specific adverse outcome. The risk characterisation may then be used to inform ‘risk management’, i.e. management of risk factors in order to reduce impacts of causative agents.

Figure 2-1 The four stages of the ‘Classical Model of Risk Assessment’



For this project, six categories of potentially hazardous agents listed in were considered (Table 2-2). The risks posed by each of these categories under different treatment methods, and end uses (listed in Table 2-1) were investigated.

Table 2-2 Categories of potentially hazardous agents that might be associated with sewage sludge

Categories of Potentially Hazardous Agents included in this assessment
Odour associated with spreading/applying sewage sludge products to agricultural land
Heavy metals and Inorganics including metals and metalloids
Organic contaminants including Polyaromatic Hydrocarbons (PAHs), Polychlorinated Biphenyls (PCBs), Dioxins and Furans, Flame retardants, Plasticisers, Synthetic phenolic compounds, Siloxanes, Benzothiazoles
Pharmaceutical and Personal Care Products (PPCPs) including anti-inflammatories, anti-epileptics, anti-histamines, selective serotonin reuptake inhibitors (SSRIs), antacids, antibiotics
Microplastics and Fibres defined as synthetic polymers measuring less than 5 mm in diameter (i.e. largest dimension)
Human/animal pathogens including bacteria, antibiotic resistant bacteria (ARB), viruses, protozoa, prions

2.1. Hazard identification and screening

The approach adopted for this stage of the QRA was adapted from Pollard et al. (2008). It was considered important by the Steering Committee (SC) that the assessment should demonstrate that all potentially hazardous agents had been considered where practicable. While the focus was on hazards not included in the original SNIFFER report (SNIFFER, 2008); it was considered neither feasible nor necessary to carry out a full QRA on each potentially hazardous agent identified. Instead, a series of filters was applied to the long list of hazards in order to produce a short list for further quantification. This filtering process has been used effectively in previous projects including those assessing risks associated with soil amendments (WRAP 2016a, b & c; Hough et al., 2012).

Initially, for each of the categories listed in Table 2-2, a comprehensive set of potentially hazardous agents were identified. As stipulated by the Steering Committee, information derived from peer-reviewed literature was used as primary source material. However, it was necessary to use some grey literature where relevant information was limited and the applicability, relevance and quality of this was judged by the project team before use. Potentially hazardous agents were included in the list if:

- They were not covered by the SNIFFER (2008) report, or significant new information published since 2008 was apparent,
- They had been identified or measured in sewage sludge, or
- Evidence was available that specific agents could enter the waste water treatment process assuming 'typical practice' was adhered to.

As peer-reviewed data from Scottish Water produced sewage sludge are limited, the identification of potentially hazardous agents included information from wider UK, EU, and North American sludge. Therefore, it should be emphasised that not all of the data reviewed was derived from sludge which had been produced to Scottish specification. Where the use of data from non-Scottish sewage sludge may have significant bearings on the findings of the QRA, this has been highlighted.

The agents to be considered were organized into the major groupings outlined in Table 2-2. A series of successive, defined, filters were then applied to each grouping of agents to identify those considered most likely to present a risk to humans (Figure 2-2). These filters are discussed in more detail in the following sub-sections.

2.1.1. Filter 1

Filter 1 asks whether the agent under consideration has a potentially serious effect on human health. This filter does not consider whether exposure is likely to occur, or if exposure would occur at a dose of concern, these factors are considered in the subsequent filters. For the majority of (chemical) hazards, a potentially serious effect was defined according to the definition used by the European Commission Enterprise and Industry Directorate (European Commission 2005):

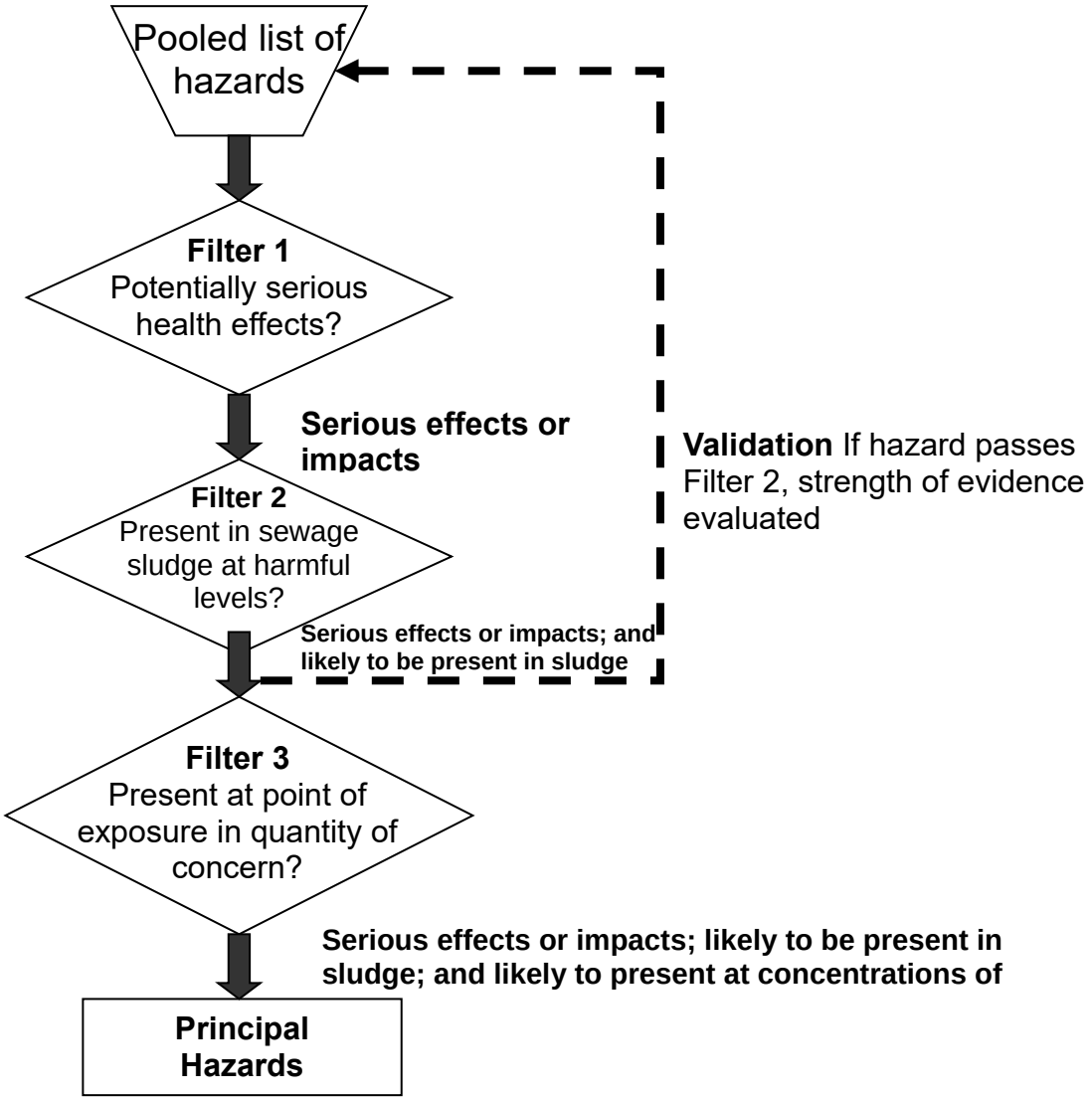
"'Serious' means a hazard that could result in death, could be life-threatening, could result in significant disability or incapacity, could be a congenital anomaly/birth defect, or which could result in hospitalisation or permanent or prolonged signs in exposed humans or animals, or which could realistically cause these effects where the product enters the environment."

This definition was adopted for most hazards potentially associated with 'traditional' health outcomes (i.e. cancer, cardiovascular disease, neurological conditions, etc.) as it has been successfully applied in previous projects that have focussed soil amendments (WRAP 2016a, b & c; Hough et al., 2012). However, this definition was widened to incorporate health outcomes associated with mental health and wellbeing, with this wider definition being applied specifically to outcomes associated with malodour. Therefore, when considering malodour and similar outcomes, we also adopted the following definition of health and wellbeing (World Health Organisation 1948 in Grad 2002):

"Health and well-being is a state of complete physical, mental and social wellbeing and not merely the absence of disease or infirmity"

All other effects were defined as being either ‘mild’ (i.e. readily reversible causing little/no short-term deleterious effects) or ‘moderate’ (i.e. reversible, but likely to cause some minor short-term deleterious effects). Where agents under consideration were associated with little or no effect, or where knowledge was insufficient, this was noted. No attempt was made to examine positive or protective effects of agents under consideration as this was considered outside the scope of this study. Only those agents considered to have a potentially serious effect were passed through Filter 1.

Figure 2-2 Flow chart for identifying principal public health hazards from the application of treated sewage sludge to agricultural land



2.1.2. Filter 2

Filter 2 considers if each agent is likely to be present in sewage sludge produced by a licensed operator at a level or concentration likely to cause harm to humans. This

filter is important when considering the water and sludge treatment processes and storage/stockpiling of sludge products. For example, a compound found to be present at a quantity of concern in sewage sludge, does not necessarily pose a risk to public health until the sludge has been spread. Further, people are not likely to be heavily exposed to the spread sewage sludge, when sludge has been spread according to current agricultural practice and other operational constraints. (The potential for the compound to pose a risk once the sewage sludge has been spread is considered in Filter 3). This filter does however highlight agents that could become an issue if good agricultural practice is not adhered to.

For some agents, numerous estimates of harmful levels are available. Where this was the case, the level of each agent considered to cause harm was determined using the concept of 'Principle Source Documents' adopted by the Environment Agency (Defra, 2002). These are set out below in descending order of priority:

1 Authoritative bodies in the UK (DEFRA), Scottish Government, Scottish Environment Protection Agency (SEPA), Environment Agency (EA))

2 European Commission Committees

3 Other national organisations (e.g. United States Environment Protection Agency (USEPA))

4 Reports produced by authoritative organisations, but for different purposes

Measured concentrations in sewage sludge were then compared to the 'harmful level' sourced using the 'Principle Source Documents' concept. Where measured concentrations exceeded the harmful level, these agents passed on to Filter 3 after validation (discussed below). Where a measured concentration in sewage sludge was not available, it was sometimes possible to find measured values for untreated sludge. In some cases, it was necessary to extrapolate from measured concentrations in waste water influent combined with knowledge of the chemical behaviour of the compound of interest, in order to estimate concentrations in sludge. In situations where little data were available describing degradation during the sludge treatment process a 'worst case' scenario of no degradation was assumed.

Any agent that reached Filter 2 and is considered to be present in quantities of concern by virtue of documentary evidence, or potentially present in quantities of concern (where documentary evidence is lacking), was then validated. As stated previously, not all of the literature was related to sewage sludge that had been produced to Scottish\UK\EU specification. Consequently, the validation process involved further examination of the reliability and appropriateness of the source of information. This included comparability with Scottish water and sludge treatment,

experimental design, and analytical procedures (including provision for Quality Assurance/ Quality Control). Where information was considered unreliable or inappropriate, these concerns were presented to the SC and wider stakeholders to reach a consensus whether it would be appropriate to consider this particular agent further.

2.1.3. Filter 3

Filter 3 assesses only those agents that have remained after the first two filters have been applied. This filter is concerned with exposure once the product has been spread in accordance with current agricultural practice. This process is further described in the following sections.

2.2. Exposure assessment

One aim of the exposure assessment was to quantify as much as possible potential exposure of individuals to the various hazardous agents. The level of quantification achieved by the exposure assessment was driven by data availability and accuracy and was different for the different potentially hazardous agents. The exposure modelling was particularly challenging due to the focus of this study on 'emerging' contaminants that had not been previously included in the SNIFFER (2008) report. As a result, different exposure modelling techniques were adopted for different hazardous agents/types of exposure. Two main modelling techniques were implemented:

- Bayesian Belief Network (BBN) models were adopted for those exposures were evidence was particularly uncertain or where information was limited/missing. BBNs have been used for a number of land-based risk assessments where information has been too incomplete to undertake a fully quantitative assessment (Troldborg et al., 2013; Aalders et al., 2011; Hough et al., 2010a)
- Multi-media fugacity modelling was implemented for the majority of organic contaminants and PPCPs. Fugacity modelling relies on partition coefficients and these have usually been derived for most commercially available chemicals/compounds. Fugacity modelling has been used successfully in a number of studies looking at sewage sludge application to land, more recently with respect to exposure to Bisphenol A (Zhang et al., 2015)

These methodologies are described in detail in Section 6 - Appendix A.

2.3. Dose-Response assessment

Dose-response data describe the magnitude of an outcome (response) in relation to the magnitude of a dose (exposure) of a specific agent. Dose-response data in the literature are in several different formats and it may therefore be necessary to convert data to a standard form. Most data are derived from laboratory experiments where discrete groups of animals, e.g. mice, are exposed to a specific dose. A number of groups of animals are used so that several exposures of different magnitude can be administered. Some data are presented as percentage of animals showing a specific response, while other studies present continuous biochemical data.

The majority of toxicologic dose-response data relate to exposures far greater than environmental levels in order to get an observable response in a limited number of experimental animals. Hence care must be taken in extrapolating such data to environmentally relevant concentrations. There are many methodological approaches to carry out such extrapolations, including various mathematical curve-fitting models. Since 1995, many agencies have started to use the benchmark dose method to estimate the no observed adverse effect level (NOAEL) and/or the expected dose (ED) (Crump, 1984). The benchmark dose is based on the lower 95 % confidence interval of the fitted dose-response model, resulting in a response in 10 % of the study animals. The rationale being that a 10 % response is at or just below the limit of sensitivity in most animal studies. The use of the lower confidence interval, rather than the model fit itself, accounts for experimental uncertainty. Overall, the benchmark dose approach improves certainty in estimates of NOAEL. However, choice of linear or curve-linear (etc.) models to extrapolate from high to low dose is still, in many instances, reliant on expert judgement and is associated with significant uncertainty.

For non-cancer end points, it is standard practice to assume that a threshold of effect exists, while no threshold is assumed with carcinogenic endpoints. Although carcinogenic, a threshold effect was also assumed to exist for dioxins and dioxin-like PCBs. This is in line with the Committees on Toxicity, Mutagenicity and Carcinogenicity of Chemicals in Food, Consumer Products and the Environment (COT COM 2001). The COT COM (2001) agreed there was sufficient information to assume a threshold existed for the effects of dioxins and hence a tolerable daily intake could be established. There were two critical components to this decision:

- There is considerable evidence that dioxins do not directly damage the genetic material.
- There is considerable understanding of the biological reactions by which dioxins cause harmful effects, and evidence that these reactions will not occur at sufficiently low levels of exposure.

For the majority of organic pollutants and PPCPs, estimates of exposure (ADD, mg kg^{-1} d^{-1}) could be compared directly to ‘safe’ reference doses (RfD, mg kg^{-1} d^{-1})

published in the literature. However, for a number of agents, RfD values had to be estimated from reported NOAELs and other points of departure as there were no published RfDs. This was done following the method of the United States Environment Protection Agency (Equation 2.1; USEPA, 1996):

$$RfD = \frac{NOAEL_5}{UF_L UF_H} \qquad \text{(Equation 2-1)}$$

The RfD is considered to be a daily dose to which the receptor can be exposed without experiencing any deleterious effects. The RfD is determined by applying Uncertainty Factors (UF) to the NOAEL or other point of departure (Barnes & Dourson, 1988; Clegg et al., 1986). In this study, a maximum of two uncertainty factors were applied to the lower 95 % confidence interval of the NOAEL ($NOAEL_5$). The first UF (UF_L) was used to account for uncertainties associated with extrapolating from the experimental population to the population at risk. This UF was applied where species differences existed, e.g. extrapolating from an experimental rat population to a human population. The second factor (UF_H) was used to account for variability within receptor populations, e.g. differences in the amount of exposure medium consumed, differences in the inherent susceptibility of different members of the population (Barnes and Dourson, 1988). Following standard procedures, each UF is usually assigned a value of 10 but can be much greater depending on the uncertainties inherent in the toxicological data or the suitability of the toxicological data for extrapolation to human receptors (Barnes and Dourson, 1988). The reference doses for the different potentially hazardous agents are given in Table 3-5 & 3-9 alongside their associated uncertainty factors.

It should be noted that within this study, dioxins and dioxin-like PCBs were assessed both on an individual basis, and collectively using Toxic Equivalency Factors (TEFs) and Toxic Equivalents (TEQs). Toxic Equivalency Factors (TEFs) are toxicity potency factors that are used by the World Health Organization (WHO) and regulators as a consistent method to evaluate the toxicities of highly variable mixtures of dioxin compounds. In previous risk assessments of agricultural soil amendments, this approach has been favoured by some members of the Steering Committees, including the Food Standards Agency (WRAP 2016a, b & c; Hough et al., 2012). While TEQs are the standard approach, it was considered appropriate for this study to also assess each congener separately because: (i) published data on the levels of all congeners in sewage sludge were not available; (ii) there are differences in the extent to which different congeners move through the environment.

The issue of mixtures and their actions has been studied using laboratory rodents. Some data suggest that even when each component of a cocktail is present at concentrations that, individually, result in no observable biological effect, the mixture can exert significant biological effects (Payne et al., 2001; Rajapakse et al., 2002; Tindall and Ashby, 2004). Despite advances, this evidence is in its infancy, and comprehensive data are not available for all of the combinations of chemicals

possibly present in all agricultural amendments, including sewage sludge. This is especially the case with respect to the 'emerging' contaminants that are the focus of this study. However, some work involving sheep exposed to mixtures of pollutants, through grazing pastures fertilised with sewage sludge, has shown that exposure to low concentrations of multiple pollutants can disrupt foetal ovarian and testis development (Paul et al, 2005; Fowler et al., 2008), offspring behaviour (Erhard and Rhind, 2004) and adult bone structure (Lind et al., 2009). It would be problematic to incorporate the issue of mixtures into the risk assessment approach used here. With thousands of pollutants known to have the potential to disrupt biological systems, understanding of mixture effects and meaningful assessment of risk will require the integration of observations from a wide range of empirical approaches together with the use of powerful, predictive computer models (Suk et al., 2002).

2.4. Risk characterisation

For the majority of agents, 'risk' was defined as the modelled probability that after spreading sewage sludge on agricultural land, an individual human receptor would experience deleterious health effects from either direct ingestion/inhalation or ingestion of food products produced on that land. This approach of calculating risk on an individual basis is the most appropriate because associated legislation, e.g. food safety, is based on individual products, rather than on the market as a whole.

Risk was calculated as the ratio (or Hazard Quotient, HQ) of the exposure (Average Daily Dose, ADD, mg kg^{-1} d^{-1}) to the appropriate reference dose (RfD, mg kg^{-1} d^{-1}) derived in Section 2.3; Equation 2-2. If the ADD exceeds the RfD, we might expect to see deleterious effects to occur during the lifetime of the receptor.

$$HQ = \frac{ADD}{RfD} \qquad \text{(Equation 2-2)}$$

Due to the significant uncertainties associated with estimating risks, an HQ greater than 1.0 indicates an issue that may require further investigation – but does not automatically imply a 'real' risk. An HQ less than or equal to 1.0 may be regarded as 'safe' (or negligible risk). For ease of interpretation, risk in this study was expressed either as 'negligible' (HQ $\leq$ 1.0) or potentially requiring further investigation (HQ > 1.0).

For agents modelled using the BBN approach, once the network structure has been optimised, health risk may be considered as an expectation value measuring the probability of the extent to which a specific (health) outcome is likely (Hough et al., 2010a):

$$Risk = \int p(x)V(x)vx \qquad \text{(Equation 2-3)}$$

Where V(x) is a numerical expression of the specific (health) outcome, p(x) is the probability of the (health) outcome arising and the integral is performed over all possible realizations (denoted by a variable x).

2.5. Sensitivity analysis

A simple point sensitivity analysis was conducted to identify which input parameters the risk assessment is most sensitive to and therefore are most important to characterise accurately in order to reduce the output uncertainty. A point sensitivity analysis investigates how the model output changes relative to the change in each input parameter while keeping all the other inputs at a fixed level. The sensitivity can be expressed in different ways. Here, the sensitivity of the model output, *O*, to a parameter *i* taking the value x_i is expressed through a normalised sensitivity index, SI, calculated as (Spitz and Moreno, 1996):

$$SI_i = \frac{|dO|}{\left(\frac{|dx_i|}{x_i}\right)}$$

(Equation 2-4)

where |*dO*|is the absolute change in the model output following a change in the input parameter value dx_i , and x_i is the initial parameter value (i.e. in the base case).

3. Findings

3.1. Malodour

3.1.1. Background

Odours remain the biggest cause of complaints in regard to environmental issues for a wide variety of industries, including wastewater and waste management, and sewage sludge application to land, and continues to grow in both number and severity of complaints (Intarakosit, 2010; Adams et al., 2003; Harrison & Oakes, 2002; Gostelow et al., 2001; van Harreveld, 2001; Shusterman, 1999). These concerns seem to be multi-dimensional, in that it is not only the detected odour that determines the impact of a malodourous exposure on a community, but cognitive appraisal, community interests, as well as several other factors play a role in shaping the effects of arduous odour. These factors may elucidate why intolerance for malodour seems to be increasing (Sucker et al., 2008a & b). In addition, the understanding of these factors may also explain why, despite significant investment in odour reduction and abatement, governing bodies have had difficulty in establishing fair and effective regulations that address community needs (Nicell, 2009; Rappert & Muller, 2005). Cervinka & Neudorfer (2004) found that canal air harbouring sewage odours caused a high degree of complaints even when those odours were drastically reduced in intensity. One of the many cited explanations for this increased complaint factor was the increased sensitivity to environmental stressors experienced by the community in the region; clearly, meeting community expectations requires a dynamic and multi-faceted understanding beyond that of an odour concentration-response paradigm (Hayes et al., 2014). This is at odds with traditional risk assessment approaches (as described in Section 2) used in this study and widely to address environmental issues.

3.1.2. Health effects

One major property of the sense of smell is to warn an individual about potential health hazards; to that end, odour often implies danger; this in turn usually leads to more health complaints from those who perceive the odour (Moffatt & Pless-Mulloli, 2003; Köster, 2002; Luginaah et al., 2002; Elliott et al. 1999). Several investigations have found that, if present, odour elicits the largest number of complaints from a community who perceive a location or activity to be “dangerous” far more than any other description (Jenkins et al., 2007; Adams et al., 2003; Harrison & Oaks, 2002; Luginaah et al., 2002). Perception of odour was found to be the most significant risk factor for multiple health effects in people residing close to hazardous waste sites in California (Neutra et al., 1991). Interestingly, even a “dummy” question relating to toothache (for which odour exposure should have no influence on even as a

stressor) had higher incidence rates for people who detected odours. This was suggested to indicate an odour-worry paradigm that might explain the association between odour and health (Neutra et al., 1991).

The health effects of odours themselves may be related to their cognitive appraisal, but assessing risk is still mired in difficulties. To begin with, there are multiple competing hypotheses as to the pathophysiological reasons behind odours causing health effects, ranging from innate odour preferences, to stress-induced illness, to mass psychological hysteria (Shusterman, 2001; 1999; Schiffman et al., 2000). It has also been suggested that perceived health effects are among the most important factors when individuals consider registering a complaint (Kolarova, 1999). An additional and difficult delineation is between odour and olfactory irritation, a separate factor that affects an individual's trigeminal nerve (Schiffman et al., 2000). Regardless, odour exposure causes increased rates of reported health effects, even when the odour contains no agents at or above toxic levels (Schiffman & Williams, 2005; Winneke, 2004a & b; Shustermann, 1999). To this end, governments have responded by setting limits of odour production from various sources and activities stating that concentrations far below toxic levels must not be breached (Hayes et al., 2014).

Despite these challenges with establishing causality, a few dose-response relationships have been published that try to relate measured or modelled concentrations of odorants with (primarily) self-reported health effects (e.g. Blanes-Vidal et al., 2014; Aatamila et al., 2011, 2010). Of note is the study of Blanes-Vidal et al. (2014) as being one of the few, possibly the only, studies that provides a quantitative estimation of the dose-response relationships between exposure to odorants and psychosocial effects caused by odour in a non-urban setting; hence analogous to sewage sludge spreading. The study also shows that the health effects are not only direct but are also mediated by other psychosocial responses. After controlling for person-specific covariates, about 45% of participants were annoyed by the odour pollution. Exposures were associated with annoyance (adjusted odds ratio [OR_{adj}] = 3.54, 95% confidence interval [CI] = 2.33 – 5.39), health risk perception (OR_{adj} = 4.94; 95% CI = 1.95 – 12.5) and behavioural interference (OR_{adj} = 3.28; 95% CI = 1.77 – 6.11), for each unit increase in $\log_e(NH_3$ exposure). Annoyance was a strong mediator in exposure-behaviour interference and exposure-health risk perception relationships (81% and 44% mediation, respectively). Health risk perception did not play a mediating role in exposure-annoyance or exposure-behavioural interference relationships. The dose-response relationships between NH_3 exposure and annoyance presented by Blanes-Vidal et al. (2014) have been used in the risk modelling undertaken in this study (Section 3.1.4), with 'annoyance' considered the health outcome of interest.

3.1.3. Sources in sewage sludge

Many compounds have been identified in odours arising from sewage sludge treatment (Gostelow et al., 2001; Table 3-1). Generally speaking, these compounds are reduced sulphur or nitrogen compounds, organic acids, aldehydes or ketones. More recently, Kotowska et al. (2012) detected over 170 volatile and potentially volatile compounds in municipal sewage sludge including a number considered detremental to human health. These included aliphatic and aromatic hydrocarbons, alcohols, esters, carbonyls, as well as sulphur, nitrogen, and chlorine containing compounds. The prevailing substances included: ethyl ether, *n*-hexane, *p*-xylene, *o*-xylene, mesitylene, *m*-ethylbenzene, limonene, *n*-decane, *n*-undecane, and *n*-dodecane. A few compounds such as methanetiol, dimethyl polisulfide, octaatomic sulphur, phthalic anhydride, and indoles were idenfied in sewage sludge for the very first time.

Sulphur compounds form the majority of odorants associated with sewage sludge and its treatment (Abbott, 1993; Bonin et al., 1990). Domestic sewage typically contains 3 – 6 mg L^{-1} organic sulphur, derived mainly from proteinaceous material, as well as sulphonates from household detergents (Boon, 1995). Inorganic sulphur, in the form of sulphate, is present in concentrations from 30 – 50 mg L^{-1} or considerably higher where industrial sources of waste water are treated (Boon, 1995; Cheremisinoff, 1988).

Hydrogen sulphide (H_2S) is the predominant odorant associated with sewage and sewage sludge and the formation of H_2S in sewers has been extensively studied, primarily because of its corrosive powers (e.g. Matos & Sousa, 1992; Halkjaer-Nielsen et al., 1998; Pomeroy & Parkhurst, 1977; Pomeroy, 1959). The formation of H_2S arises from two sources: the reduction of sulphate and the desulphurisation of organic compounds containing sulphur in a reduced state (Harkness, 1980).

Nitrogen-containing odorants (primarily ammonia, amines, indole and scatole) can also be significant sources of odour. Nitrogen sources in sewage are urine, proteins, and amino acids. Amines in particular are produced from amino acids by the removal for the carboxyl (COOH) group (Harkness, 1980). Volatile fatty acids, aldehydes, alcohols and ketones are the by-products of carbohydrate fermentation and are generally associated with anaerobic treatment, and in particular with the treatment of sewage sludge (Bonnin et al., 1990).

Table 3-1 Odorants associated with sewage sludge and its treatment

Adapted from Gostelow et al., 2001 (with reference to: Abbott, 1993; Bonnin et al., 1990; Brennan, 1993; Cheremisinoff, 1988; Koe, 1989; Vincent & Hobson, 1998; Young, 1984)

Class	Compound	Formula	Character
Sulphurous	Hydrogen sulphide	H_2S	Rotten eggs
	Dimethyl sulphide	$(CH_3)_2S$	Decayed vegetables
	Diethyl sulphide	$(C_2H_5)_2S$	Nauseating, ether
	Diphenyl sulphide	$(C_6H_5)_2S$	Burnt rubber
	Diallyl sulphide	$(CH_2CHCH_2)_2S$	Garlic
	Carbon disulphide	CS_2	Decayed vegetables
	Dimethyl disulphide	$(CH_3)_2S_2$	Putrification
	Methyl mercaptan	CH_3SH	Decayed cabbage
	Ethyl mercaptan	C_2H_5SH	Decayed cabbage
	Propyl mercaptan	C_3H_7SH	Unpleasant
	Butyl mercaptan	C_4H_9SH	Unpleasant
	tButyl mercaptan	$(CH_3)_3CSH$	Unpleasant
	Allyl mercaptan	CH_2CHCH_2SH	Garlic
	Crotyl mercaptan	$CH_3CHCHCH_2SH$	Skunk, rancid
	Benzyl mercaptan	$C_6H_5CH_2SH$	Unpleasant
	Thiocresol	$CH_3C_6H_4SH$	Skunk, rancid
	Thiophenol	C_6H_5SH	Putrid, nauseating
	Sulphur dioxide	SO_2	Sharp, pungent, irritating
Nitrogenous	Ammonia	NH_3	Sharp, pungent
	Methylamine	CH_3NH_2	Fishy
	Dimethylamine	$(CH_3)_2NH$	Fishy
	Trimethylamine	$(CH_3)_3N$	Fishy, ammoniacal
	Ethylamine	$C_2H_5NH_2$	Ammoniacal
	Diethylamine	$(C_2H_5)_2NH_2$	
	Triethylamine	$(C_2H_5)_3N$	
	Diamines, e.g.	$NH_2(CH_2)_5NH_2$	Decomposing meat
	Cadaverine	C_6H_5N	Disagreeable,
	Pyridine	C_8H_6NH	irritating
	Indole	C_9H_8NH	Faecal, nauseating
	Scatole or Skatole		Faecal, nauseating
Acids	Acetic (ethanoic)	CH_3COOH	Vinegar
	Butyric (butanoic)	C_3H_7COOH	Rancid, sweaty
	Valeric (pentanoic)	C_4H_9COOH	Sweaty

Aldehydes & Ketones	Formaldehyde Acetaldehyde Butyraldehyde Isobutyraldehyde Isovaleraldehyde Acetone Butanone	HCHO CH_3CHO C_3H_7CHO $(CH_3)_2CHCHO$ $(CH_3)_2CHCH_2CHO$ CH_3COCH_3 $C_2H_5COCH_3$	Acrid, suffocating Fruit, apple Rancid, sweaty Fruit Fruit, apple Fruit, sweet Green apple

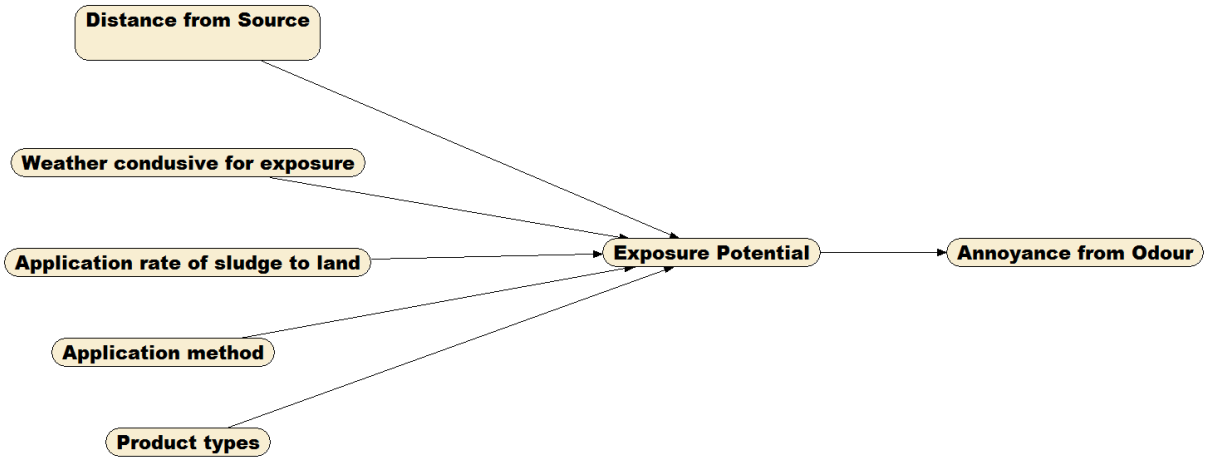
3.1.4. Risk assessment

Due to the limitations and uncertainties in the data linking odour exposure to health outcomes, as well as the nature of the health outcome data (self-reported) and the psychosocial aspects of the exposure-response relationship (not least the odour-worry paradigm, Neutra et al., 1991), it was considered that risks to humans from odours originating from sewage sludge could only be assessed using a Bayesian Network approach (Section 2.2.1).

The BBN model described here was developed using Netica™ version 3.25 by Norsys Systems Corp. In line with the guidance from Marcot et al (2006), the development of a BBN for this paper started with the creation of a conceptual model of the relationships (influence) diagram using data on sewage sludge production and application, data from studies linking odour exposure to health outcomes, as well as expert knowledge and experience of sludge handling and odour modelling (Figure 3-1). The final outcome node is *Annoyance from odour* with 'annoyance' defined according to Blanes-Vidal et al. (2012).

The diagram represents key drivers (in nodes) of annoyance from odour due to sewage sludge application in Scotland. In the next phase each factor (node) was allocated values. In accordance with the considerable uncertainty in the data, all values were discretised into a number of categories or states. The number of states per node was kept to a minimum, and where possible Boolean values were adopted.

Figure 3-1 Conceptual diagram (used as the basis for the final BBN model) describing the main factors that drive level of annoyance (defined according to Blanes-Vidal et al. 2012) from odour originating from sewage sludge application.



Model structure

The model structure (Figure 3-1) was broadly based around three main drivers of odour exposure; namely sludge handling (e.g. product type, application method, application rate), climate and weather (i.e. are weather conditions likely to promote relatively localised concentration of odour), and location of receptors (i.e. the distance from source to receptor). These groupings are based on a synthesis of the available literature and there is either implicit or explicit evidence that they contribute to exposure to odour either in isolation or more often in combination. The relationships are often complex; for example, it is often the combination of temperature, rainfall and wind in a specific sequence that can trigger an exposure event, regardless of sludge handling characteristics.

Parameter learning

The current model (Figure 3-1) consists of 7 nodes and 6 links. The outcome node, *Annoyance from Odour*, considers the process by which a particular receptor, given various sludge handing, distance from the source of odour, and weather conditions during the exposure event, may become annoyed due to perception of odour. The relationships between the different nodes are represented in conditional probability tables (CPTs). Parameter learning determines the prior CPT of each node of the network, given the link structures and available data. A CPT $P(A|B_1 \ldots B_n)$ was attached to each variable A with parents $B_1 \ldots B_n$. If A had no parents (e.g. *Product types* in Figure 3-1), the table was reduced to unconditional probabilities i.e. P(A). To illustrate, the prior unconditional probabilities for *Product types* was P(*Product types*), while the prior conditional probabilities for *Exposure potential* was P(*Exposure potential* | *Distance from source*, *Weather*, *Application rate*, *Application method*, *Product types*). Prior conditional probabilities were calculated by applying Bayes rule:

P(Exposure potential, Distance from source, Weather, Application rate, Application method, Product types)

= P(Exposure potential | Distance from source, Weather, Application rate, Application method, Product types)

* P(Distance from source, Weather, Application rate, Application method, Product types)

(Equation 3-1)

Prior unconditional probabilities were built using both ‘expert’ knowledge and actual observations. The ultimate objective is that all nodes are populated with empirical evidence. However, given the limitations of the information available, in the early stages of development the BBN was populated using a combination of expert knowledge and empirical evidence. With BBN’s ability to learn from new data, the model developed through an iterative process which improved our understanding and knowledge. Table 3-2 provides a list of all nodes and the information sources used to populate the prior CPTs. The aim was to develop prior probabilities that reflected the national (Scotland) situation; for example, looking at the *Product type* node, the prior distribution reflects the probability that any sewage sludge product available in Scotland is e.g. lime treated. The finalised BBN, complete with prior distributions is depicted in Figure 3-2

Figure 3-2 Bayesian belief network (BBN) model of risk of Annoyance from odour from sewage sludge, with prior probability distributions representative of Scotland

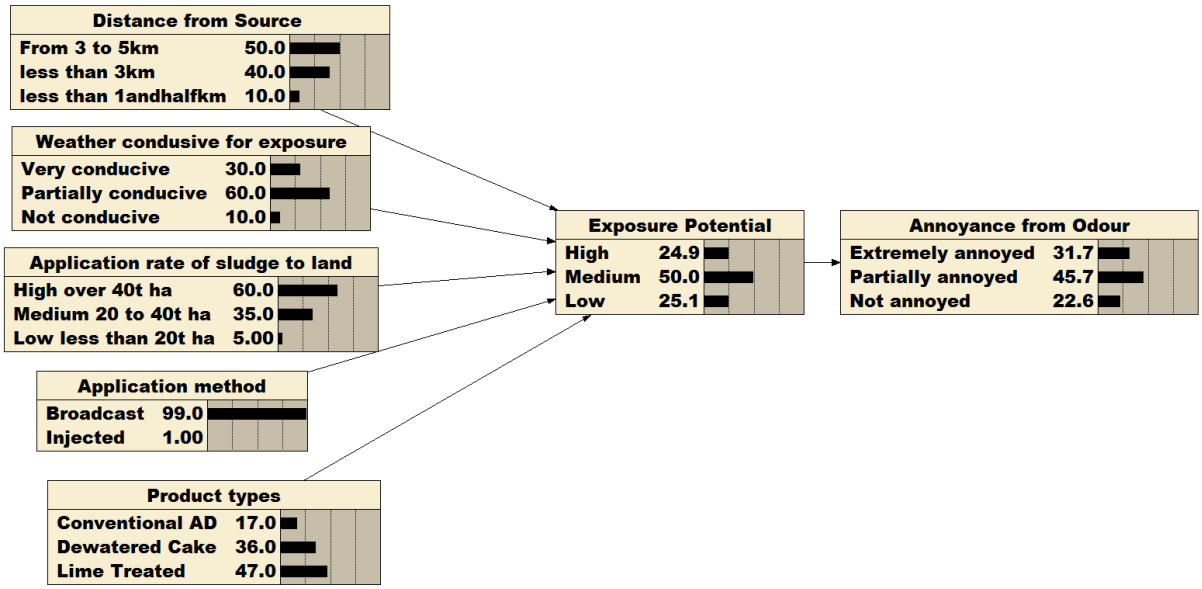


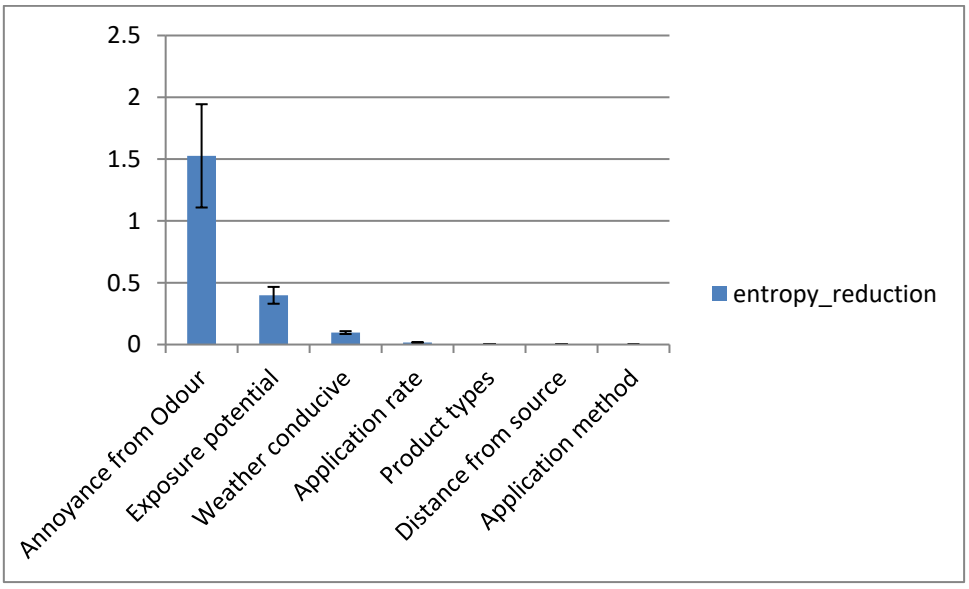
Table 3-2 List of nodes included in the odour annoyance BBN, and the information used to populate prior conditional probability tables

Node	Description	Information type/source
Distance from source	Distance of receptors from nearest edge of source of odour (<1.5 km: >1.5<3 km:>3<5 km)	Relationship between distances and exposure levels derived from Aatamila et al. 2011, 2010
Weather conducive for exposure	Proportion of the time that weather conditions are considered to promote exposure to odour (very conducive:partially conducive:not conducive)	Probability of atmospheric stability class being conducive for odour perception. Based on the relationship between the Pasquill atmospheric stability classes (Pasquill 1961) and odour perception of point sources (pig farms) as reported by Guo et al. (2006)
Application rate of sludge to land	Application rate in t fw ha^{-1} (>40 t ha^{-1}: >20<40 t ha^{-1} : <20 t ha^{-1})	Based on application rates used in WRAP (2016c) and Hough et al. (2012) which were themselves based on expert judgement
Application method	Method by which sewage sludge is applied to agricultural land (Broadcast: Injected)	Based on personal communication
Product Types	Probability of a sewage sludge product used in Scotland to be of a specific type (Conventional AD: Dewatered cake: Lime Treated)	Based on sludge production figures obtained from Scottish Water
Exposure potential	Probability that receptors will experience odour perception (High: Medium: Low). Levels of exposure assumed to equate to approximately: High = 100 $\mu g\ m^{-3}\ NH_4$ Medium = 10 $\mu g\ m^{-3}\ NH_4$ Low exposure = 1 $\mu g\ m^{-3}\ NH_4$ (Blanes-Vidal et al. 2012)	Expert inference based on all combinations of states of its 5 parent nodes. The contribution of product type to exposure potential was derived from the ADAS Odour Report undertaken as part of this project.
Annoyance from Odour	Probability of perceiving the odour and becoming annoyed as the health outcome. Levels of annoyance (Extremely annoyed: Partially annoyed: Not annoyed) as defined by Blanes-Vidal et al. (2012).	Derived from dose-response relationships between exposure to NH_4 and level of annoyance (Blanes-Vidal et al., 2012).

Structural learning

Sensitivity to findings analysis was performed to determine which variables had most influence on the outcome node, *Annoyance from Odour*. This was determined by calculating an entropy reduction value (ERV). The higher the value of ERV, the stronger a node affects another. Estimates of ERV, and the associated variance of beliefs, were calculated for *Annoyance from Odour* and then for the influence of each node in turn on *Annoyance from Odour* (Figure 3-3). It is noteworthy that all of the variables where operators have significant control have minimal influence on *Annoyance from Odour*, suggesting that changing product type, application method or even increasing the distance between source and receptors (i.e. a 'buffer zone') will have little influence on how annoyed the local population are. Weather conditions play the most significant role in the outcome of *Annoyance from Odour* and suggest that timing of sewage sludge application to coincide with specific weather conditions might help reduce levels of annoyance in the community. This seems logical as the weather conditions play a key role in the source – pathway – exposure linkage. If the weather conditions are such that odour is not transported from the source to the receptor, then no exposure will occur. This does not however mean that no one will be annoyed. As described above, annoyance from odour has a psychosocial etiology, thus regardless of odour exposure a small minority of people will still report being 'extremely annoyed'; in the case of the presented model there remains a 10.7% probability of being extremely annoyed even in the absence of odour exposure (Figure 3-4). This reflects that fact that a proportion of individuals within communities living within 5 km of regular sewage sludge spreading activities develop odour-worry condition (Neutra et al., 1991).

Figure 3-3 Sensitivity of Annoyance from Odour due to a finding at another node. Bars represent entropy reduction value; error bars indicate the associated variance of beliefs.



Scenario testing – forward inference

The BBN model can be used to test a variety of scenarios by forward inference. Here each node is set to its highest or lowest state to understand the impact of each factor on the risk of individuals becoming extremely annoyed by odour exposure. Figure 3-4 shows, by way of illustration, a scenario where weather conditions are very conducive to exposure. Comparing the distribution of Annoyance from odour with Figure 3-2 you can see an 18.1% increase in the probability that individuals will become extremely annoyed. A number of different scenarios were explored in turn (Table 3-3).

Figure 3-4 Examples of forward inference scenario. Top: here Weather conducive for exposure is set to P(Very conducive) = 100% resulting in Annoyance from odour P(Extremely annoyed) = 49.8% (cf. 37% in Figure 3-2). Bottom: here Weather conducive for exposure is set to P(Not conducive) = 100% resulting in Annoyance from odour P(Extremely annoyed) = 10.7% (cf. 37% in Figure 3-2).

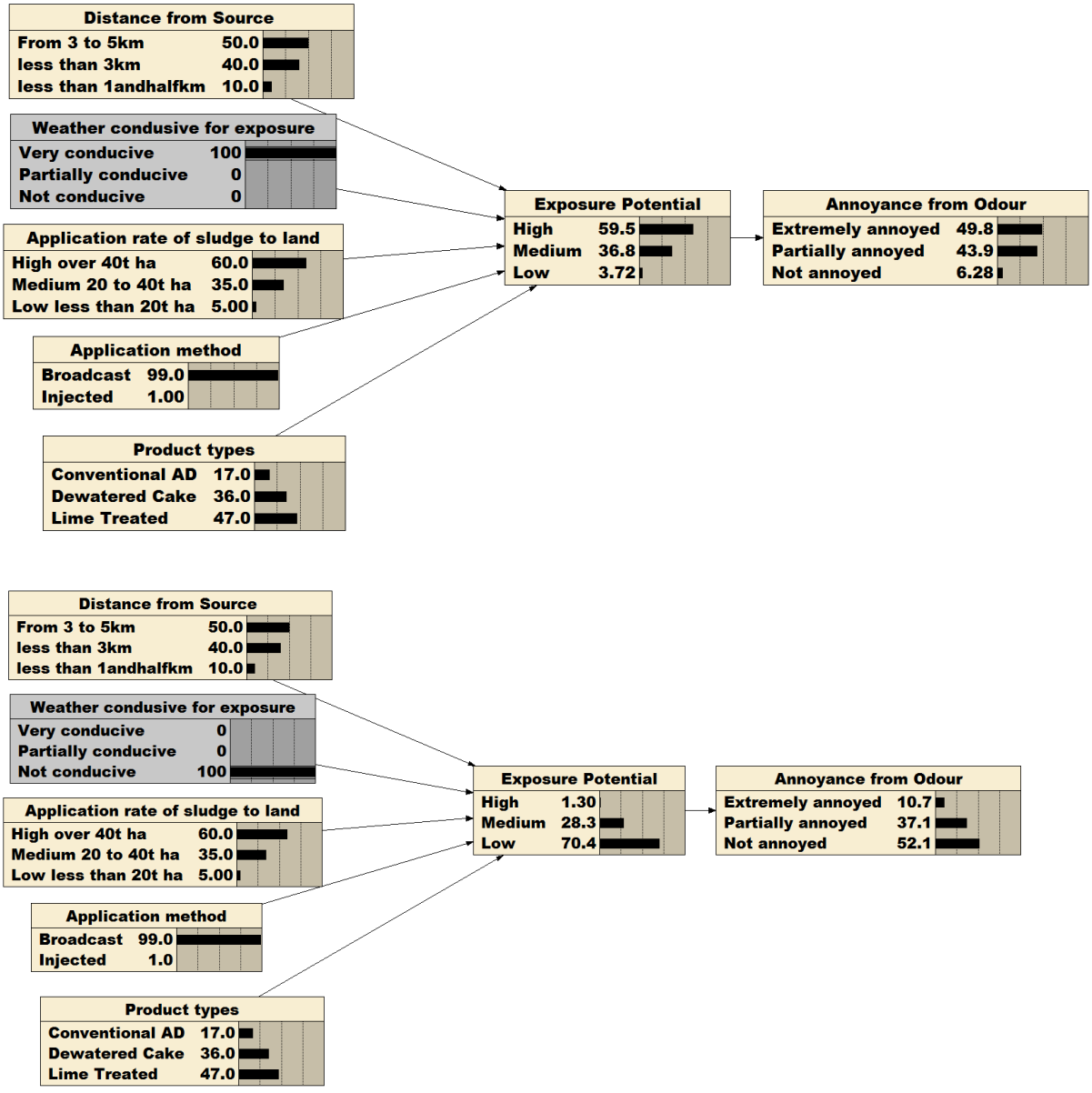


Table 3-3 Change in probability of being Extremely annoyed (ΔP(Extremely annoyed)) arising from different forward inference scenarios tested Weather conducive for exposure either set as its prior probability, or P(Very conducive) = 100%

Node	Scenario	ΔP(Extremely annoyed)
Weather conducive for exposure	P(Not conducive) = 100%	-21%
	P(Very conducive) = 100%	+18.1%
Product types	P(Conventional AD) = 100%	-2.2%
	P(Lime treated) = 100%	+2.4%
Product types; Weather conducive for exposure	P(Conventional AD) = 100%; P(Very conducive) = 100%	-3.9%
	P(Lime treated) = 100%; P(Very conducive) = 100%	+4.4%
Application method	P(Injected) = 100%	-6.7%
Application method; Weather conducive for exposure	P(Injected) = 100%; P(Very conducive) = 100%	-9.9%
Application rate	P(Low less than 20t ha) = 100%	-16.2%
	P(High over 40t ha) = 100%	+3.1%
Application rate; Weather conducive for exposure	P(Low less than 20t ha) = 100%; P(Very conducive) = 100%	-20.3%
	P(High over 40t ha) = 100%; P(Very conducive) = 100%	+3%
Distance from source	P(From 3 to 5km) = 100%	-2.6%
	P(Less than 1andhalfkm) = 100%	+6.1%
Distance from source; Weather conducive for exposure	P(From 3 to 5km) = 100%; P(Very conducive) = 100%	-2.3%
	P(Less than 1andhalfkm) = 100% P(Very conducive) = 100%	+5.3%

The forward inference scenario analysis concurs with the sensitivity to findings analysis, highlighting the pivotal role that weather conditions play in the source – pathway – exposure linkage. The CPT for Weather conducive to exposure is derived from the relationship between atmospheric stability classes (Pasquill 1961) and levels of odour perception from point sources (Guo et al., 2006). The 'Not conducive' state is the probability of Scottish weather being Pasquill stability class A characterised by wind speed <2 m s^{-1} and strong daytime solar radiation; the 'Very

conducive' state is the probability of Scottish weather being Pasquill stability class D characterised by wind speed >6 m s^{-1}, slight daytime solar radiation, and night time cloud >50% of the time. 'Partially conducive' is the probability of Scottish weather being Pasquill stability classes B, C, E, F or G. Guo et al. (2006) found that out of 298 recorded odour events, none were reported when conditions were Pasquill A, but 184 (~62%) odour events (including 26 very high intensity events) were recorded under Pasquill D conditions. The remaining 114 odour events were split between the remaining Pasquill classes.

Application rate was also highlighted as being an important risk factor that essentially reduces the source term and hence intensity of the odour that is released from the spreading processes. Reducing application rate down to <20 t ha^{-1} reduces P(Extremely annoyed) by 16.2 %. In many circumstances, the Safe Sludge Matrix already requires that application rates are of a similar magnitude.

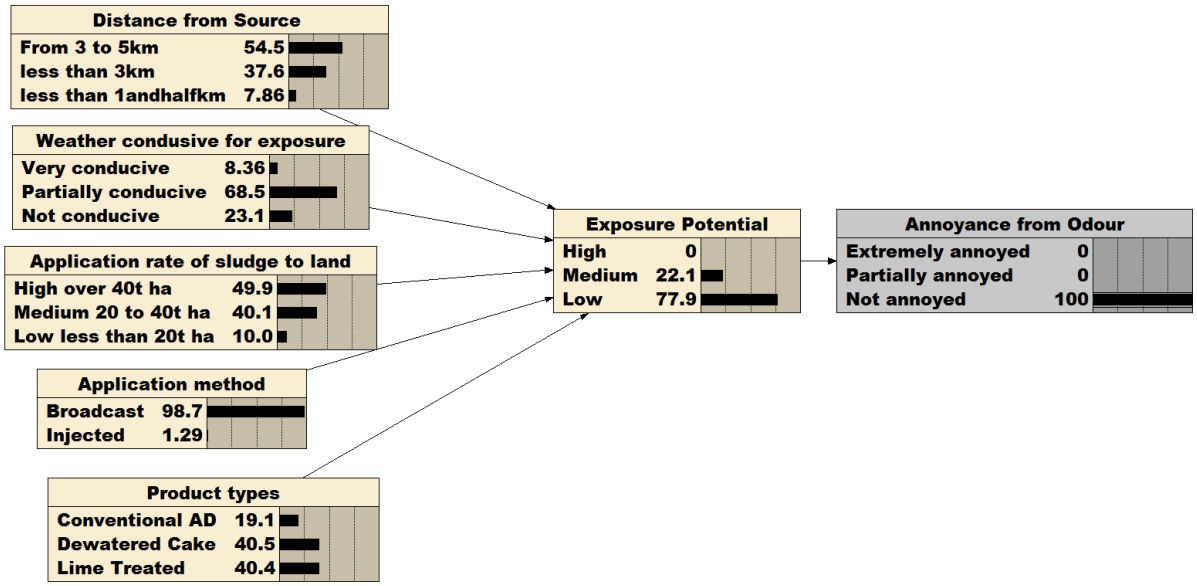
While Weather conducive for exposure is the most important risk factor for Annoyance from odour; it is interesting to explore which management options help mitigate odour most effectively. To do this, Weather conducive for exposure was set to P(Very conducive) = 100% and the management factors varied in turn (Table 3-3). The biggest reductions in P(Extremely annoyed) were again seen by reducing the application rate (reduction in P(Extremely annoyed) of 20.3%).

Scenario testing – backwards inference

To investigate the conditions required to achieve Annoyance from odour P(Not annoyed) = 100%, a backwards inference was performed (Figure 3-5). While (due to phenomena such as odour-worry condition) it would not be possible to achieve a complete absence of annoyance in the community, the backwards inference does inform the steps that could be taken to reduce probability of annoyance. In summary, these are:

- Ensure that the distance from the sewage spreading operations to the nearest residencies is at least 3 km
- Restrict spreading of sewage sludge to weather conditions that are less conducive for exposure, with partially conducive (i.e. Pasquill classes B, C, E, F & G) being protective enough if the other measures in this list are in place
- Application rates as recommended by the Safe Sludge Matrix
- Reduce the amount of lime-treated sewage sludge that is applied to land

Figure 3-5 Backwards inference by setting Annoyance from odour to P(Not annoyed) = 100% (cf. Figure 3-2)



3.2. Heavy metals and inorganics

3.2.1. Background

In Europe and North America, regulations have been in place to limit concentrations of heavy metals in sewage sludge intended for land application. The USEPA adopted regulations in 1993 (USEPA, 1993) to set concentration limits for nine metals (Arsenic (As), Cadmium (Cd), Copper (Cu), Lead (Pb), Mercury (Hg), Molybdenum (Mo), Nickel (Ni), Selenium (Se), and Zinc (Zn)). In Europe, EU sewage sludge Directive 86/278/EEC sets regulatory limits for seven heavy metals (Cd, chromium (Cr), Pb, Ni, Cu, Hg and Zn) in soil and sewage sludge itself (Kelessidis & Stasinakis, 2012). In different countries, however, various models are adopted for regulating the application of sewage sludge to land. In the UK and US a risk-based approach has been adopted (Smith, 2009), whereas a precautionary principle is preferred in the majority of EU member states (Schoof & Houkal, 2005). This divergence has resulted in more stringent concentration limits (either proposed or enacted) in Europe than in the US for metals (McGrath et al., 1994).

The presence of heavy metals in sewage sludge is primarily due to the mixing of industrial wastewater with domestic sewage (Cornu et al., 2001). Even though there are legislative frameworks in place to regulate concentrations of heavy metals in sewage sludge, there remains scepticism over the safety of land application of sewage sludge due to potential risk of metal accumulation in the soil (Mamindy-Pajany et al., 2014) and in food crops (Latare et al., 2014), or of transport to groundwater and eventually the food chain (Kidd et al., 2007).

3.2.2. Risk analysis

Due to the potential risks of heavy metals entering food chain, many studies have investigated their uptake by food crops grown on land where sewage sludges have been applied (e.g. Latare et al., 2014; McBride et al., 1999; 2004). The results of these studies have confirmed metal accumulation in crops, therefore the authors concluded that uptake of metals by crop plants is a potential route of exposure to humans via the food chain. Under some circumstances (localised food production and consumption combined with long-term elevates use of sewage sludge, and high levels of consumption by vulnerable individuals), this might represent a health risk (Hough et al., 2003). However, many of these studies were laboratory-based, with the plants exposed to unrealistically high concentrations of heavy metals, or the plants were cultivated in media that favoured the uptake of heavy metals (e.g. aqueous growth media, low pH), or that had been purposely enriched with heavy metals and are therefore not representative of standard agricultural practices (Prosser & Sibley, 2015). Other studies based on field and greenhouse trial are more realistic compared with agriculture practise, however due to crop varieties, soil characteristics and many other factors, the measured uptake of heavy metals can vary significantly (Harrison et al., 1999). These experimental difficulties therefore lead to uncertainties in the environmental and human health risk assessment.

The SNIFFER report (SNIFFER 2008) reported minimal risks associated with the presence of heavy metals in sewage sludge. Hosseini Koupaie & Eskicioglu (2015) commented that most of the previous risk assessments of heavy metals in biosolids were implemented using a deterministic (non-probabilistic) approach, which may not be realistic due to the highly variable parameters involved in the risk assessment. Using a probabilistic risk analysis approach, the authors concluded that potential risks to human health were acceptable even when considering a long-term application of sewage sludge to land at an annual rate of 100 t ha^{-1}.

In addition, under usual agricultural soil conditions, potential risks from heavy metals are directly related to the concentrations of these contaminants in sewage sludges. In a European scale study conducted by the European Commission's (EC's) Joint Research Committee (JRC), 114 chemicals including 21 metal elements were evaluated in 63 sewage sludge samples originating from 15 different countries. The study found that concentrations of all regulated heavy metals were well below the legislative limits. Therefore, the study concluded that monitored concentrations did not justify the introduction of new threshold limits for considered parameters within the Directive as no unacceptable risk had been identified (EC, 2012). This is in agreement with a recent UK based study assessing the statutory limits of HM set by the UK Sludge (Use in Agriculture) Regulations in a long-term sludge experiment (Charlton et al., 2016).

Due to these findings, the low risk reported by SNIFFER 2008 and the fact that the regulation of heavy metals in sewage sludge has a long history and is well developed, no formal risk analysis was undertaken in this study.

3.3. Organic contaminants

3.3.1. Background

Organic contaminants are chemical substances that persist in the environment, bioaccumulate through the food web, and pose a risk of causing adverse effects to human health. They include industrial chemicals (such as polychlorinated biphenyls - PCBs), unintentional by-products of industrial processes (such as dioxins and furans), incomplete products of combustion (e.g. polycyclic aromatic hydrocarbons - PAHs), flame retardants (such as Polybrominated diphenyl ethers - PBDEs) and plasticisers (such as Benzyl butyl phthalate - BBP). Organic contaminants can enter waste water treatment plants (WWTPs) from a variety of sources including urban and agricultural run-off, domestic wastewater and industrial point source discharges and also via atmospheric deposition. Organic contaminants are typically lipophilic (fat soluble) and hydrophobic, and sorption to sewage sludge solids is the primary pathway for their removal from wastewater.

The application of sewage sludge to land, whether on to the soil surface or through incorporation in to the topsoil, will directly expose the soil and any associated crops to organic contaminants. Similarly, atmospheric deposition will result in soil exposure to organic contaminants and potential crop uptake via roots or leaves. Some non-persistent organic pollutants, such as lesser molecular mass PAHs, can be degraded during composting (Brandli et al., 2007) and in the soil, but other contaminants such as PCBs or polychlorinated dibenzodioxins and furans (PCDDs/Fs) could accumulate in soil when contaminated sewage sludge is applied repeatedly (Umlauf et al., 2011).

The migration of pollutants, in particular from soil to plants, could facilitate a potential entry pathway into the human food chain and subsequent human exposure. The magnitude of plant uptake, as well as the pathway by which organic contaminants enter vegetation is a function of the chemical and physical properties of each pollutant. Experiments and model simulations have shown that persistent, polar (log octanol:water partition coefficient, Kow < 3) and non-volatile (dimensionless Henry's constant (the proportionality constant in Henry's gas law that states that the amount of dissolved gas is proportional to its partial pressure in the gas phase) < 10^{-6}) contaminants generally have the highest potential for accumulation from soil into plants. Concentrations in roots and leaves may even exceed the concentrations in soil (in some cases by several orders of magnitude), which among other things is because the water content in roots (up to 95%) usually is higher than in soils (about 30%). Volatile contaminants have a low potential for accumulation, because they quickly escape to air (Trapp and Legind, 2011).

3.3.2. Emerging contaminants

It is not the intention of this study to reiterate what has already been reported in SNIFFER (2008) and elsewhere on organic contaminants in sewage sludge. Instead, the focus of this work is on potential human health risks associated with so called ‘emerging’ and ‘priority’ organic contaminants.

Research has shown that for most ‘traditional’ organic contaminants plant uptake is minimal, strong sorption to the soil matrix prevents groundwater contamination and restrictions on applications to grazing grassland reduces the likelihood of ingestion by livestock. The concentration of ‘traditional’ chemicals in sludge has typically declined over time due to effective source control. Many are governed by the Stockholm Convention, which has eliminated or restricted the use of the organic chemicals deemed to be of most risk. However, ‘emerging’ chemicals (i.e. those that have not been well studied) may pose risks that are not well characterised or have not yet been identified. The 2017 EU Future Brief recognised the importance of newly emerging organic chemicals such perfluorochemicals (PFCs) and polybrominated diphenyl ethers-PBDEs (Science for Environment Policy, 2017). It acknowledged the smaller evidence base for these chemicals, which may behave in a different way to some older organic chemicals, for example binding to protein. In addition, a range of European industry organisations, (e.g. European Environmental Bureau-EEB and the European Sustainable Phosphorus Platform-ESPP), published a joint position statement in 2017 on the need for data and research on organic chemicals (and pharmaceuticals) in sewage sludge. This highlighted the threat to land application due to the real or perceived concerns about organic contaminants and the lack of information regarding “emerging” contaminants.

A number of reviews have considered which ‘emerging’ chemicals should be prioritised in future research (e.g. Clarke et al., 2016; Stewart et al., 2016; Thomadi et al., 2016; McCarthy et al., 2015; Jensen et al., 2012; Clarke & Smith, 2011, Table 3-4) and these were used to select the organic chemicals that have been considered in more detail in this report (Table 3-5). It should be noted that where a chemical is *sensu stricto* an organic chemical but is used primarily as a pharmaceutical or in personal care products, this chemical has not been included in the risk assessment presented here but in Section 3.4.

Table 3-4 Identification of priority chemicals in the literature

Clarke and Smith 2011[1]	Jensen *et al.* (2012)[2]	Thomaidi *et al.* (2016)[3]	Stewart *et al.* (2016)[4]	Clarke *et al.* (2016)[5]
10/11: PFCs 9/11: PCAs; PCNs 7/11: PBDEs; OTs; TCS; TCC 6/11: Benzothiazoles 5/11: Antibiotics, pharmaceuticals 3/11: Synthetic musks 2/11: BPA; QACs; Steroids 1/11: PAEs; PDMSs	Low risk [b] • BFRs • Musks • Pharmaceuticals • PCBs • PFCs [c] For future review [d] • PCAs • PCNs • TCC • TCS • Parabens	Synthetic phenolic compounds • NP • NP1EO • NP2EO • TCS Siloxanes • Decamethylcyclopentasiloxane • Dodecamethylcyclohexasiloxane • Dodecamethylpentasiloxane • Tetradecamethylhexasiloxane Benzothiazoles • 2-hydroxybenzothiazole Pharmaceuticals • Caffeine • Benzothiazoles	Flame retardants • BDE 77, 99 and 209 • TDCP, • TPP • TCPP Plasticisers • DEHP • BBP • Bisphenol A Perfluorinated compounds • PFOS/PFOA	• NP, NP1EO, NP2EO (1-3) • TCC (4) • TCS (5) • BPA (6) • Carbamazephine (7) • PBDE (8) • PCBs (9) • PFOA (10) • PFOS (11) • PCDD/Fs (14)

[1] Ranking of research priorities for emerging organic contaminants in biosolids (10 highest priority to 1 least priority) perfluorochemicals (PFCs); polychlorinated alkanes (PCAs); polychlorinated naphthalenes (PCNs); polybrominated diphenyl ethers (PBDEs); organotins (OTs); triclosan (TCS); triclocarban (TCC);. bisphenol A (BPA); quaternary ammonium compounds (QACs); phthalate acid esters (PAEs) and polydimethylsiloxanes (PDMSs)

[2]Prioritisation and selection of chemicals to assess the risk to soil organisms from the application of sludge (brominated flame retardants (BFR), musks, pharmaceuticals, PCBs and PFCs). Identification of chemicals for future evaluation.

[3]Greece was used as case study and the environmental risk associated with the existence of 99 emerging OCCs in sludge-amended soil was estimated using risk quotient (RQ) approach. Chemical with a risk quotient >1 are listed.

[4]Identification of indicator compounds for use in the assessment of organic chemical removal during wastewater treatment and their fate in receiving environments (Tris[2-chloro-1-(chloromethyl)ethyl]phosphate (TDCP); Triphenyl phosphate (TPP); Tris (1-chloro-2-propyl) phosphate (TCPP); Di (2-ethyhexyl)phthalate (DEHP); benzyl butyl phthalate (BBP); = perfluorooctanesulfonic acid (PFOS); perfluorooctanoic acid (PFOA)).

[5]A quantitative risk ranking model was developed for human exposure to emerging contaminants following biosolids application to Irish agricultural land. Chemicals are ranked by predicted environmental concentration in soil.

3.3.3. Risk assessment

Chemicals listed in Table 3-4 were included in the risk assessment if; (i) their primary application was not for pharmaceutical or personal care products, (ii) if reliable distribution coefficients were available that satisfied the requirements of the multi-media fugacity models (Section 2.2.2). The full list of organic chemicals, their physico-chemical properties and reference doses (RfD) are listed in Table 3-5.

Table 3-5 Physico-chemical data and reference doses for organic chemicals entered into the risk assessment

Chemical	Water solubility (mg l^{-1} @ 25°C)	Vapour pressure (mm Hg @ 25°C)	Henry's constant (atm-m^3 mol^{-1})	Log K_{OW}	RfD (mg kg^{-1} d^{-1}) / Uncertainty Factor
Benzo(a)anthracene	$9.40x10^{-3}$ (May et al. 1978)	$2.10x10^{-7}$ (Sonnefeld et al. 1983)	$1.20x10^{-5}$ (NCCT)	5.76 (Wang et al. 1986)	$1.40x10^{-3}$ / 3000 (Hoogenboom et al., 2003)
Benzo(a)pyrene	$1.62x10^{-3}$ (May et al. 1983)	$5.49x10^{-9}$ (Murray et al. 1974)	$4.57x10^{-7}$ (NCCT)	6.13 (Demaagd et al. 1998)	$3.00x10^{-4}$ / 3000 (USEPA IRIS)
Benzo(b)fluoranthene	$1.50x10^{-3(a)}$ (Yalkowsky et al. 2010)	$5.00x10^{-7(b)}$ (Coover & Sims 1987)	$6.57x10^{-7}$ (NCCT)	5.78 (Wang et al. 1986)	$1.40x10^{-3}$ / 3000 (Hoogenboom et al., 2003)
Benzo(k)fluoranthene	$7.60x10^{-4}$ (USEPA 1987)	$9.65x10^{-10}$ (Murray et al. 1974)	$5.84x10^{-7}$ (NCCT)	6.11 (Demaagd et al. 1998)	$1.40x10^{-3}$ / 3000 (Hoogenboom et al., 2003)
Chrysene	$2.00x10^{-3}$ (Miller et al. 1985)	$6.23x10^{-9}$ (Hoyer & Peperle 1958)	$9.40x10^{-7}$ (NCCT)	5.73 (Hansch et al. 1995)	$1.40x10^{-2}$ / 3000 (Hoogenboom et al., 2003)
Indeno(1,2,3-cd)pyrene	$6.20x10^{-2(b)}$ (Sims & Overcash 1983)	$1.25x10^{-10}$ (USEPA 2012)	$3.48x10^{-7}$ (NCCT)	6.70 (USEPA 2012)	$4.90x10^{-5}$ / 10000 (USEPA Chem Dash)
Naphthalene	$3.10x10^{1}$ (Pearlman et al. 1984)	$8.50x10^{-2}$ (Ambrose et al. 1975)	$4.40x10^{-4}$ (NCCT)	3.30 (Hansch et al. 1995)	2.00×10^{-2} /3000 (Shopp et al., 1984)
PCB 28	$1.17x10^{-1}$ (USEPA OPERA)	$3.46x10^{-4}$ (USEPA OPERA)	$2.05x10^{-4}$ (USEPA OPERA)	5.64 (USEPA OPERA)	2.00×10^{-5} / 300 (Arnold et al., 1993a; 1993b; Tryphonas et al., 1989; 1991a; 1991b)
PCB 52	$3.00x10^{-2}$ (USEPA OPERA)	$9.90x10^{-5}$ (USEPA OPERA)	$3.50x10^{-5}$ (USEPA OPERA)	6.10 (USEPA OPERA)	
PCB 95	$1.00x10^{-2}$	$1.45x10^{-5}$	$9.34x10^{-5}$	6.56	

Chemical	Water solubility (mg l^{-1} @ 25°C)	Vapour pressure (mm Hg @ 25°C)	Henry's constant (atm-m^3 mol^{-1})	Log K_{OW}	RfD (mg kg^{-1} d^{-1}) / Uncertainty Factor
	(USEPA OPERA)	(USEPA OPERA)	(USEPA OPERA)	(USEPA OPERA)	
PCB 101	$1.00x10^{-2}$ (USEPA OPERA)	$1.62x10^{-5}$ (USEPA OPERA)	$8.39x10^{-5}$ (USEPA OPERA)	6.43 (USEPA OPERA)	
PCB 118	$1.00x10^{-3}$ (USEPA OPERA)	$1.36x10^{-5}$ (USEPA OPERA)	$7.86x10^{-5}$ (USEPA OPERA)	6.77 (USEPA OPERA)	
PCB 132	$1.00x10^{-3}$ (USEPA OPERA)	$3.19x10^{-6}$ (USEPA OPERA)	$3.91x10^{-5}$ (USEPA OPERA)	7.24 (USEPA OPERA)	
PCB 138	$1.00x10^{-3}$ (USEPA OPERA)	$4.39x10^{-6}$ (USEPA OPERA)	$2.46x10^{-5}$ (USEPA OPERA)	7.33 (USEPA OPERA)	
PCB 149	$1.00x10^{-3}$ (USEPA OPERA)	$5.53x10^{-6}$ (USEPA OPERA)	$3.34x10^{-5}$ (USEPA OPERA)	7.16 (USEPA OPERA)	
PCB 153	$1.00x10^{-3}$ (USEPA OPERA)	$4.01x10^{-6}$ (USEPA OPERA)	$3.08x10^{-5}$ (USEPA OPERA)	6.97 (USEPA OPERA)	
PCB 174	$1.00x10^{-3}$ (USEPA OPERA)	$1.05x10^{-6}$ (USEPA OPERA)	$9.45x10^{-6}$ (USEPA OPERA)	7.71 (USEPA OPERA)	
PCB 180	$1.00x10^{-3}$ (USEPA OPERA)	$1.02x10^{-6}$ (USEPA OPERA)	$7.63x10^{-6}$ (USEPA OPERA)	7.65 (USEPA OPERA)	
2,3,7,8-TeCDD	$2.00x10^{-4}$ (Shiu et al. 1988)	$1.50x10^{-9}$ (Rordorf 1987)	$8.95x10^{-6}$ (USEPA OPERA)	6.80 (Shiu et al. 1988)	2.00×10^{-9} / 9.6 (COT)
1,2,3,7,8-PeCDD	$1.93x10^{-5}$ (USEPA OPERA)	$6.03x10^{-10}$ (USEPA OPERA)	$1.09x10^{-5}$ (USEPA OPERA)	6.58 (USEPA OPERA)	2.00×10^{-9} / 9.6 (COT)
1,2,3,4,6,7,8-HpCDD	$1.90x10^{-3}$ (Miyata et al. 1989)	$7.40x10^{-8}$ (Nestrick 1980)	$2.18x10^{-5}$	8.11 (USEPA OPERA)	2.00×10^{-7} / 9.6 (COT)

Chemical	Water solubility (mg l^{-1} @ 25°C)	Vapour pressure (mm Hg @ 25°C)	Henry's constant (atm-m^3 mol^{-1})	Log Kow	RfD (mg kg^{-1} d^{-1}) / Uncertainty Factor
			(Hine & Mookerjee 1975)		
2,3,4,7,8-PeCDF	$4.19x10^{-4}$ (USEPA OPERA)	$2.01x10^{-9}$ (USEPA OPERA)	$3.97x10^{-5}$ (USEPA OPERA)	6.62 (USEPA OPERA)	2.00×10^{-9} / 9.6 (COT)
1,2,3,4,7,8-HxCDF	$2.40x10^{-6}$ (USEPA OPERA)	$2.13x10^{-9}$ (USEPA OPERA)	$9.31x10^{-6}$ (USEPA OPERA)	7.45 (USEPA OPERA)	2.00×10^{-8} / 9.6 (COT)
1,2,3,6,7,8-HxCDF	$8.53x10^{-4}$ (USEPA OPERA)	$2.12x10^{-9}$ (USEPA OPERA)	$9.31x10^{-6}$ (USEPA OPERA)	7.45 (USEPA OPERA)	2.00×10^{-8} / 9.6 (COT)
2,3,4,6,7,8-HxCDF	$8.55x10^{-4}$ (USEPA OPERA)	$2.12x10^{-9}$ (USEPA OPERA)	$9.31x10^{-6}$ (USEPA OPERA)	7.45 (USEPA OPERA)	2.00×10^{-8} / 9.6 (COT)
Nonylphenol	$7.00x10^{0}$ (Yalkowsky & Dannenfelser 1992)	$8.18x10^{-4}$ (Bidleman & Renberg 1985)	$3.50x10^{-5}$ (NCCT)	5.76 (Itokawa et al. 1989)	$7.50x10^{-3}$ / 1000 (USEPA Chem Dash)
Nonylphenol diethoxylate	$1.05x10^{0}$ (ECHA 2013)	$9.14x10^{-9}$ (ECHA 2013)	$2.56x10^{-9}$ (ECHA 2013)	5.30 (ECHA 2013)	$7.00x10^{-3}$ / 10000 (derived from USGS screening level)
PBDE 99	$1.33x10^{-2}$ (EU 2001)	$3.50x10^{-7}$ (Hardy & Smith 1999)	$2.50x10^{-5}$ (NCCT)	6.84 (Geyer et al. 2000)	$1.00x10^{-4}$ / 3000 (USEPA IRIS)
PBDE 209	$1.00x10^{-4}$ (ECHA 2015a)	$6.96x10^{-11}$ (Lorber & Cleverly 2010)	$1.20x10^{-8}$ (NCCT)	9.97 (Environment Canada 2010)	$2.00x10^{-4}$ / NR (ASTDR RAIS)
PFOA	$3.30x10^{3}$ (Inoue et al. 2012)	$3.16x10^{-2}$ (Bhhatarai & Gramatica 2011)	$2.02x10^{-10}$ (USEPA OPERA)	4.81 (USEPA 2012)	$2.00x10^{-5}$ / NR (ASTDR RAIS)

Chemical	Water solubility (mg l^{-1} @ 25°C)	Vapour pressure (mm Hg @ 25°C)	Henry's constant (atm-m^3 mol^{-1})	Log K_{OW}	RfD (mg kg^{-1} d^{-1}) / Uncertainty Factor
PFOS	$3.20x10^{-3}$ (USEPA 2012)	$2.00x10^{-3}$ (USEPA 2012)	$1.85x10^{-11}$ (USEPA OPERA)	4.49 (USEPA 2012)	$3.00x10^{-5}$ / NR (ATSDR RAIS)
TCDP	$7.00x10^{0}$ (Yalkowsky et al. 2010)	$2.86x10^{-7}$ (USEPA 2012)	$2.60x10^{-9}$ (NCCT)	3.65 (Chem Insp Test Inst 1992)	$2.00x10^{-2}$ / NR (ATSDR RAIS)
DEHP	$2.70x10^{-1}$ (DeFoe et al. 1990)	$1.42x10^{-7}$ (Hinckley et al. 1990)	$2.70x10^{-7}$ (NCCT)	7.60 (de Bruijin et al. 1989)	$2.00x10^{-2}$ / 1000 (USEPA IRIS)
BBP	$2.69x10^{0}$ (Howard et al. 1985)	$8.25x10^{-6}$ (Howard et al. 1985)	$1.30x10^{-6}$ (NCCT)	4.73 (Ellington & Floyd 1996)	$2.00x10^{-1}$ / 1000 (USEPA IRIS)

[(a)]No temperature specified.

[(b)]20°C

Initial concentrations in sewage sludge were set based on the 'realistic worst case' paradigm (WRAP 2016c; Hough et al. 2012). A series of recent reviews documenting measured concentrations in sewage sludge were used and starting concentrations were derived from 90%ile values reported in these data sets (Table 3-6).

Table 3-6 Initial concentrations of organic contaminants in sewage sludge prior to spreading

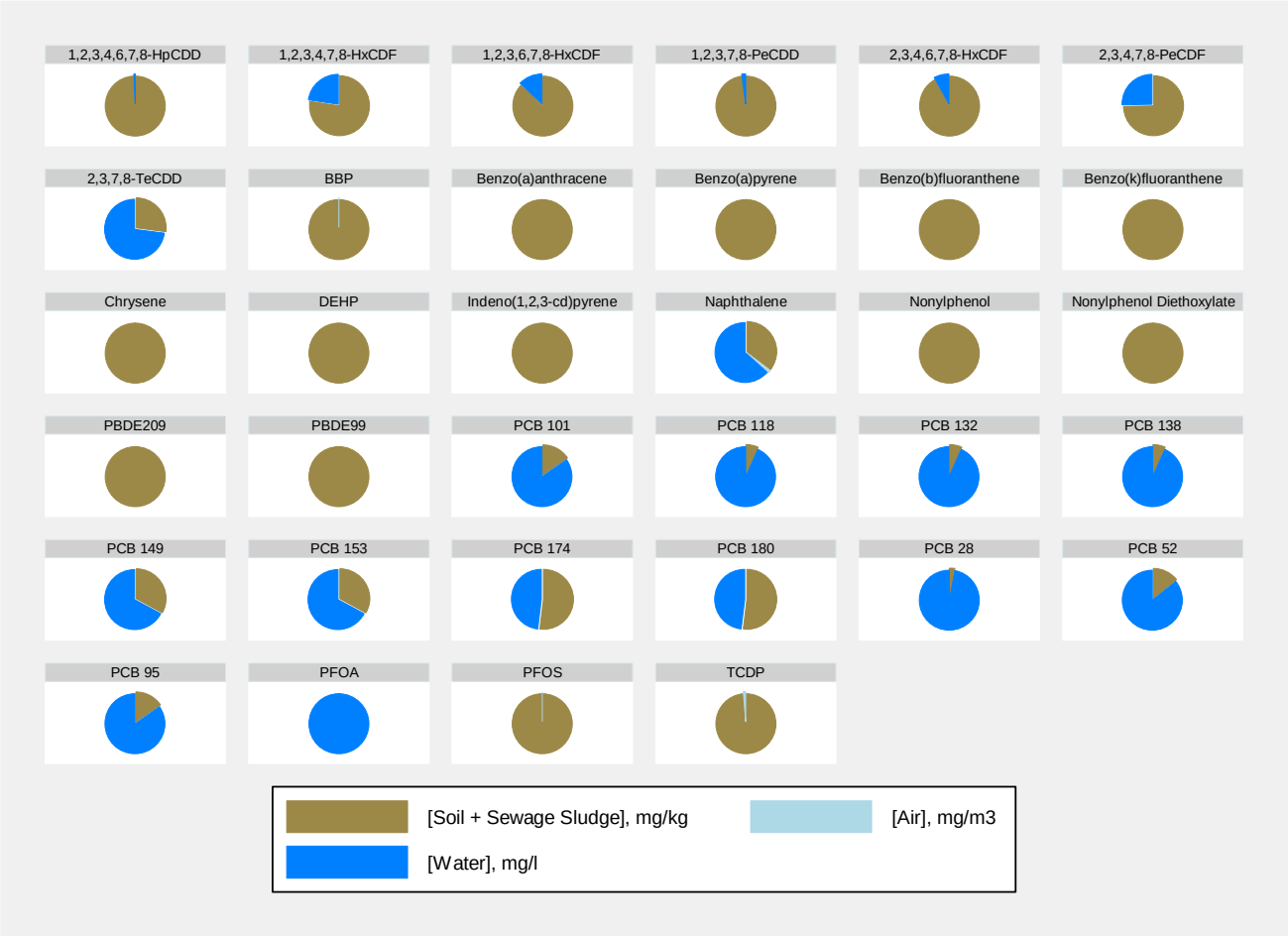
Chemical (IUPAC)	Concentration in Sewage Sludge (mg kg^{-1})	Reference
Benzo(a)anthracene (benzo[a]anthracene)	$8.10x10^{-1}$	WRAP et al. 2016c
Benzo(a)pyrene (benzo[a]pyrene)	$6.20x10^{-1}$	
Benzo(b)fluoranthene (benzo[b]fluoranthene)	$8.90x10^{-1}$	
Benzo(k)fluoranthene (benzo[k]fluoranthene)	$4.80x10^{-1}$	
Chrysene (Chrysene)	$1.10x10^{0}$	
Indeno(1,2,3-cd)pyrene (Indeno[1,2,3-cd]pyrene)	$9.60x10^{-1}$	
Naphthalene (Naphthalene)	$1.40x10^{-1}$	
PCB 28 (2,4-dichloro-1-(4-chlorophenyl)benzene)	$2.00x10^{-2}$	
PCB 52 (1,4-dichloro-2-(2,5-dichlorophenyl)benzene)	$3.00x10^{-2}$	
PCB 95 (1,2,4-trichloro-3-(2,5-dichlorophenyl)benzene)	$3.00x10^{-2}$	
PCB 101 (1,2,4-trichloro-5-(2,5-dichlorophenyl)benzene)	$7.00x10^{-2}$	
PCB 118 (1,2,4-trichloro-5-(3,4-dichlorophenyl)benzene)	$1.30x10^{-1}$	
PCB 132 (1,2,3-trichloro-4-(2,3,6-trichlorophenyl)benzene)	$7.00x10^{-2}$	
PCB 138 (1,2,3-trichloro-4-(2,4,5-trichlorophenyl)benzene)	$1.30x10^{-1}$	
PCB 149 (1,2,4-trichloro-3-(2,4,5-trichlorophenyl)benzene)	$8.00x10^{-2}$	
PCB 153 (1,2,4-trichloro-5-(2,4,5-trichlorophenyl)benzene)	$1.40x10^{-1}$	
PCB 174 (1,2,3,4-tetrachloro-5-(2,3,6-trichlorophenyl)benzene)	$2.00x10^{-2}$	
PCB 180	$6.00x10^{-2}$	

Chemical (IUPAC)	Concentration in Sewage Sludge (mg kg^{-1})	Reference
(1,2,3,4-tetrachloro-5-(2,4,5-trichlorophenyl)benzene)		
2,3,7,8-TeCDD (2,3,7,8-tetrachlorodibenzo-p-dioxin)	$1.20x10^{-6}$	
1,2,3,7,8-PeCDD (1,2,3,7,8-pentachlorodibenzo-p-dioxin)	$8.80x10^{-6}$	
1,2,3,4,6,7,8-HpCDD (1,2,3,4,6,7,8-heptachlorodibenzo-p-dioxin)	$1.20x10^{-4}$	
2,3,4,7,8-PeCDF (2,3,4,7,8-pentachlorodibenzofuran)	$1.10x10^{-5}$	
1,2,3,4,7,8-HxCDF (1,2,3,4,7,8-hexachlorodibenzofuran)	$1.50x10^{-5}$	
1,2,3,6,7,8-HxCDF (1,2,3,6,7,8-hexachlorodibenzofuran)	$1.60x10^{-5}$	
2,3,4,6,7,8-HxCDF (2,3,4,6,7,8-hexachlorodibenzofuran)	$1.40x10^{-5}$	
Nonylphenol (4-nonylphenol)	$2.38x10^{2}$	Gibson et al. 2005
Nonylphenol diethoxylate (2-[2-(4-nonylphenoxy)ethoxy]ethanol)	$1.35x10^{2}$	González et al. 2010
PBDE 99 (1,2,4-tribromo-5-(2,4-dibromophenoxy)benzene)	$2.45x10^{0}$	Harrison et al. 2006
PBDE 209 (1,2,3,4,5-pentabromo-6-(2,3,4,5,6-pentabromophenoxy)benzene)	$2.45x10^{0}$	Harrison et al. 2006
PFOA (2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-pentadecafluorooctanoic acid)	$2.40x10^{-5}$	Zareitalabad et al. 2013
PFOS (1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluorooctane-1-sulfonic acid)	$1.10x10^{-5}$	Zareitalabad et al. 2013
TDCP (tris(1,3-dichloropropan-2-yl) phosphate)	$2.60x10^{-1}$	Marklund et al. 2005

Chemical (IUPAC)	Concentration in Sewage Sludge (mg kg^{-1})	Reference
DEHP (bis(2-ethylhexyl) benzene-1,2-dicarboxylate)	$2.70x10^{0}$	Bright & Healey 2003
BBP (2-O-benzyl 1-O-butyl benzene-1,2-dicarboxylate)	$3.80x10^{-1}$	Bright & Healey 2003

Combining partition coefficients (Table 3-5) with initial concentrations in sewage sludge (Table 3-6) within Equations 2-6 - 2-9, it was possible to estimate the concentrations of each organic contaminant that would remain in soil vs. migrate to soil pore water or soil pore air (Figure 3-6). Those contaminants with the greatest propensity to partition into the soil pore water are subsequently more likely to be taken up by food crops or pasture, and therefore enter the human food chain.

Figure 3-6 Partitioning [ppm] of organic contaminants between soil amended with sewage sludge, soil pore water, and soil air spaces given initial starting concentrations in sewage sludge (Table 3-6).



Concentrations of each organic contaminant in root crops, leafy vegetables, cereal crops, groundwater (private drinking supplies) and volatilisation to air were estimated using Equations 2-10, 2-11 & 2-12. These were related to human exposure by assuming that a proportion of produce consumed was from the locations where sewage sludge was applied, a proportion of the air breathed in was derived from air into which contaminants had volatilised into, and a proportion of the water consumed was derived from a supply into which contaminants had leached. Using realistic worst case assumptions, the following proportions were selected (Table 3-7) and estimates of exposure for each individual organic contaminant calculated (Figure 3-7). For comparability with other studies, exposure attributable to the sum of PAHs (ΣPAH, Figure 3-8), PCBs (ΣPCB, Figure 3-9), and PCDD/Fs (ΣPCDD/F, Figure 3-10), were also calculated.

Table 3-7 Intake rates of contaminated media derived using 'realistic worst case' principles.

Media	Consumption rate of contaminated media	Assumptions
Water	1.44×10^{-5} L d^{-1}	An individual whose main household supply is a private well in close proximity to sewage spreading activities. Assuming an average consumption rate of 1.2 L d^{-1} (Hough et al. 2010b) and a total period per year when the water supply is affected by sewage sludge applications of 28 days (i.e. fractional exposure of 0.12), averaged over 1 year. A dilution rate of 10^4 is assumed.
Air	1.38×10^{-5} m^3 d^{-1}	An individual who resides within 1 km of sewage spreading activities. Assumes 100% partially conducive weather conditions (see Figure 3-2). Assumes inhalation rate of 180 m^3 d^{-1} outdoors (Hough et al. 2004) and a total period of 28 days when the air is affected by sewage sludge volatiles. A dilution rate of 10^6 is assumed.
Soil	1.76×10^{-5} kg d^{-1}	Assumes the 95th%ile soil ingestion rate for an adult (Hough et al. 2004 and references therein) and that 25% of all soil ingestion is derived from locations where sewage sludge has been spread. Given that all organic chemicals adsorb and accumulate in soil, the fractional exposure is considered to be 1, i.e. 365.25 days per year.
Roots	1.10×10^{-2} kg d^{-1}	Assumes the 95th%ile root vegetable (including potatoes) ingestion rate for an adult (Hough et al. 2004) and that 10% of all ingested vegetables are derived from local sources where sewage sludge has been applied to the soil.

Leaves	$8.08x10^{-3}$ kg d^{-1}	Assumes the 95th%ile leafy vegetable (including salad veg) ingestion rate for an adult (Hough et al. 2004) and that 10% of all ingested vegetables are derived from local sources where sewage sludge has been applied to the soil.
Cereals	$8.40x10^{-03}$ kg d^{-1}	Assumes the 95th%ile bread consumption rate 2010 for an adult male (O'Connor 2012) and that 10% of all ingested bread was made with flour derived from locally grown cereals where sewage sludge has been applied to the soil.

Figure 3-7 Relative exposure profiles for each organic contaminant via six different exposure medium (water, air, soil, root vegetables (including potatoes), leafy vegetables (including salad vegetables), and cereals.

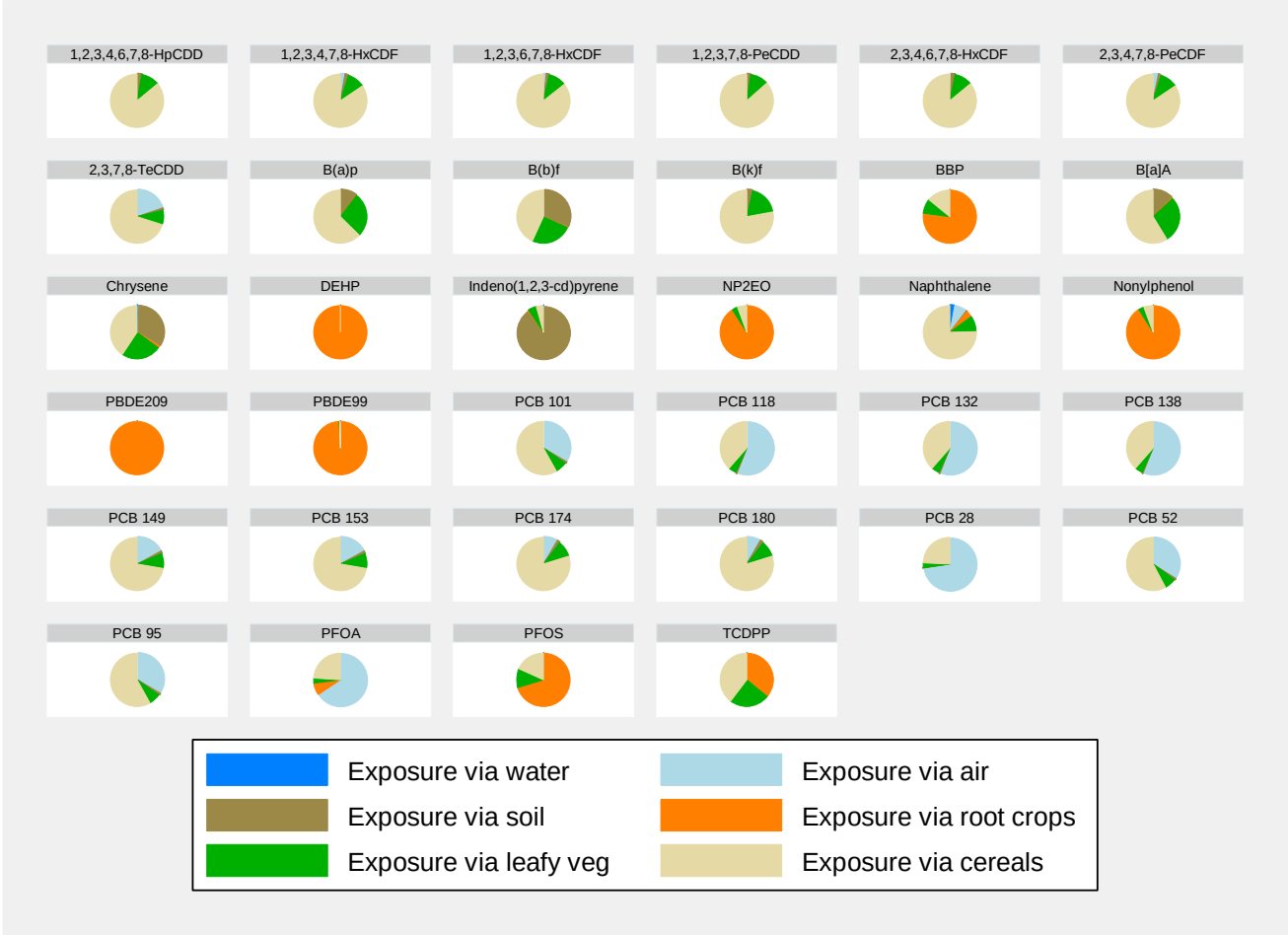


Figure 3-8 Exposure: Sum of PAHs

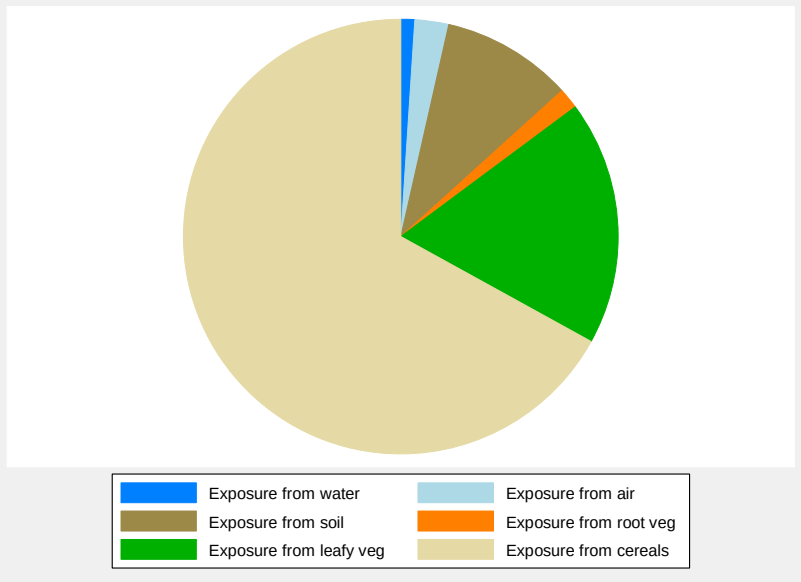


Figure 3-9 Exposure: Sum of PCBs

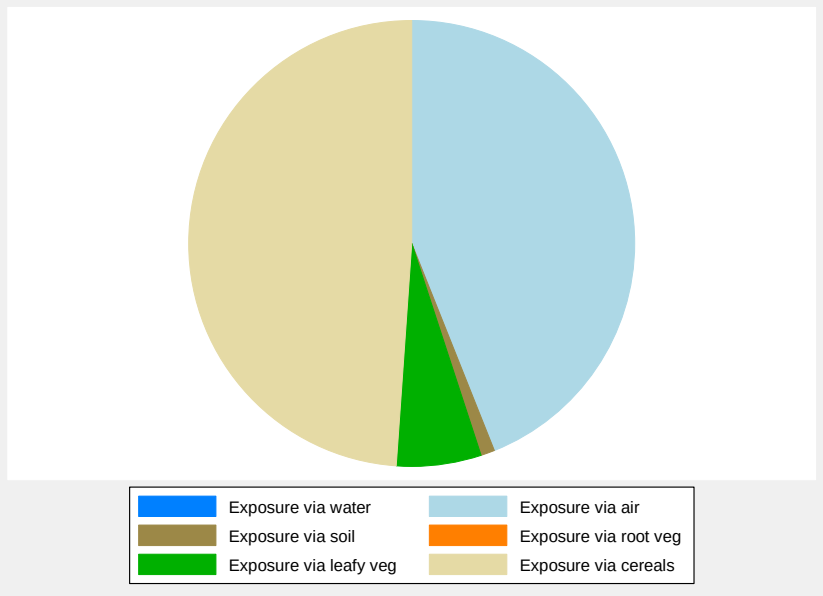
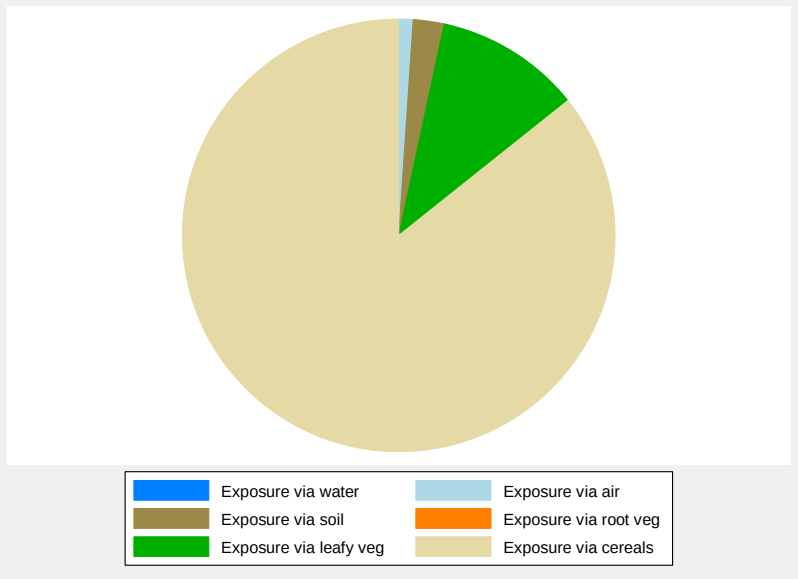
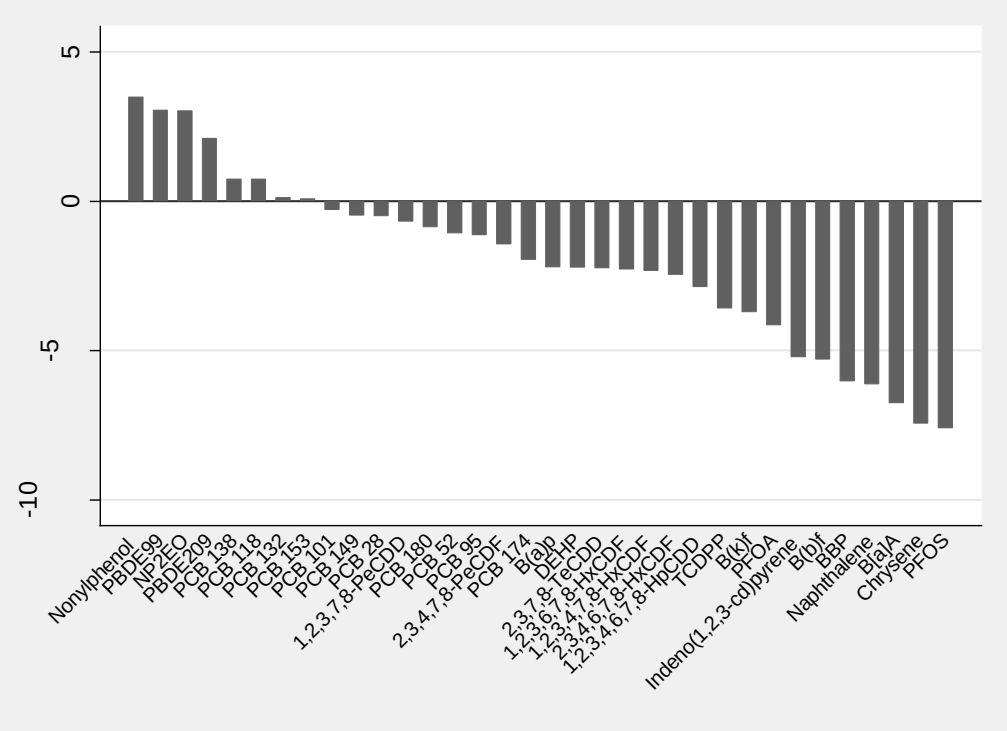


Figure 3-10 Exposure: Sum of PCCD/Fs



Estimates of exposure (Figure 3-7) were compared to the reference doses (RfDs, Table 3-5) in order to derive the Hazard Quotient, HQ as defined in Equation 2-15 (Figure 3-11).

Figure 3-11 Estimates of hazard quotient, log(HQ), for each organic contaminant based on the ratios of the exposures described in Figure 3-7 and the reference doses (RfD) for each organic contaminant as detailed in Table 3-5. A log(HQ) > 0 indicates that further investigation may be required.



Conclusions

Even with ‘realistic worst case’ assumptions, the majority of the organic contaminants investigated posed no appreciable risk to local inhabitants.

The following organic contaminants returned a hazard quotient that was significantly greater than one; the detergents nonylphenol and nonylphenol diethoxylate (NP2EO), and the flame retardants PBDE-99 and PBDE-209 (Figure 3-11); suggesting that if the worst case assumptions described above held true, these individuals would experience deleterious health effects due to lifetime exposure to these compounds as a result of sewage sludge application to land. It is interesting to note that for all these chemicals, the exposure is dominated by consumption of root vegetables (Figure 3-7) and therefore these exposures (if proven to be real) could be mitigated by simply restricting the use of sewage sludge in root vegetable production. There are also opportunities to reduce concentrations of these chemicals in sludges via the water and sludge treatment processes, with processes such as anaerobic digestion proving effective at partially removing a number of the agents highlighted (see Table 4-1). It should be noted that the RfD for nonylphenol diethoxylate has especially large uncertainties as it is derived from an USGS screening level for drinking water rather than from a toxicologic point of departure.

The analysis also suggests that PCB 138 and PCB 118 might also pose a risk, however given the ‘realistic worst case’ assumptions it is likely that this risk is minimal. The main exposure route for the PCBs is via air and therefore represents a short-term exposure while sewage sludge handling is taking place. As seen in Section 3.2, avoidance of the most conducive weather conditions as well as the use of buffer zones may reduce this exposure.

These estimates of HQ are based on realistic worst case assumptions, thus the exposure scenarios will only apply to a very small proportion of the population (if at all). The vast majority of individuals living close to sewage sludge application activities are not going to be exposed via drinking water, and it is unlikely that they will be consuming 10% of their food intake from locally-grown produce.

The use of ‘realistic worst case’ scenarios is a conservative approach aimed at protecting the most vulnerable in society and hence the approach tends to drive up risk estimates. This is helpful as it gives a level of confidence where we identify compounds that despite the worst case assumptions pose little risk to inhabitants. The recommendations made here have been made on the assumption that the potential exposures highlighted are subsequently found to be real and reasonably widespread.

3.4. Pharmaceuticals and personal care products

3.4.1. Background

Pharmaceuticals and personal care products (PPCPs) are used for health or cosmetic purposes, and include products used on both animals and humans. A diverse collection of thousands of chemical substances can be classed as PPCPs, comprising prescription and over-the-counter medicines, veterinary medicines, fragrances and cosmetics.

Pharmaceuticals are designed to modify biochemical and physiological functions of biological systems in humans and animals – these properties can unintentionally influence the behaviour and fate of microorganisms in the soil or water should their habitats become contaminated with these compounds. Major groups of pharmaceuticals include antibiotics including antimicrobials, antivirals, etc., disinfectants, steroids, hormones and nutraceuticals, as well as their various degradation products. Some pharmaceutical compounds are easily broken down and processed in the human or animal body, whilst in other cases, a significant proportion of the compounds and their metabolites are eliminated through urine or faeces. Through these routes, some pharmaceuticals and their degradation products enter the wastewater stream, and many compounds have been detected in sewage sludge produced by wastewater treatment.

Unlike pharmaceuticals, personal care products such as cosmetics, shampoos and lotions, are directly washed into wastewater during showering and bathing, and hence enter waste water treatment. Many of these compounds and their degradation products have been detected in sewage sludge (e.g. Richardson et al., 2005). Once present in sewage sludge, there are both theoretical and measured pathways by which PCPPs can pass from sewage sludge up the food chain resulting in human exposure (Latare et al., 2014).

Numerous studies report a range of different classes of pharmaceuticals in domestic sewage, including: antibiotics, antiepileptics, anticoagulants, analgesics and anti-inflammatories, lipid regulators, steroidal compounds, cosmetics, psycho-stimulants (Luo *et al*., 2014). Several studies have showed that the fate and transport of these compounds varies during wastewater treatment, with some compounds completely degraded and some only partly degraded (WHO, 2012).

The half-lives of various PPCPs in sewage sludge applied to land have been investigated by a number of authors. Walters et al. (2010) collated data from a number of previous studies and compared these with their own empirical evidence from pot incubation studies. The results indicated that the ‘environmental half-lives’ were greater than those from controlled laboratory experiments, as well as values calculated in chemical fate models. This was thought to be due to a variety of factors:

1. Binding of the compounds within the sewage sludge matrix, reducing their bioavailability
2. The presence of complex mixtures that may inhibit microbial activity and limit degradation
3. The water content of the matrix
4. The quantity of readily available nutrients
5. Ambient temperatures
6. Microbial adaptation / acclimatisation to the compounds of interest
7. The initial concentration of the compounds of interest

To compensate for this potential underestimation in fluxes of PPCPs from the multi-media fugacity modelling used in this study (Section 2.2.2), the exposure assessment of the risk modelling has adopted 'reasonable worst case' assumptions that will tend towards over-estimating exposure (see Section 2.2).

Other studies have investigated the uptake of PPCPs from sewage sludge into crops/food chain. For example, Wu et al. (2010) reported findings from a greenhouse experiment in which the uptake of three pharmaceuticals (carbamazepine, diphenhydramine, and fluoxetine) and two personal care products (triclosan and triclocarban) by soybean grown in sewage sludge-amended soil was measured. Carbamazepine, triclosan, and triclocarban were found in both root tissues and above ground plant parts, whereas uptake and translocation for diphenhydramine and fluoxetine was limited. Holling et al. (2012) reported findings from greenhouse studies with Chinese cabbage where sewage sludge was applied at normal agronomic rates. Concentrations of carbamazepine, sulfamethoxazole, salbutamol, triclosan and trimethoprim in soils and crop (shoots and roots) were determined. Sulfamethoxazole was only detected in the roots, whilst the other compounds were found in both roots and shoots. Shargil et al. (2015) found estrone and testosterone in lettuce plants grown in soils amended with sewage sludge at rates equivalent to 500 kg total N ha^{-1}. However, the experimental design suffered from low statistical power and it was not possible to make strong inferences from these data with respect to sewage sludge being a source of estrone and testosterone in the food chain.

Overall, there is an increasing body of evidence showing that certain PCPP compounds introduced to soils via sewage sludge can be taken up by a range of crop plants, and into those parts of the plants that are normally consumed by humans (and animals). Ecotoxicologic impacts are also evident, and while not a direct health impact on humans can be seen as an indication of potential impacts on living organisms, including humans. For example, Carter et al. (2016) showed that earthworms could accumulate a number of pharmaceutical compounds (fluoxetine, carbamazepine, diclofenac and orlistat), while Konradi & Vogel (2013) studied cirpofloxacin, sulfamtheoxazole, ofloxcin and clarithromycin; recommending that the presence of these and other antibiotics in sewage sludge is monitored to mitigate

risks of change to soil microbial populations. Toxicity and fate data are still absent (or insufficient) for many emerging substances of concern (Higgins et al., 2010), but risk assessments have already been undertaken for a number of PCPPs.

Previous risk assessments

The Norwegian Food Safety Authority conducted a risk assessment for a number of chemical hazards in sewage sludge, relevant to application of sludge to agricultural land (Eriksen et al., 2009). A total of 14 pharmaceuticals were included in the assessment based on previous investigations undertaken by the Norwegian Pollution Control Authority. The risk assessment used the classical source-pathway-receptor approach, using 12 different exposure routes in order to evaluate the risks to "soil living organisms, the aquatic environment, grazing animals, animals eating feed based on plants from sludge treated soil, children eating soil, and humans consuming drinking water, crop plants and/or meat affected by the use of sludge as soil conditioner" (Eriksen et al., 2009).

The overall conclusion of the risk assessment was that exposure to the hazards considered was likely to be at levels well below predicted no-effect concentration (PNEC) values in most cases and should not constitute a significant risk to humans and biota. The risks of antibiotic resistance were also evaluated, and it was deemed unlikely for most antibiotics analysed, with the possible exception of ciprofloxacin, due of its persistence and mobility in soils. Those compounds recommended for further study as a result of this project are listed in Table 3-8.

A qualitative assessment by Smith (2009) identified over a dozen PPCPs or classes of compounds present in sewage sludge (Table 3-8). The main conclusion was that the overall risk to crop yields, animal health, groundwater, surface water, and air quality was low when applying sewage sludge according to European and British regulations. Smith (2009) also identified several "possible risks" to human health and soil fertility from land application of sewage sludge, where "there is some reported evidence that current operational practice may result in a potential impact on the environment on the basis that one or [both] of the following conditions apply: there is uncertainty about the environmental implications of particular sludge components, [and/or] effects may occur under certain extreme 'worst-case' conditions, given the current regulations and codes of practice." In a more recent assessment (Clarke & Smith, 2011), different groups of PPCPs were ranked to determine research priorities on the effects of chemical hazards after land application of sewage sludge. The following criteria were used to develop a priority index ranging from 0 to 11 points, with a higher index representing a higher priority for research and monitoring.

The prioritised list of chemicals is presented in Table 3-8. Triclosan and triclocarban were given the highest score (7/11), followed by benzothiazoles, antibiotics and synthetic musks. The lowest priority index was given to quaternary ammonium compounds (QACs), steroids, phthalate acid esters (PEAs), and polydimethylsiloxane (PDMS).

Table 3-8 Chemicals present in sewage sludge and considered in various risk assessment studies

Imperial College Reviews		Danish Risk Assessment	Norwegian Risk Assessment	WEAO Reports	EPA Research Report	CIP2 Sludge Investigations
Smith 2009[a]	Clarke & Smith 2011[a]	Jensen et al 2012	Eriksen et al 2009	WEAO 2010, 2001	Healy et al 2017	Gibbs & Jones 2017
>1000: linear alkylbenzene sulphonates (LASs) >100 <1000: Nonylphenols, Nonylphenol esters >1 <100: Triclosan, Triclocarbans, Musks	>1000: Steroids: Cholesterol, Coprostanol, Epicoprostanol, Quaternary ammonium compounds (QACs) >100 <1000: Polychlorinated alkanes, Polydimethylsiloxanes, Steroids: Campesterol, Stigmasterol >1 <100: Phthalate acid esters, Tricocarbans, Synthetic musks, Triclosan, Antibiotics <1: Steroids: E1, E2, E3, EE2	Musks, Pharaceuticals	OP/OPEs NP/NPEs LASs Pharmaceuticals: Atorvastatin, Carisoprodol, Chloprothixene, Dipyridamole, Fexofenadine, Gabapentin, Levetiracetam, Losartan, Mesalazine, MTP, Ranitidine, Sotalol, TC, Chlorophenols[b], Chlorobenzenes[b], TCS[b], Musks (galaxolide, tonalide)[b], BHT[b]	VOCs, LASs, Estrogenic hormones, PCDD/Fs, Pharmaceuticals, PBDEs, Musks, Triclosan, Triclocarbans	Triclosan, Triclocarbans	Diclofenac, Ibuprofen, Atorvastatin, Ortho-hyroxyatortvastatin, Para-hydroxyatorvastatin, Propranolol, Atenolol, Erythromycin, Norerythromycin, Azithromycin, Clarithromycin, Ciprofloxacin, Metformin, Ranitidine, Carbamazepine, Epoxycarbamazepine, Sertraline, Norsertraline, Fluoxetine, Tamoxifen, Trixylenyl phosphate

[a]Values correspond to average concentrations in sewage sludge (mg kg^{-1} DW)

[b]These compounds were not formally risk assessed due to data limitations

3.4.2. Risk Assessment

Chemicals listed in Table 3-8 were included in the risk assessment if; (i) their primary application was for pharmaceutical or personal care products, (ii) if reliable distribution coefficients were available that satisfied the requirements of the multi-media fugacity models (Section 2.2.2). The full list of pharmaceutical and personal care products (PPCPs), their physico-chemical properties and reference doses (RfD) are listed in Table 3-9.

Table 3-9 Physico-chemical data and reference doses for pharmaceuticals and personal care products (PPCPs) entered into the risk assessment.

Chemical	Water solubility (mg l^{-1} @ 25°C)	Vapour pressure (mm Hg @ 25°C)	Henry's constant (atm-m^3 mol^{-1})	Log K_{OW}	RfD (mg kg^{-1} d^{-1}) / Uncertainty Factor
Triclocarban	$2.37x10^{-3}$ (USEPA 2012)	$3.60x10^{-9}$ (USEPA 2012)	$4.50x10^{-11}$ (NCCT)	4.90 (USEPA 2012)	$2.50x10^{-3}$ / 10000 (HPVIS)
Triclosan	$1.00x10^{1}$[(a)] (Yalkowsky et al 2010)	$4.60x10^{-6}$(a) (USEPA 2012)	$2.10x10^{-8}$ (NCCT)	4.76 (NITE 2012)	$3.00x10^{-1}$ / NR (ACToR)
Carbamazepine	$1.80x10^{1}$ (USEPA 2004)	$1.84x10^{-7}$ (USEPA 2004)	$1.10x10^{-10}$ (NCCT)	2.45 (Dal Pozzo et al 1989)	$3.00x10^{-2}$ / 10000[(c)] (ToxRefDB)
Cyclomethicone 5 (Decamethylcyclopentasiloxane)	$1.70x10^{-2}$ (Kochetkov et al 2001)	$3.00x10^{-1}$ (ECHA 2015b)	$3.30x10^{1}$ (NCCT)	8.06 (Xu et al 2014)	$1.00x10^{-1}$ / 10000 (EPA Chem Dash)
Cyclomethicone 6 (Dodecamethylcyclohexasiloxane)	$5.10x10^{-3}$[(b)] (Varaprath et al 1996)	$1.69x10^{-2}$ (Lei et al 2010)	$2.50x10^{1}$ (NCCT)	8.87 (Xu et al 2014)	$1.50x10^{-1}$ / 10000 (EPA Chem Dash)
Caffeine	$2.16x10^{4}$ (Yalkowsky et al 2010)	$9.00x10^{-7}$	$1.10x10^{-11}$ (NCCT)	-0.07 (Hansch et al 1995)	$8.00x10^{-4}$ / 10000 (EPA Chem Dash)

Chemical	Water solubility (mg l^{-1} @ 25°C)	Vapour pressure (mm Hg @ 25°C)	Henry's constant (atm-m^3 mol^{-1})	Log Kow	RfD (mg kg^{-1} d^{-1}) / Uncertainty Factor
		(Emel'yanenko & Verevkin 2008)			
Diclofenac	$2.37x10^{0}$ (Fini et al 1986)	$6.14x10^{-8}$ (USEPA 2012)	$1.55x10^{-10}$ (USEPA OPERA)	4.51 (Avdeef 1987)	$4.00x10^{0}$ / 10000[(d)] (Derived from Dietrich et al 2010)
Ibuprofen	$2.10x10^{1}$ (Yalkowsky & Dannenfelser 1992)	$4.74x10^{-5}$ (Daubert & Danner 1989)	$1.50x10^{-7}$ (NCCT)	3.97 (Avdeef 1993)	$2.00x10^{-4}$ / 10000[(e)] (Derived from Nallani et al 2011)
Atorvastatin	$1.12x10^{-3}$ (USEPA 2012)	$6.56x10^{-10}$ (USEPA OPERA)	$2.40x10^{-23}$ (NCCT)	6.36 (USEPA 2012)	$1.00x10^{-3}$ / 10000[(f)] (Derived from Richards & Cole 2006)
Atenolol	$1.33x10^{4}$ (McFarland et al 2001)	$1.11x10^{-9}$ (USEPA OPERA)	$4.35x10^{-10}$ (USEPA OPERA)	0.16 (Hansch et al 1995)	$1.00x10^{-3}$ / 10000[(g)] (Derived from Zhang & Gong 2013)
Erythromycin	$2.00x10^{3}$[(h)] (O'Neil 2006)	$2.12x10^{-25}$ (USEPA 2009a)	$1.28x10^{-11}$ (USEPA OPERA)	3.06 (McFarland et al 1997)	$1.50x10^{-3}$ / 10000 (EPA Chem Dash)
Azithromycin	$2.37x10^{0}$ (USEPA 2012)	$2.65x10^{-24}$ (USEPA 2012)	$1.33x10^{-11}$ (USEPA OPERA)	4.02 (McFarland et al 1997)	$1.50x10^{-3}$ / 10000[(i)] (Derived from EPA Chem Dash)
Clarithromycin	$1.69x10^{0}$ (USEPA 2012)	$2.32x10^{-25}$ (USEPA 2012)	$1.01x10^{-10}$ (USEPA OPERA)	3.16 (McFarland et al 1997)	$1.00x10^{-1}$ / 10000[(j)] (Derived from Isidori et al 2005)
Ciprofloxacin	$3.00x10^{4}$ (Nowara et al 1997)	$2.85x10^{-13}$ (USEPA 2012)	$9.46x10^{-12}$	0.28	$1.33x10^{-1}$ / 100

Chemical	Water solubility (mg l^{-1} @ 25°C)	Vapour pressure (mm Hg @ 25°C)	Henry's constant (atm-m^3 mol^{-1})	Log Kow	RfD (mg kg^{-1} d^{-1}) / Uncertainty Factor
			(USEPA OPERA)	(Takács-Novák et al 1992)	(Derived from USFDA max therapeutic dose)
Metformin	$1.06x10^{6}$ (USEPA 2012)	$7.58x10^{-5}$ (USEPA 2012)	$7.60x10^{-16}$ (NCCT)	-2.64 (USEPA 2012)	$5.00x10^{-1}$ / 100 (Derived from USFDA max therapeutic dose)
Ranitidine	$2.47x10^{1}$ (Gerhartz1985)	$2.99x10^{-9}$ (USEPA OPERA)	$7.29x10^{-9}$ (USEPA OPERA)	0.22 (USEPA OPERA)	$1.00x10^{—2}$ / 10000 (Derived from LD50 in rats Acute Tox)
Sertraline	$0.35x10^{-1}$ (Drugbank)	$3.36x10^{-6}$ (USEPA OPERA)	$1.15x10^{-6}$ (USEPA OPERA)	4.55 (USEPA OPERA)	$1.00x10^{-3(k)}$ / 10000 (Derived from Richards & Cole 2006)
Norsertraline	$1.04x10^{-6}$ (USEPA OPERA)	$1.07x10^{-9}$ (USEPA OPERA)	$6.43x10^{-7}$ (USEPA OPERA)	4.94 (USEPA OPERA)	$1.00x10^{-3(l)}$ / 10000
Tamoxifen	$1.67x10^{1}$ (USEPA 2004)	$3.46x10^{-8}$ (USEPA 2004)	$2.21x10^{-8}$ (USEPA OPERA)	6.30 (USEPA 2004)	$4.00x10^{-5(m)}$ / 10000 (Derived from Cevasco et al 2008)
Dipyridamole	$9.22x10^{2}$ (Human Metabolome Database)	$9.30x10^{-10}$ (USEPA OPERA)	$1.21x10^{-11}$ (USEPA OPERA)	2.06 (USEPA OPERA)	$1.68x10^{-2}$ / 10000 (Derived from LD50 in rats Acute Tox)
Fexofenadine	$2.40x10^{-2}$ (USEPA 2004)	$2.56x10^{-9}$ (USEPA OPERA)	$2.51x10^{-8}$ (USEPA OPERA)	2.81 (USEPA 2004)	No Tox Data Available

Chemical	Water solubility (mg l^{-1} @ 25°C)	Vapour pressure (mm Hg @ 25°C)	Henry's constant (atm-m^3 mol^{-1})	Log K_{OW}	RfD (mg kg^{-1} d^{-1}) / Uncertainty Factor
Gabapentin	$4.49x10^{3}$ (USEPA 2012)	$2.94x10^{-10}$ (USEPA 2012)	$4.87x10^{-8}$ (USEPA OPERA)	-1.10 (Sangster 2005)	$2.00x10^{-1}$ / 100 (Derived from USFDA maximum theraputic dose)
Levetiracetam	$1.04x10^{5}$ (Thomson & Montvale 2007)	$3.50x10^{-6}$ (USEPA 2004)	$1.77x10^{-9}$ (USEPA OPERA)	-0.49 (USEPA 2004)	$6.00x10^{-1}$ / 100 (Derived from USFDA maximum theraputic dose)
Sotalol	$5.51x10^{3}$ (Drugbank)	$2.19x10^{-10}$ (USEPA OPERA)	$1.25x10^{-9}$ (USEPA OPERA)	0.55 (USEPA OPERA)	$5.00x10^{-2}$ / 100 (Derived from USFDA maximum theraputic dose)
Benzothiazole	$4.30x10^{0}$ (Human Metabolome Database)	$1.60x10^{-2}$ (USEPA OPERA)	$9.53x10^{-6}$ (USEPA OPERA)	2.05 (USEPA OPERA)	$5.10x10^{-4}$ / 10000 (USEPA Chem Dash)

[a]at 20°C; [b]at 23°C; [c]Low confidence - derived from USGS screening level; [d]Very low confidence - derived from lowest point of departure from ecotoxicologic study with water fleas; [e]Very low confidence – derived from lowest point of departure from ecotoxicologic study with catfish; [f]Very low confidence – derived from lowest point of departure from ecotoxicologic study with frog larvae; [g]Very low confidence – derived from lowest point of departure from ecotoxicologic study with zebradanio fish; [h]28°C; [i]Very low confidence – assumed the same RfD as Arythromycin; [j]Very low confidence – derived from lowest point of departure from exotoxicologic study with zebradanio fish; [k]Very low confidence – derived from lowest point of departure from ecotoxicologic study with frog larvae; [l]Very low confidence – assumed to have the same RfD as Sertraline; [m] Very low confidence – derived from lowest point of departure from ecotoxicologic study with frog larvae;

Initial concentrations in sewage sludge were set based on the 'realistic worst case' paradigm (WRAP 2016c; Hough et al. 2012). A series of recent reviews documenting measured concentrations in sewage sludge were used and starting concentrations were derived from 90%ile values reported in these data sets (Table 3-10).

Table 3-10 Initial concentrations of pharmaceuticals and personal care products (PPCPs) in sewage sludge prior to spreading

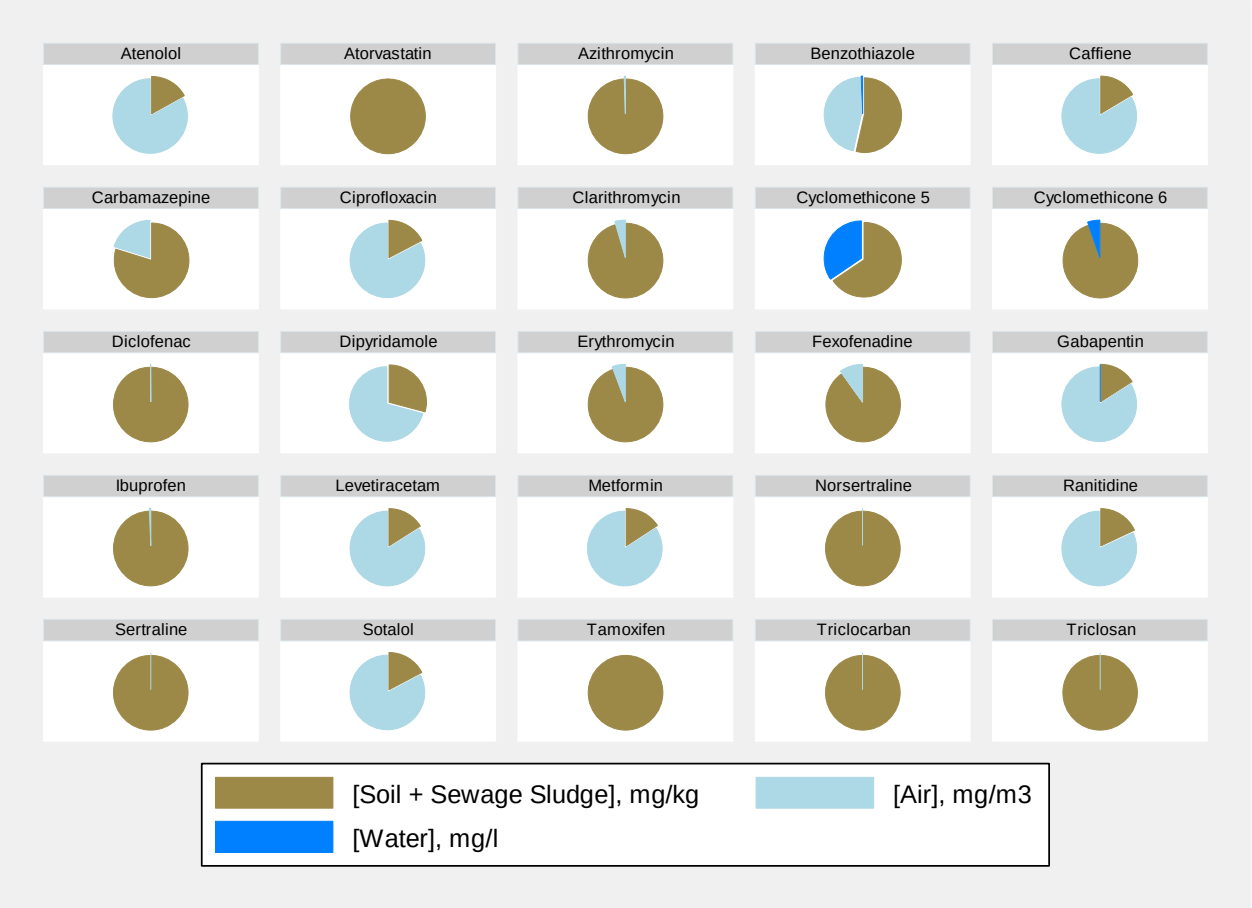
Chemical (IUPAC)	Concentration in sewage sludge (mg kg^{-1})	Reference(s)
Triclocarban (1-(4-Chlorophenyl)-3-(3,4-dichlorophenyl)urea)	$2.21x10^{2}$	USEPA 2009b
Triclosan (5-Chloro-2-(2,4-dichlororophenoxy)phenol)	$4.74x10^{0}$	USEPA 2009b Stasinakis et al 2008
Carbamazepine (5H-Dibenzo[b,f]azepine-5-carboxamide)	$2.09x10^{-2}$	USEPA 2009b UKWIR 2012 Gibbs & Jones 2017 JRC 2012
Cyclomethicone 5 (2,2,4,4,6,6,8,8,10,10-decamethyl-1,3,5,7,9,2,4,6,8,10-pentaoxapentasilecane)	$2.58x10^{3}$	Harrison et al 2006
Cyclomethicone 6 (2,2,4,4,6,6,8,8,10,10,12,12-dodecamethyl-1,3,5,7,9,11-hexaoxa-2,4,6,8,10,12-hexasilacyclododecane)	$2.58x10^{3}$	Harrison et al 2006
Caffiene (1,3,7-Trimethylxanthine)	$4.96x10^{-2}$	USEPA 2009b JRC 2012
Diclofenac (2-(2,6-dichloranilino) phenylacetic acid)	$6.00x10^{-5}$	UKWIR 2012 Gibbs & Jones 2017 JRC 2012
Ibuprofen (2-(4-Isobutylphenyl)propanoic acid)	$2.70x10^{-4}$	Carballa 2004 Gomez et al 2007 USEPA 2009 UKWIR 2012 Gibbs & Jones 2017

Chemical (IUPAC)	Concentration in sewage sludge (mg kg^{-1})	Reference(s)
		JRC 2012
Atorvastatin ((3R,5R)-7-[2-(4-fluorophenyl)-3-phenyl-4-(phenylcarbamoyl)-5-propan-2-ylpyrrol-1-yl]-3,5-dihydroxyheptanoic acid)	$8.73x10^{-2}$	Gibbs & Jones 2017
Atenolol (2-[4-[2-hydroxy-3-(propan-2-ylamino)propoxy]phenyl]acetamide)	$4.76x10^{-1}$	UKWIR 2012 Gibbs & Jones 2017
Erythromycin ((3R,4S,5S,6R,7R,9R,11R,12R,13S,14R)-6-[(2S,3R,4S,6R)-4-(dimethylamino)-3-hydroxy-6-methyloxan-2-yl]oxy-14-ethyl-7,12,13-trihydroxy-4-[(2R,4R,5S,6S)-5-hydroxy-4-methoxy-4,6-dimethyloxan-2-yl]oxy-3,5,7,9,11,13-hexamethyl-oxacyclotetradecane-2,10-dione)	$7.82x10^{-2}$	USEPA 2009b UKWIR 2012 Gibbs & Jones 2017 Jones et al 2014
Azithromycin ((2R,3S,4R,5R,8R,10R,11R,12S,13S,14R)-11-[(2S,3R,4S,6R)-4-(dimethylamino)-3-hydroxy-6-methyloxan-2-yl]oxy-2-ethyl-3,4,10-trihydroxy-13-[(2R,4R,5S,6S)-5-hydroxy-4-methoxy-4,6-dimethyloxan-2-yl]oxy-3,5,6,8,10,12,14-heptamethyl-1-oxa-6-azacyclopentadecan-15-one)	$1.06x10^{-1}$	USEPA 2009b Gibbs & Jones 2017
Clarithromycin ((3R,4S,5S,6R,7R,9R,11R,12R,13S,14R)-6-[(2S,3R,4S,6R)-4-(dimethylamino)-3-hydroxy-6-methyloxan-2-yl]oxy-14-ethyl-12,13-dihydroxy-4-[(2R,4R,5S,6S)-5-hydroxy-4-methoxy-4,6-dimethyloxan-2-yl]oxy-7-methoxy-3,5,7,9,11,13-hexamethyl-oxacyclotetradecane-2,10-dione)	$1.38x10^{-1}$	USEPA 2009b Gibbs & Jones 2017 JRC 2012
Ciprofloxacin (1-cyclopropyl-6-fluoro-4-oxo-7-piperazin-1-ylquinoline-3-carboxylic acid)	$6.49x10^{-1}$	USEPA 2009b UKWIR 2012 Gibbs & Jones 2017 Vieno et al 2006 Golet et al 2003

Chemical (IUPAC)	Concentration in sewage sludge (mg kg^{-1})	Reference(s)
Metformin (1,1-Dimethylbiguanide)	$4.00x10^{1}$	Eggen et al 2011 USEPA 2009b Gibbs & Jones 2017
Ranitidine (6.N (2-(((5-((Dimethylamino)methyl)-2-furanyl)methyl)thio)ethyl)-N'-methyl-2-nitro-1,1-ethenediamine)	$9.00x10^{-2}$	USEPA 2009b Gibbs & Jones 2017
Sertraline ((1S,4S)-4-(3,4-dichlorophenyl)-N-methyl-1,2,3,4-tetrahydronaphthalen-1-amine)	$1.20x10^{0}$	Gibbs & Jones 2017
Norsertraline ((1s,4s)-4-(3,4-dichlorophenyl)-1,2,3,4-tetrahydronaphthalen-1-amine)	$7.60x10^{-1}$	Gibbs & Jones 2017
Tamoxifen (2-[4-[(Z)-1,2-diphenylbut-1-enyl]phenoxy]-N,N-dimethylethanamine)	$5.36x10^{-2}$	Gibbs & Jones 2017
Dipyridamole (2-[[2-[bis(2-hydroxyethyl)amino]-4,8-di(piperidin-1-yl)pyrimido[5,4-d]pyrimidin-6-yl]-(2-hydroxyethyl)amino]ethanol)	$2.48x10^{-1}$	Okuda et al 2009
Fexofenadine (2-[4-[1-hydroxy-4-[4-[hydroxy(diphenyl)methyl]piperidin-1-yl]butyl]phenyl]-2-methylpropanoic acid)	$1.70x10^{-4}$	Golovko et al 2014
Gabapentin (1-(Aminomethyl)cyclohexaneacetic acid)	$1.00x10^{-3}$	Writer et al 2013
Levetiracetam ((2S)-2-(2-oxopyrrolidin-1-yl)butanamide)	$1.25x10^{-2}$	Gurke et al 2015
Sotalol (N-[4-[1-hydroxy-2-(propan-2-ylamino)ethyl]phenyl]methanesulfonamide;hydrochloride)	$5.09x10^{-4}$	Radjenović et al 2009
Benzothiazole (1,3-benzothiazole)	$6.44x10^{1}$	Harrison et al 2006

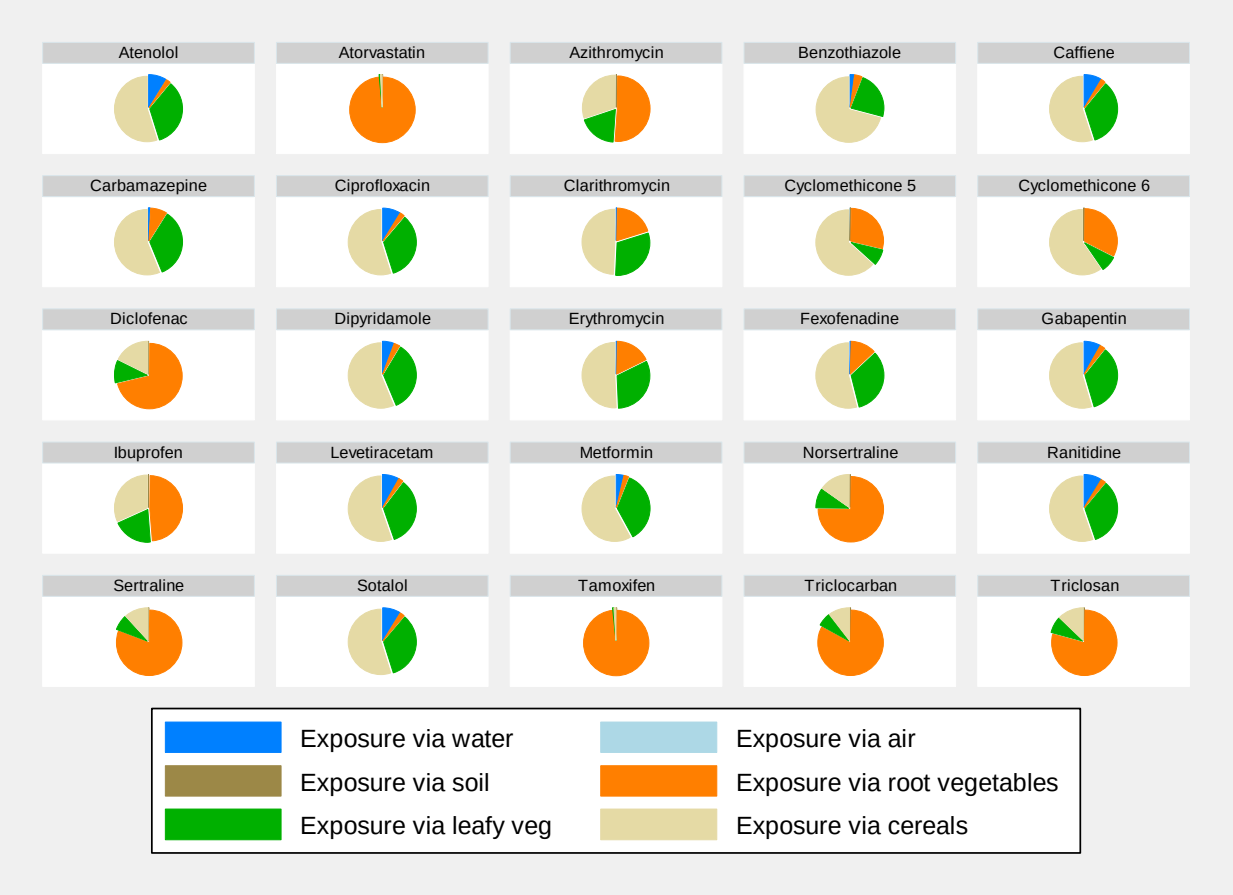
Combining partition coefficients (Table 3-9) with initial concentrations in sewage sludge (Table 3-10) within Equations 2-6 - 2-9, it was possible to estimate the concentrations of each PPCP that would remain in soil vs. migrate to soil pore water or soil pore air (Figure 3-12). Those contaminants with the greatest propensity to partition into the soil pore water are subsequently more likely to be taken up by food crops or pasture, and therefore enter the human food chain.

Figure 3-12 Partitioning [ppm] of pharmaceutical and personal care products (PPCPs) between soil amended with sewage sludge, soil pore water, and soil air spaces given initial starting concentrations in sewage sludge (Table 3-10)



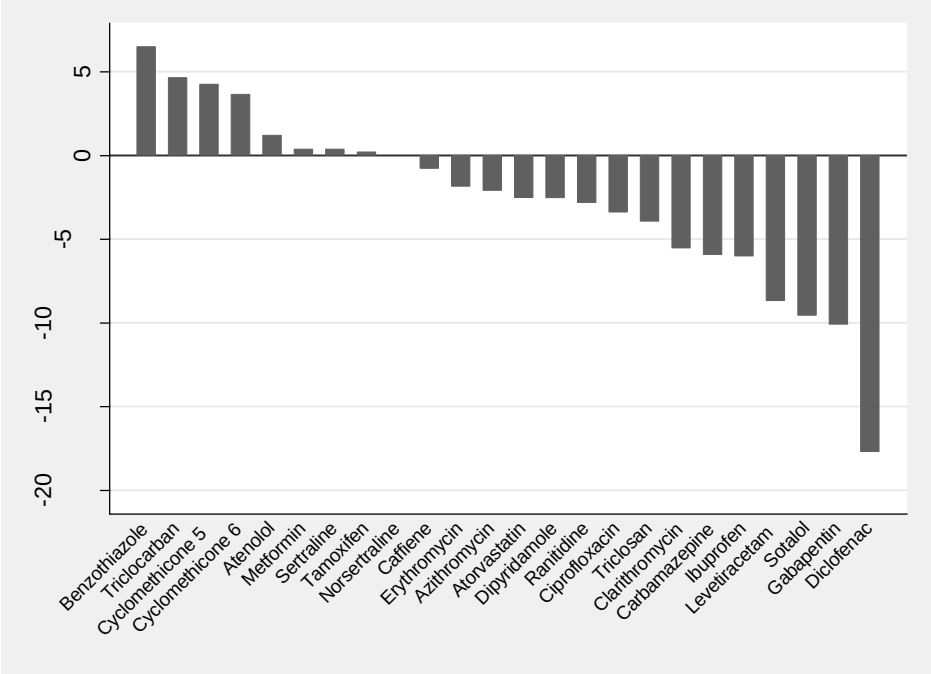
Concentrations of each PCPP in root crops, leafy vegetables, cereal crops, groundwater (private drinking supplies) and volatilisation to air were estimated using Equations 2-10, 2-11 & 2-12. These were related to human exposure by assuming that a proportion of produce consumed was from the locations where sewage sludge was applied, a proportion of the air breathed in was derived from air into which contaminants had volatilised into, and a proportion of the water consumed was derived from a supply into which contaminants had leached. The assumptions made were the same as those made in the risk assessment of organic contaminants are detailed in Table 3-7. Estimates of exposure for each individual PPCP were then calculated (Figure 3-13).

Figure 3-13 Relative exposure profiles for each pharmaceutical and personal care product (PPCP) via six different exposure medium (water, air, soil, root vegetables (including potatoes), leafy vegetables (including salad vegetables), and cereals.



Estimates of exposure (Figure 3-13) were compared to the reference doses (RfDs, Table 3-9) in order to derive the Hazard Quotient, HQ as defined in Equation 2-15 (Figure 3-14).

Figure 3-14 Estimates of hazard quotient, log(HQ), for each pharmaceutical and personal care product (PPCP) based on the ratios of the exposures described in Figure 3-13 and the reference doses (RfD) for each organic contaminant as detailed in Table 3-9. A log(HQ) > 0 indicates that further investigation may be required.



Conclusions

Even with ‘realistic worst case’ assumptions, the majority of the phamaceuticals and personal care products investigated posed no appreciable risk to local inhabitants.

The following PPCPs returned a hazard quotient that was significantly greater than one; benzothiazole, triclocarban, cyclomethicone 5 & 6 and atenolol (Figure 3-14); suggesting that for the worst case exposure scenarios described above, individuals would experience deleterious health effects due to lifetime exposure to these compounds as a result of sewage sludge application to land. It is interesting to note that for all these compounds, the exposure is dominated by food consumption with consumption of cereals driving the risk from benzothiazole, atenolol and the cyclomethicones (although in all these cases, consumption of leafy vegetables is also important), while root vegetable consumption drives the risk from triclocarban (Figure 3-13). Therefore these exposures (if found to be real) could be mitigated by restricting the use of sewage sludge to crops for animal feed, e.g. grass and other forage crops. There are also a number of opportunities within the treatment processes to mitigate against these chemicals, with partial reductions readily achieved using anaerobic digestion and thermal hydrolysis (see Table 4-1)

It should be noted that the RfD for atenolol has especially large uncertainties as it is derived from a point of departure from ecotoxicologic data where fish have been

exposed to atenolol in their water environment. This exposure scenario clearly does not represent very well the human exposure pathways of interest here, as well as the uncertainties around the appropriateness of using fish as a proxy for human beings. The RfDs for benzothiazole, triclocarban and the cyclomethicones were all values that are currently used in regulatory situations.

The analysis also suggests that metformin, sertraline and tamoxifen might also pose a risk, however given the 'realistic worst case' assumptions it is likely that this risk is minimal. The main exposure route for sertraline and tamoxifen is via consumption of root vegetables, while consumption of cereals drives the risk from metformin (Figure 3-13). Therefore, again the restriction of sewage sludge application to forage crops would minimise this risk if necessary.

These estimates of HQ are based on realistic worst case assumptions, thus the exposure scenarios will only apply to a very small proportion of the population (if at all). The majority of individuals living close to sewage sludge application activities are not going to be exposed via drinking water, and it is unlikely that they will be consuming 10% of their food intake from locally-grown produce.

The use of 'realistic worst case' scenarios is a conservative approach aimed at protecting the most vulnerable in society and hence the approach tends to drive up risk estimates. This is helpful as it gives a level of confidence where we identify compounds that despite the worst case assumptions pose little risk to inhabitants. The recommendations made here have been made on the assumption that the potential exposures highlighted are found to be real and reasonably widespread.

3.5. Microplastics and fibres

3.5.1. Background

Microplastics are a heterogeneous mixture of differently shaped materials referred to as fragments, fibres, spheroids, granules, pellets, flakes or beads. There is no internationally recognised definition, with microplastics and related fibres most-commonly defined as synthetic polymers measuring less than 5 mm in diameter (i.e. largest dimension); while definitions vary, the majority do not include a lower size limit. They are derived from a wide range of sources including; synthetic fibres from clothing (Browne et al., 2011; Astrom, 2016), polymer manufacturing and processing industries (Lechner & Ramler, 2015) and personal care products (Fendall & Sewell, 2009). A distinction can be made between primary and secondary microplastics. Primary microplastics are plastics that were originally manufactured to be that size (for specific purposes e.g. use in cosmetic products) while secondary microplastics originate from fragmentation of larger items, e.g. plastic debris (Wright et al., 2013). Microplastics can arise from direct sources, such as industrial accident spillages or release of microbeads from cosmetics through wastewaters.

Fibres are examples of secondary microplastics. The majority of our clothing and textile goods contain synthetic textiles such as polyester, polylactide, olefin,

spandex, nylon, lyocell, Lurex, Ingeo, acrylics, aramids, Tencel, acetate, rayon and polypropylene (Carr, 2017). These synthetic textile materials shed microfibres (threadlike residues which are invisible to the naked eye and are usually persistent being made of non-biodegradable materials), as they are worn, handled and exposed to the elements. Washing machines can produce secondary microplastics fibres that can enter into the environment through sewage wastewaters (Cauwenberghe et al., 2013). Given the volume of synthetic fibres manufactured and used throughout the world it is suspected that microfibre contamination of the environment is even higher than that of particulate plastics (Carr, 2017).

Microplastics and fibres have the potential to adsorb organic contaminants (Engler, 2012; Teuten et al., 2009) and [priority] metals (Brennecke et al., 2016; Rochman et al., 2014; Holmes et al., 2012; Nakashima et al., 2012; Ashton et al., 2010) from the surrounding environment. These may be released upon digestion by biota or through environmental degradation, leading to possible impacts to human health and ecosystems (Bouwmeester et al., 2015; Andrady, 2011; Cooper & Corcoran, 2010).

Over the last ten years, many studies have investigated the distribution and effects of microplastics and (to a lesser extent) fibres within the marine (e.g. Watts et al., 2016; Remy et al., 2015; Cole et al., 2013; von Moos et al., 2012) and freshwater (e.g. Horton, 2017) environments. It was estimated in 2014 that there were 250,000 tons of plastic particles afloat in the world's oceans (Eriksen et al., 2014). Microplastics have been found in Polar Regions (Lusher et al., 2014) and in a range of freshwater environments worldwide (Castañeda et al., 2014; Free et al., 2014; McCormick et al., 2014; Eriksen et al., 2013). Overall, the literature on plastic pollution in the marine environment is extensive and includes some very comprehensive reviews (e.g. Bosker et al., 2017; Duis & Coors, 2016; Eriksen et al., 2014). Despite this, very few studies have sought to determine land-based sources of microplastics and fibres. Therefore, in this assessment, we have attempted to draw inferences relevant to sewage sludge recycling from those reported in the vast aquatic literature.

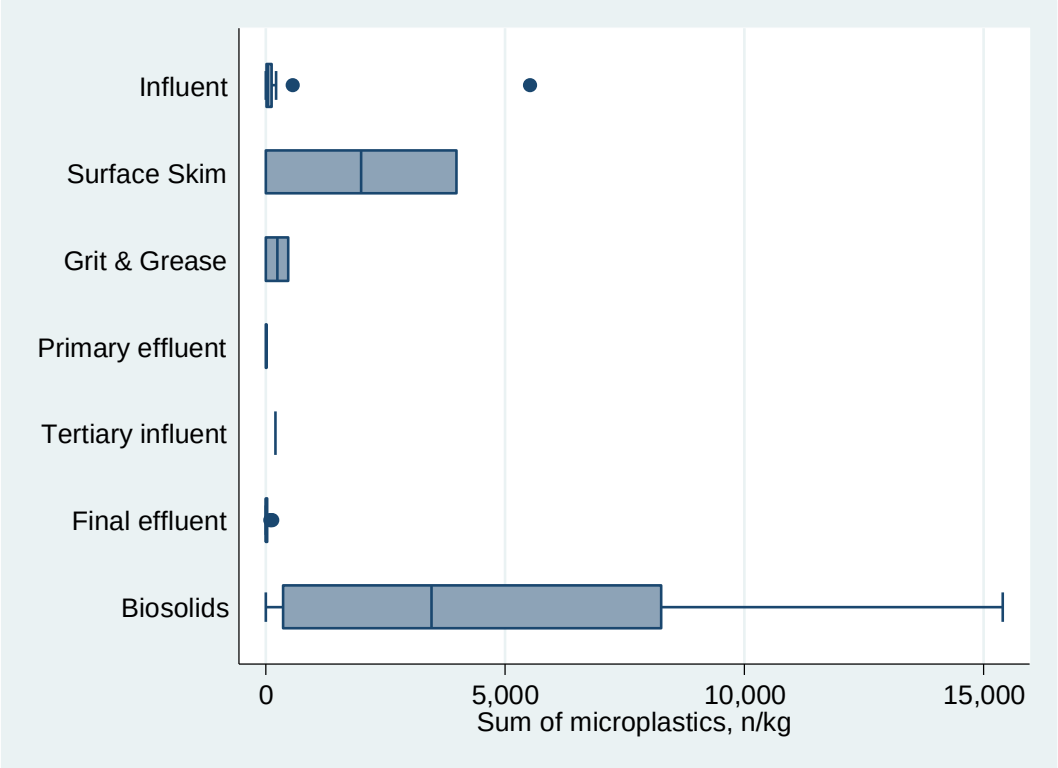
Wastewater treatment plants (WWTPs) have been identified as receptors of microplastic pollution and are effective in capturing the majority of microplastics and fibres during the sludge settlement process (Carr et al., 2016). Almost 120,000 tonnes ds of sewage sludge were produced by waste water treatment plants in the UK (Eurostat, 2016). As approximately 99% of microplastics and fibres are retained in sewage sludge (Mahon et al., 2017; Magnusson & Norén, 2014), there is a strong possibility that land applied sludge, even having undergone treatment, could be a significant source of microplastics pollution. Estimates of the contribution of various sources of microplastics to the environmental pool have been derived. However, such estimates are subject to considerable uncertainty due to the complexity of the sources, the lack of quantitative data on transport and fate of microplastics, and the high geographic variability of the relevance of different sources and introduction routes (often caused by differences in sewerage infrastructure; Duis & Coors, 2016).

A first estimate of the relative contribution of microplastics from personal care products to the plastic debris in the North Sea has been provided by Gourin et al. (2015) who assumed that 6 % of liquid skin cleansing products contained 10 % microplastics, equating to 2300 t yr-1 of microplastic particles derived from the countries in the watershed of the North Sea (UK, Norway, Denmark, Germany, Belgium, The Netherlands, France, Switzerland, and Czech Republic) via WWTPs. For the UK alone, the estimate was 680 t microplastic beads yr^{-1} (30 mg beads $capita^{-1}$ day^{-1}). As Gourin et al. (2015) assume a 90 % removal rate of microplastics during waste water treatment (likely to be closer to 99 % (Magnusson & Norén, 2014)); this suggests an annual loading in sewage sludge of 612 - 673 t microplastics from the UK (equates to roughly ~0.2 – 0.3 % of the global microplastics burden being generated by ~0.9% of the global population – remembering that this is just for microplastics from personal care products). Due to current EU and UK practice, it is highly likely that the majority of these microplastics will be spread to agricultural land.

3.5.2. Levels of microplastics in sewage sludge

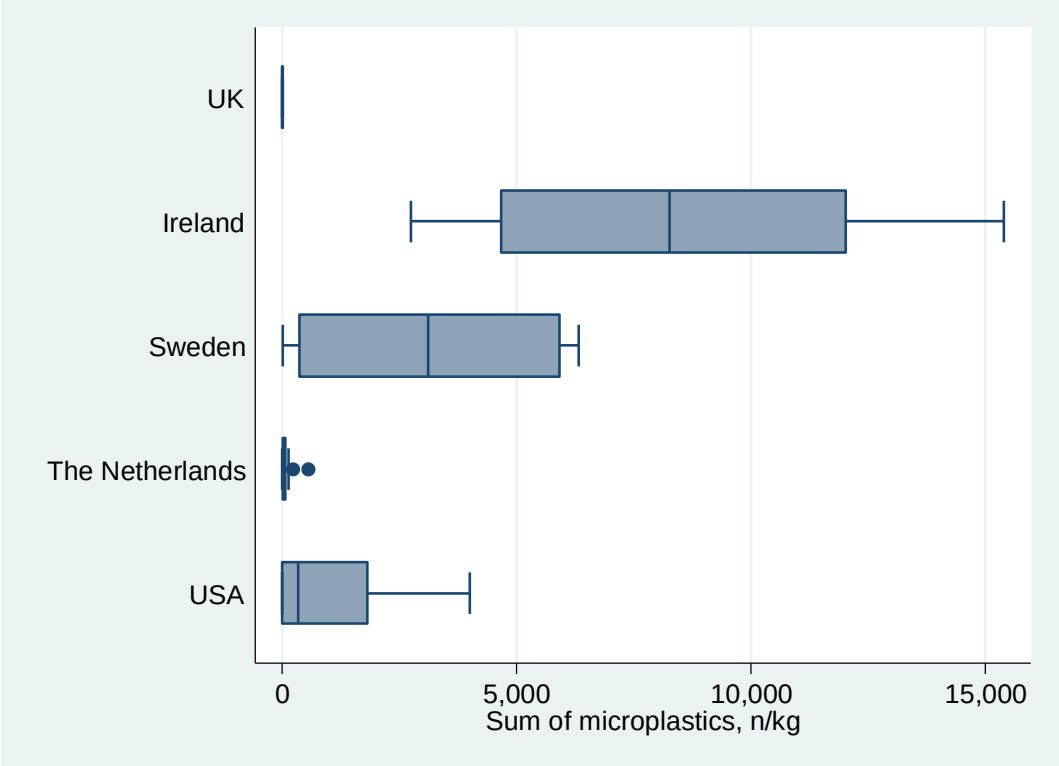
The majority of microplastics literature has focussed on detection and quantification of plastic particles in the aquatic environment, with the literature pertaining to sources of microplastics being somewhat limited. A few studies have been undertaken during the last 5 years investigating levels of microplastics at different points in the waste water treatment process, and even fewer studies from countries of relevance to Scotland; these data are summarised in Figure 3-15. One of the main limitations is that waste water composition, as well as associated treatment processes, is extremely variable and there is little way of knowing how representative the available data on microplastics are. Also, the available data do not cover the full range of treatment processes commonly used in Scotland. Therefore, while there is no doubt that microplastics are found in waste water, the quality of the available data mean that it is only possible to draw one or two firm inferences. The first one is the observation that levels of microplastics in any influent, effluent or biosolids are very variable. The second observation is that the presence of microplastics is most strongly associated with solid fractions. The majority of microplastics within waste water are retained in the biosolids (Leslie et al., 2017; Mahon et al., 2017; Carr et al., 2016), but also in grit and grease (Carr et al., 2016; Murphey et al., 2016), as well as being associated with surface scums (Carr et al., 2016). Final effluent tends to have much reduced concentrations of microplastics (Leslie et al., 2017; Carr et al., 2016; Murphey et al., 2016). The inference therefore is that the main route of microplastics from waste water to the environment is through the recycling of sewage sludge [to land]. However, it is worth bearing in mind that all data have been reported in terms of concentration (numbers of individual microplastic particles or fibres per unit volume or per unit mass) rather than mass of plastics. The volumes of liquids and solids being handled by the treatment process will influence the total flux of plastics to the environment via effluent compared to sewage sludge.

Figure 3-15 Sum of total microplastic particles/fibres (number kg^{-1}) in influents, effluents and biosolids associated with waste water treatment processes (EU and N. American data only). Data collated from Leslie et al. (2017), Mahon et al. (2017), Carr et al. (2016), Michielssen et al. (2016), Murphey et al. (2016), Magnusson & Noren (2014), and references therein.



Grouping the available data for sewage sludge by country also suggests significant within- and between-country variation (Figure 3-16). The available data suggest that the UK and The Netherlands have the lowest levels of micoplastics (in terms of numbers of particles/fibres per unit mass) associated with waste water treatment; however, there are so few studies in these two countries that these figures cannot support such a generalisation. The countries that display the widest variation, e.g. Ireland, are also those that have the largest numbers of studies published and simply reflect the sheer variation in waste water quality and treatment technologies employed within any one jurisdiction.

Figure 3-16 Sum of total microplastics (n kg^{-1}) in sewage sludge associated with waste water treatment processes grouped by country. Data collated from Leslie et al. (2017), Mahon et al. (2017), Carr et al. (2016), Michielssen et al. (2016), Murphey et al. (2016,) Magnusson & Noren (2014), and references therein.

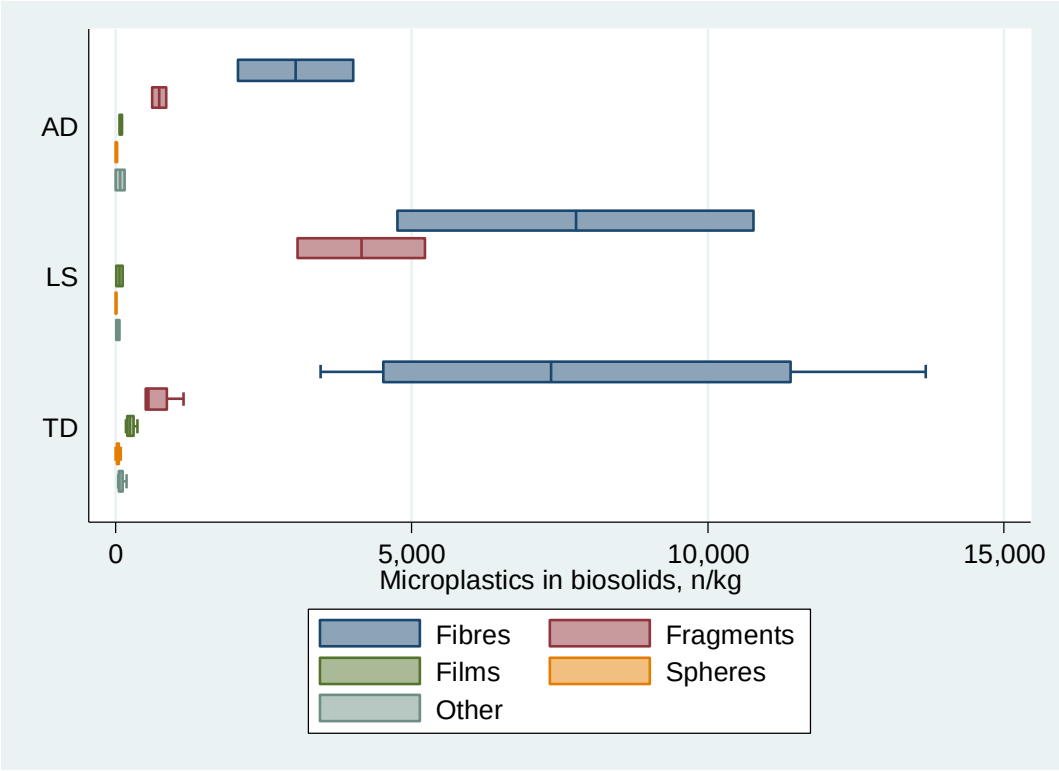


In terms of fibres, Habib et al. (1998) found elevated levels of synthetic fibres in harbour sediments close to a sewage treatment plant outfall in New York State and Talvitie et al. (2015) found that fibres (and microplastics) were present in the treated effluent from a WWTP in Finland. Similarly, Dris et al. (2015) found high levels of fibres in Parisian wastewater (260–320×10^3 particles m^{-3}) but less in treated effluent (14–50×10^3 particles m^{-3}). The partitioning of microplastics and fibres via settlement processes during wastewater treatments results in the majority becoming entrained in the sewage sludge (Talvitie et al., 2015, Mahon et al., 2017). Verschoor et al. (2014) reported that 90% of microplastics were removed during wastewater treatment. Similarly, Leslie et al. (2017) reported that the mean microplastic retention efficiency in the sludge for Dutch WWTPs was 72%, but this varied widely (standard deviation 61%). However, in there is very little published information on typical levels of fibres in sewage sludge applied to agricultural land.

One study, Mahon et al. (2017), investigated the effect of treatment processes (anaerobic digestion (AD), lime stabilisation (LS), thermal drying (TD)) on concentrations of microplastics in sewage sludge. While it may seem biased to place so much emphasis on this single study, it should be noted that this is the only study of its type currently available that has investigated the implications of water treatment

processes on levels of microplastics in sewage sludge. Mahon et al. suggested that AD might reduce concentration of microplastics, and more importantly reduce the concentrations of the smaller size fractions of microplastics that have been most strongly associated with adverse outcomes in toxicological studies (see Section 3.6.3); Figure 3-17. It is noteworthy that fibres were the most commonly measured microplastic (in terms of count of numbers of plastic particles per unit mass) in this study. As with all of the microplastic studies discussed in this section, these data have been reported as concentrations. If the same data were expressed as fluxes of plastic, the inferences drawn from the study might well be different.

Figure 3-17 Concentration of microplastics of various types in sewage sludge generated using three different treatment processes; anaerobic digestion (AD), lime stabilisation (LS), and thermal drying (TD). With respect to size fraction: Fibres > Fragments > Films > Spheres > Other. Adapted from Mahon et al. (2017).



3.5.3. Impacts/Toxicology of microplastics

Impacts of microplastics tend to be categorised either as:

- Direct physical impact (i.e. the ability of a small particle to block ducts or damage tissues, see e.g. Wright et al., 2013);
- Direct chemical impact (i.e. degradation of plastic particles resulting in the release of potentially toxic chemicals, see Fries et al., 2013);

- Indirect chemical impacts (i.e. potentially toxic chemicals become adsorbed onto microplastic particles that are then transported along with the microplastic before being released under different chemical conditions such as those found inside an animal's stomach, see Koelmans et al., 2016).

Adsorbed chemical pollutants are either derived from additives used during the polymer synthesis process, or adsorbed directly from the environment (Engler, 2012; Teuten, et al., 2009; Rios, et al., 2007). The hydrophobicity of organic xenobiotics and the large surface area of plastic polymers facilitate the adsorption of these chemicals on microplastics at concentrations orders of magnitude higher than those detected in the surrounding environment (Ogata et al., 2009). The possibility for plastic particles to adsorb chemical pollutants from the surrounding environment has also been explored under laboratory conditions. Different particles polymers, like polyvinyl chloride, polyethylene, polypropylene and polystyrene, were shown to have high sorption capacity for dichlorodiphenyltrichloroethanes (DDTs), polycyclic aromatic hydrocarbons (PAHs), hexachlorocyclohexanes and chlorinated benzenes (Lee et al., 2014; Bakir et al., 2012). Consistent with these studies, several persistent organic pollutants (POPs), polychlorinated biphenyls (PCBs), organohalogenated pesticides, nonylphenol, PAHs and dioxins have been detected in plastic pellets washed up on different beaches around the world (Heskett et al., 2012; Hirai et al., 2011; Ogata et al., 2009; Endo et al., 2005).

With respect to sewage sludge, the inference must be that microplastics present in wastewater are able to adsorb and concentrate hydrophobic chemicals also present in waste water (such as those assessed in Section 3.4). These pollutants would then likely be delivered to agricultural land when sewage sludge is applied. Similarly, there is therefore potential for microplastics present in agricultural soils to adsorb and concentrate hydrophobic chemicals present in this environment such as various agri-chemicals.

Microplastic uptake has been reported in a wide range of animals including mammals and invertebrates (Cole et al., 2015; Cole et al., 2013; Lee et al., 2013; Von Moos et al., 2012; Wegner et al., 2012; Browne et al., 2008). Specifically, microplastic uptake has been investigated in plankton (Cole et al., 2015; Cole et al., 2013; Lee et al., 2013), the blue mussel (Von Moos et al., 2012; Wegner et al., 2012; Browne et al., 2008) and the lugworm (Besseling et al., 2013). In laboratory conditions, microplastics have been shown to be ingested (among others) by amphipods, barnacles, and lugworms (Thompson et al., 2004). However, the fate of microplastics after ingestion is still largely unknown. For instance, it remains an outstanding question whether ingested microplastics can translocate into other organs via the circulatory system. However, the accumulation of ingested microplastics in various organs has been reported. For example, in mussels plastic particles (3.0 – 9.6 μm) were shown to have accumulated in digestive tissues and translocated to the haemolymph (Browne et al., 2008). In the same organisms, the uptake of microplastics caused notable histological changes in digestive cells with

string inflammatory responses, formation of granulocytomas and lysosomal destabilisation which increased with exposure time (Von Moos et al., 2012). It has been shown that microplastics exhibit reasonably strong size-dependent toxicity (e.g. Jeong et al., 2016). Therefore, it is likely that any impacts associated with microplastics will be heightened as particle size decreases. Thus potential toxicity of microplastic particles could be treated in a similar way to airborne dusts (with current air quality standards based around particle size).

With respect to the transfer of microplastics up the [human] food-chain, there is some evidence related to the food industry. However, the focus is on consumption of fish and seafood (although honey and beer have also been looked at) and the assumption has been made (in the absence of other data) that these are the primary sources of microplastics in the [human] food-chain. This seems naïve, given what we know about sewage sludge being a significant source of microplastics.

There is a lack of information on the fate of microplastics in the [human] GI tract. The available data on toxicokinetics only include absorption and distribution, whereas no information is available on metabolism and excretion. This is primarily because microparticles have been investigated for their drug delivery potential, rather than from an exposure or toxicological perspective. Only microplastics smaller than 150 μm may translocate across the gut epithelium causing systemic exposure (reported in Hussain et al., 2001). The absorption of these microplastics is expected to be limited (≤ 0.3%; Carr et al., 2012). Only the smallest fraction (size < 1.5 μm) may penetrate deeply into organs (Yoo et al., 2011). There is a lack of knowledge about the local effects of microplastics in the GI tract, including microbiota. Toxicological data on the effects of microplastics as such are essentially lacking for human risk assessment (EFSA, 2016).

No studies have been identified that address the potential human health effects of microplastics ingested by humans through the food chain. In an investigation of the effects of coarse bran on small bowel transit time in adults, microplastic beads (barium-labelled polyethylene (PE)) were used as a control. A single dose of 15 g PE in rice pudding hastened the arrival of the label at the colon to the same degree as course bran (McIntyre et al., 1997). Although this is a pronounced effect, the dose is highly unrealistic in terms of human exposure through the food chain (EFSA, 2016).

While there are potentially serious impacts associated with exposure to microplastics, there is currently no knowledge regarding exposure to microplastics incorporated into sewage sludge and applied to agricultural land. The focus of research has been almost exclusively on the marine environment, but there is no knowledge regarding the proportion of the marine microplastic burden that can be attributed to sewage sludge. Similarly there is no information regarding the potential uptake of microplastics by food crops or grazing animals, hence limited inferences can be made regarding potential uptake of plastics from land-applied sewage sludge and into the human foodchain.

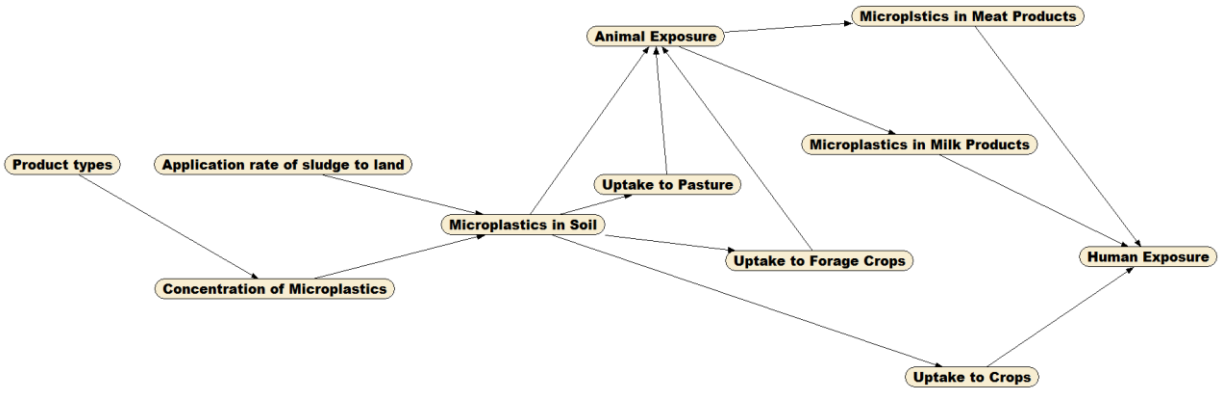
3.5.4. Risk assessment

Due to the limited quantitative information available on microplastics in the terrestrial environment, the risk assessment was attempted using the Bayesian network approach (Section 2.2.1).

The BBN model described here was developed using Netica™ version 3.25 by Norsys Systems Corp. In line with the guidance from Marcot et al (2006), the development of a BBN for this paper started with the creation of a conceptual model of the relationships (influence) diagram using data on sewage sludge production and application, information from studies linking exposure to microplastics and (primarily toxicological) outcomes, as well as expert knowledge and experience of sludge handling (Figure 3-18). The final outcome node is *Human exposure* [to microplastics and fibres].

The diagram represents key drivers (in nodes) of human exposure to microplastics and fibres due to sewage sludge application in Scotland. In the next phase each factor (node) was allocated values. In accordance with the considerable uncertainty in the data, all values were discretised into a number of categories or states. The number of states per node was kept to a minimum, and where possible Boolean values were adopted.

Figure 3-18 Conceptual diagram describing the main factors that drive human exposure to microplastics and fibres originating from sewage sludge application.



Model structure

The model structure (Figure 3-18) was broadly based around three main drivers of microplatic exposure; namely consumption of crops (this includes leafy vegetables, root vegetables, and cereals grown in soil amended with sewage sludge), consumption of milk products (milk, cheese, etc. sourced from cattle grazed on land amended with sewage sludge or cattle fed fodder crops grown on land amended with sewage sludge), and consumption of meat products (e.g. beef, lamb, pork etc. sourced from animals grazed on land amended with sewage sludge or animals fed fodder crops grown on land amended with sewage sludge). These groupings are based on a synthesis of the available literature and there is either implicit or explicit

evidence that they contribute to exposure to microplastics either in isolation or more often in combination.

Parameter learning

The current model (Figure 3-17) consists of 11 nodes and 14 links. The outcome node, *Human Exposure*, considers the process by which a particular receptor, given various dietary combinations and sources of food products, may become exposed to microplastics. The relationships between the different nodes are represented in conditional probability tables (CPTs). Parameter learning determines the prior CPT of each node of the network, given the link structures and available data. A CPT $P(A|B_1 \ldots B_n)$ was attached to each variable A with parents $B_1 \ldots B_n$. If A had no parents (e.g. *Product types* in Figure 3-18), the table was reduced to unconditional probabilities i.e. P(A). To illustrate, the prior unconditional probabilities for *Product types* was P(*Product types*), while the prior conditional probabilities for *Microplastics in soil* was P(*Microplastics in soil* | *Application rate of sludge to land*, *Concentration of microplastics*). Prior conditional probabilities were calculated by applying Bayes rule:

P(Microplastics in soil, Application rate of sludge to land, Concentration of microplastics)

= P(Microplastics in soil | Application rate of sludge to land, Concentration of microplastics)

* P(Application rate of sludge to land, Concentration of microplastics)

(Equation 3-2)

Prior unconditional probabilities were built using both ‘expert’ knowledge and actual observations. The ultimate objective is that all nodes are populated with empirical evidence. However, given the limitations of the information available, in the early stages of development the BBN was populated using a combination of expert knowledge and empirical evidence. With BBN’s ability to learn from new data, the model developed through an iterative process which improved our understanding and knowledge. Table 3-11 provides a list of all nodes and the information sources used to populate the prior CPTs. The aim was to develop prior probabilities that reflected the national (Scotland) situation; for example, looking at the *Product type* node, the prior distribution reflects the probability that any sewage sludge product available in Scotland is e.g. lime treated. The finalised BBN, complete with prior distributions is depicted in Figure 3-19.

Figure 3-19 Bayesian belief network (BBN) model of risk of human exposure to microplastics from sewage sludge, with prior probability distributions representative of Scotland

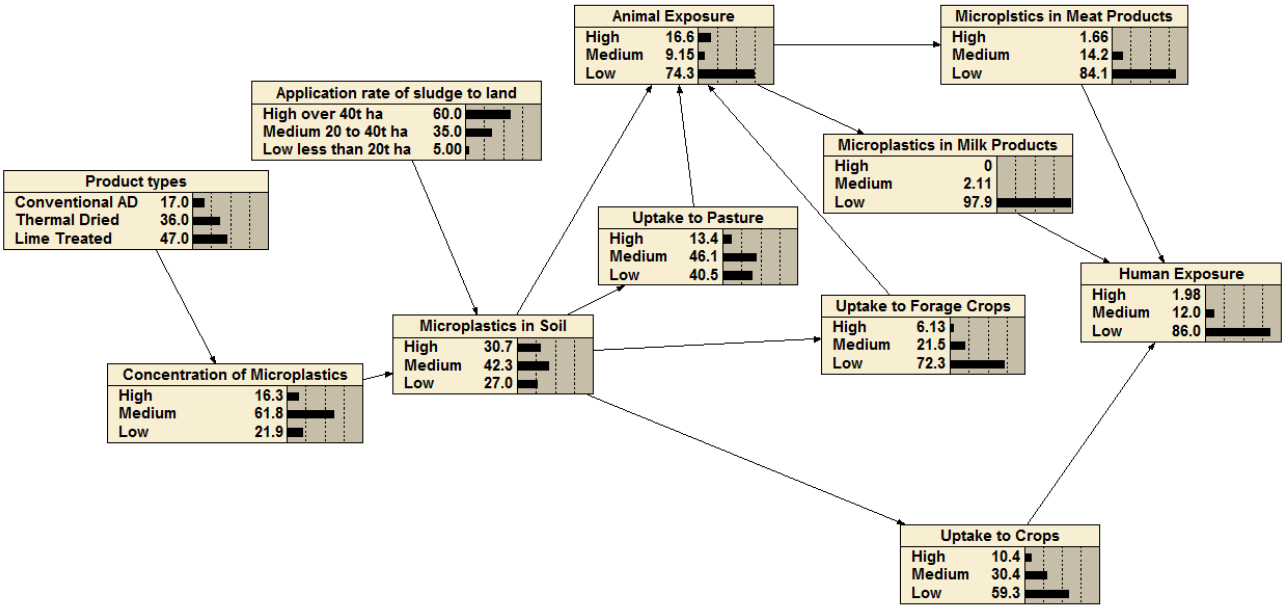


Table 3-11 List of nodes included in the Microplastics BBN, and the information used to populate prior conditional probability tables

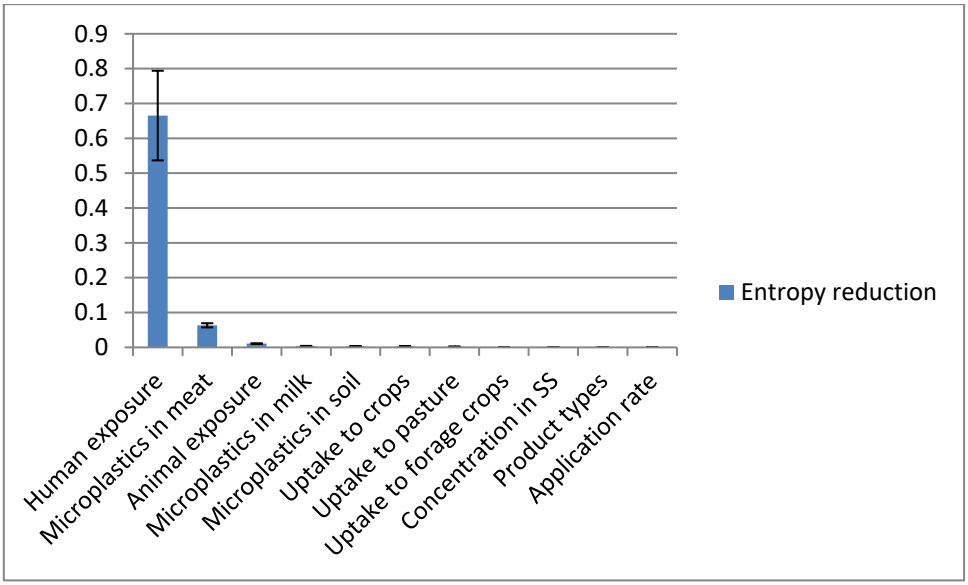
Node	Description	Information type/source
Product types	Probability of a sewage sludge product used in Scotland to be of a specific type (Conventional AD: Thermal drying: Lime Treated)	Based on sludge production figures obtained from Scottish Water
Concentration of microplastics	Probability that concentration of microplastics in sewage sludge product is high (>10000 n kg^{-1}); medium (>5000 <10000 n kg^{-1}); low (<5000 n kg^{-1})	Derived from Mahon et al. (2017); See Figure 3-17
Application rate of sludge to land	Application rate in t fw ha^{-1} (>40 t ha^{-1}: >20<40 t ha^{-1} : <20 t ha^{-1})	Based on application rates used in WRAP (2016c) and Hough et al. (2012) which were themselves based on expert judgement
Microplastics in soil	Probability that the concentration in soil post-application will be high (>120 n kg^{-1}); medium (>60 <120 n kg^{-1}); low (<60 n kg^{-1}).	Algorithm based on combination of concentration of microplastics, application rate and soil plough depth (i.e. volume) as used in WRAP (2016c)

Node	Description	Information type/source
Uptake to pasture	Probability that concentration of microplastics within the grass sward (not necessarily internalised) will be high; medium; low	Expert judgement assuming sewage sludge applied to surface of pasture.
Uptake to forage crops	Probability that concentration of microplastics within forage crops (not necessarily internalised) will be high; medium; low	Expert judgement assuming sewage sludge applied to soil surface prior to sowing
Uptake to crops	Probability that concentration of microplastics within crops for human consumption (not necessarily internalised) will be high; medium; low	Expert judgement assuming sewage sludge applied to soil surface prior to sowing
Microplastics in meat products	Probability that concentration of microplastics in meat products will be high; medium; low	Expert judgement informed by exotoxicology literature including Hussain et al. (2001); Carr et al. (2012); Yoo et al. (2011)
Microplastics in milk products	Probability that concentration of microplastics in milk products will be high; medium; low	Microplastics measured in milk have only been attributed to shearing from bottles etc.
Human exposure	Probability that human exposure to microplastics is high; medium; low	Algorithm combining probabilities of microplastics in the three exposure media

Structural learning

Sensitivity to findings analysis was performed to determine which variables had most influence on the outcome node, *Human exposure*. This was determined by calculating an entropy reduction value (ERV). The higher the value of ERV, the stronger a node affects another. Estimates of ERV, and the associated variance of beliefs, were calculated for *Human exposure* and then for the influence of each node in turn on *Human exposure* (Figure 3-20). It is noteworthy that all of the variables where operators have significant control have minimal influence on *Human exposure*, suggesting that changing product type or application rate will have little influence on levels of exposure to microplastics experienced by the local population. Levels of microplastics in meat products play the most significant role in the outcome of *Human exposure*. This reflects the fact that microplastics contained in meat products tend to be <1.5 μm (Yoo et al., 2011) which is the size fraction that can penetrate into the deep organs. Thus intake of microplastics in meat products is likely to pose the greatest risk to human health compared to the other exposure media included in this model.

Figure 3-20 Sensitivity of Human exposure due to a finding at another node. Bars represent entropy reduction value; error bars indicate the associated variance of beliefs.



Scenario testing – Forward inference

The BBN model can be used to test a variety of scenarios by forward inference. Here each node is set to its highest or lowest state to understand the impact of each factor on the risk of individuals being exposed to microplastics. Figure 3-21 shows, by way of illustration, a scenario where concentrations of microplastics in sewage sludge products are very high. Comparing the distribution of Human exposure with Figure 3-19 you can see a 0.52% increase in the probability that individuals will experience high levels of exposure to microplastics. The sensitivity to findings analysis (Figure 3-20) was used to structure a number of different scenarios, which were explored in turn (Table 3-12).

Figure 3-21 Examples of forward inference scenario. Top: here Concentration of microplastics is set to P(High) = 100% resulting in Human exposure P(High) = 2.5% (cf. 1.98% in Figure 3-19). Bottom: here Concentration of microplastics is set to P(Low) = 100% resulting in Human exposure P(High) = 1.36% (cf. 1.98% in Figure 3-19).

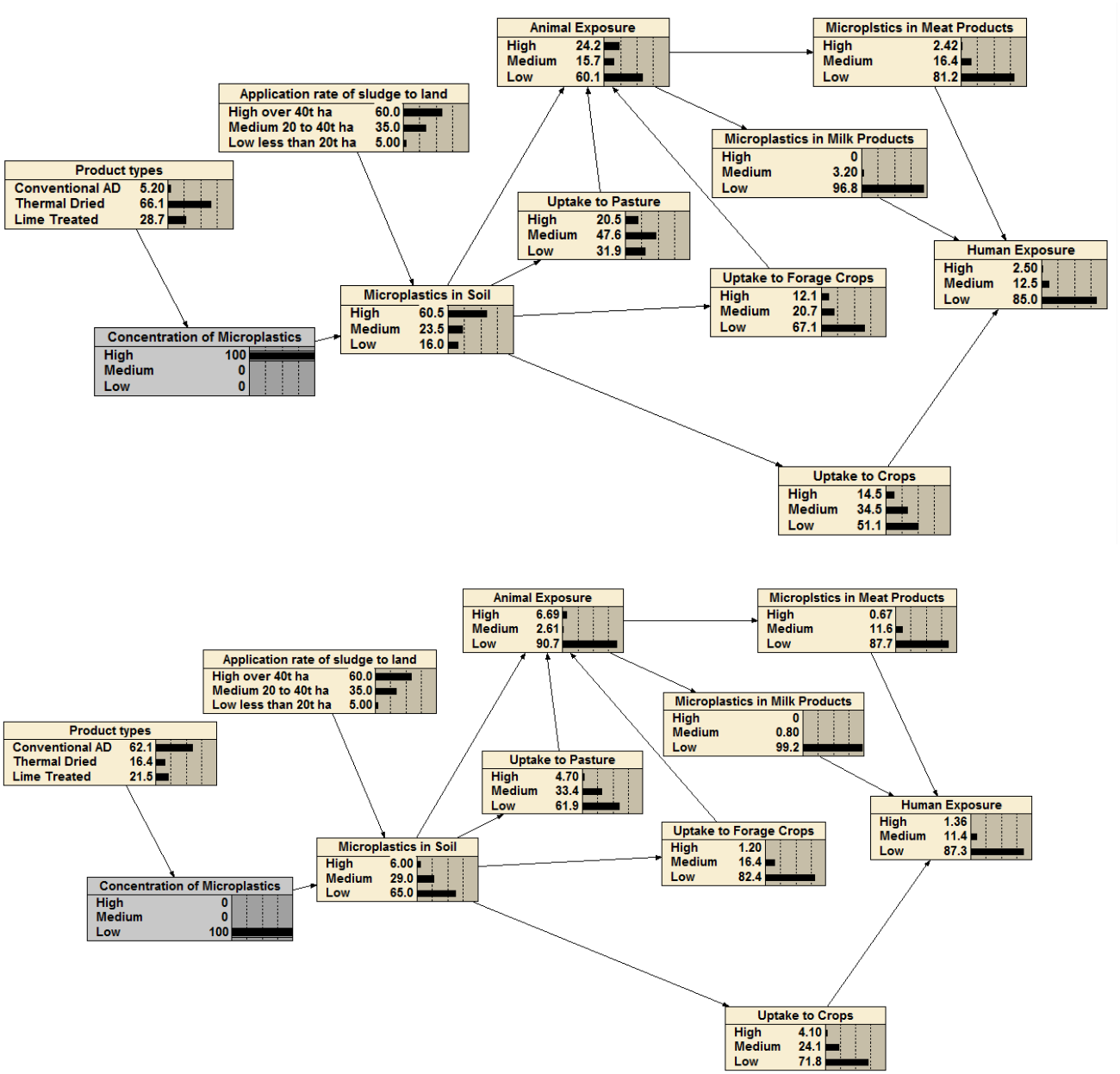


Table 3-12 Change in probability of human exposure to microplastics being high (ΔP[Human exposure(High)]) arising from different forward inference scenarios tested; Microplastics in meat products either set as its prior probability, or P(High) = 100%

Node	Scenario	ΔP[Human exposure(High)]
Microplastics in meat	P(High) = 100%	+14.92%
	P(Low) = 100%	-1.89%
Animal exposure	P(High) = 100%	+3.66%
	P(Low) = 100%	-0.90%
Microplastics in milk products	P(Medium) = 100%	+7.50%
	P(Low) = 100%	-0.16%
Microplastics in milk products; Microplastics in meat	P(Medium) = 100% P(High) = 100%	+15.01%
	P(Low) = 100% P(High) = 100%	+15.01%
Microplastics in soil	P(High) = 100%	+1.22%
	P(Low) = 100%	-0.94%
Microplastics in soil; Microplastics in meat	P(High) = 100% P(High) = 100%	+16.02%
	P(Low) = 100% P(High) = 100%	+10.02%
Uptake to crops	P(High) = 100%	+2.46%
	P(Low) = 100%	-0.51%
Uptake to crops; Microplastics in meat	P(High) = 100% P(High) = 100%	+28.02%
	P(Low) = 100% P(High) = 100%	+8.02%
Uptake to pasture	P(High) = 100%	+1.41%
	P(Low) = 100%	-0.64%
Uptake to pasture; Microplastics in meat	P(High) = 100% P(High) = 100%	+15.22%
	P(Low) = 100% P(High) = 100%	+15.72%
Uptake to forage crops	P(High) = 100%	+1.47%
	P(Low) = 100%	-0.18%
Uptake to forage crops; Microplastics in meat	P(High) = 100% P(High) = 100%	+16.02%
	P(Low) = 100% P(High) = 100%	+14.82%
Concentration of microplastics	P(High) = 100%	+0.52%
	P(Low) = 100%	-0.62%
Concentration of microplastics; Microplastics in meat	P(High) = 100% P(High) = 100%	+15.62%
	P(Low) = 100% P(High) = 100%	+13.62%

Node	Scenario	ΔP[Human exposure(High)]
Product types	P(Conventional) = 100%	-0.46%
	P(Lime treated) = 100%	+0.06%
Product types; Microplastics in meat	P(Conventional) = 100% P(High) = 100%	+14.22%
	P(Lime treated) = 100% P(High) = 100%	+14.82%
Application rate of sludge	P(High over 40 t Ha) = 100%	+0.15%
	P(Low less than 20 t Ha) = 100%	-0.62%
Application rate of sludge; Microplastics in meat	P(High over 40 t Ha) = 100% P(high) = 100%	+15.12%
	P(Low less than 20 t Ha) = 100% P(High) = 100%	+14.12

The forward inference scenario analysis concurs with the sensitivity to findings analysis, highlighting the pivotal role that Microplastics in meat has with respect to Human exposure to microplastics. The greatest risk of exposure to microplastics occurs where uptake of microplastics to crops is high combined with high levels of microplastics in meat products. The model suggests that very little can be done, from a management perspective, to mitigate against these exposures.

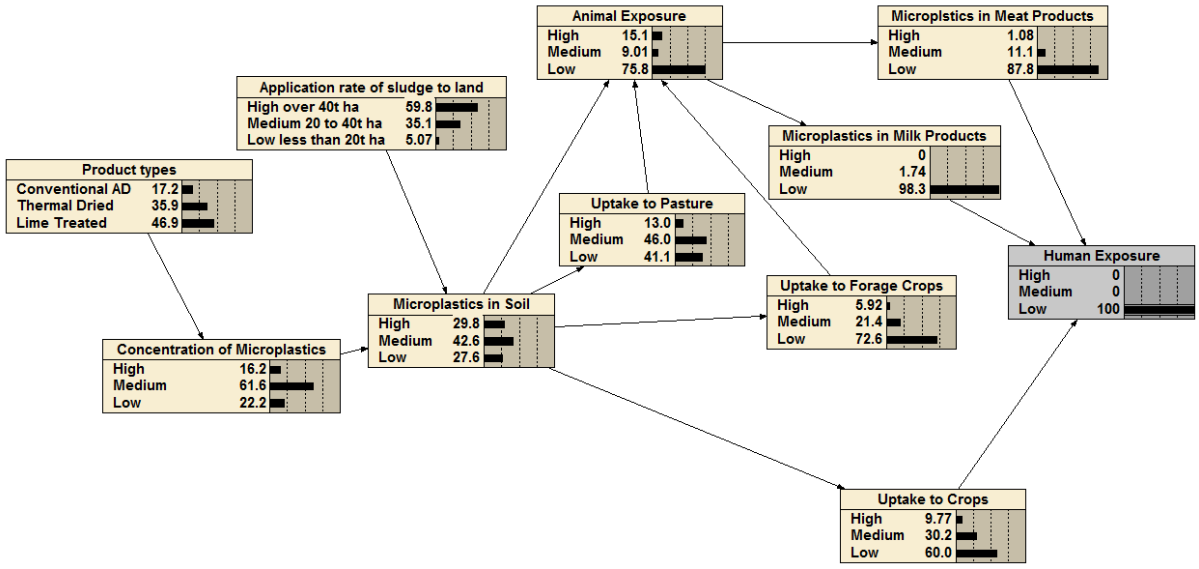
Any inferences drawn using this model should be treated with significant caution as a significant proportion of the CPTs within the model structure have been populated using judgement (essentially 6 out of the 11 nodes). Therefore, this model should be treated as a representation of the state of current knowledge based on the literature review undertaken as part of this assessment. The fact that the biggest drivers of risk are 'downstream' factors, suggests that the relationship between the presence of microplastics in sewage sludge, and final human exposure, is poorly understood.

Scenario testing – backwards inference

To investigate the conditions required to achieve *Human exposure* P(Low) = 100%, a backwards inference was performed (Figure 3-22). While the uncertainties in the model structure mean that any inferences should be treated with significant caution, the backwards inference does provide an indication of the steps that could be taken to reduce probability of exposure (given our current level of understanding of the system). In summary, these are:

- Reduce exposure of animals to microplastics – minimise use of sewage sludge on pasture, or incorporate it into the soil prior to pasture re-seeding
- Reduce sewage sludge application rates where practicable

Figure 3-22 Backwards inference by setting *Human exposure* to P(Low) = 100% (cf. Figure 3-19)



3.6. Human and animal pathogens

3.6.1. Background

In the UK and other developed countries, the application of sewage sludge to land is strictly regulated (ADAS, 2001; USEPA, 1993; Carrington, 2001; EPA Victoria, 2004; Pritchard et al., 2010; CEC, 1986; Defra, 2017). Under these legislative frameworks, sludge treatment processes are required to minimise or eliminate the presence of pathogens. In the UK, to ensure microbiological safety and consumer confidence, sewage sludges are usually treated and applied according to the requirements of the Safe Sludge Matrix. This sets out a risk-based 'table of crop types, together with clear guidance on the minimum acceptable level of treatment for any sewage sludge-based product which may be applied to that crop or rotation' (ADAS, 2001).

After application to land, depending on the soil type and rainfall, a proportion of the microorganisms associated with sewage sludge are retained in the soil surface layers. The practice of incorporating sewage sludge into the topsoil provides an initial immediate dilution on pathogens when expressed as a concentration. Numbers of pathogens in surface soils will further decline if the pathogens are transported to depth via percolation. Sewage sludge contains a range of pathogens (generally reflecting the prevalence of pathogens in the local human population), some of which are better adapted to survival in the soil environment than others. Depending on the pathogen, the soil physical conditions (i.e. temperature, soil type and moisture content) and soil biotic processes, there will be varying degrees of pathogen decay

in the soil (Pritchard et al., 2010; Gerba et al., 1975; van Elsas et al., 2012, McLaughlin et al., 2011).

Whilst studies in the US have provided limited evidence of health impacts associated with the application of sewage sludge (treated to EPA standards) to land (Dorn et al., 1985; NRC, 2002), it has been acknowledged that there are significant knowledge and research gaps. These include the occurrence, fate and potential health risks of known and emerging pathogens (WEAO, 2010; Sidhu & Toze, 2009; Viau et al., 2011). This section of the report aims to identify current and emerging risks to public health associated with pathogens in sewage sludges that are applied to land.

3.6.2. Emerging pathogens of concern

Bacteria

Previous reviews of pathogens in sewage sludge have identified the range of potential bacterial pathogens of concern (Sidhu & Toze, 2009; Pepper et al., 2006).

Escherichia coli is an important species in the context of the microbiological quality of sewage sludge. *E. coli* is a natural and ever-present member of the microbial community in the healthy human gut. Quality assurance approaches such as the Safe Sludge Matrix incorporate the use of *E. coli* as a faecal indicator organism (FIO) i.e. an indicator of the sanitary quality of the product to assess the effectiveness of treatment processes and by association the likely effect on pathogen reduction. The use of FIO bacteria for this purpose is well established in environmental microbiology and public health protection. There are, however, well-founded criticisms of this approach which surround the critical question of whether it is possible to infer reliably anything about pathogen risk from the presence of *E. coli*. The ability of *E. coli* to reappear (i.e. regrow or be resuscitated from an unculturable state) in dewatered sludge or in soil to which it is applied (Gerba et al., 2002) adds to the questions about the meaningfulness of the standards that rely on it.

Certain serotypes of E. coli are enteric pathogens in their own right. *E. coli* O157 emerged in the 1980s and has since been considered as an important bacterial hazard in risk assessments. The emergence of new pathogenic *E. coli* serotypes should be expected. In 2011 the novel *E. coli* O104 strain caused an outbreak of disease which resulted in more than 50 deaths and was eventually traced back to the consumption of beansprouts grown from fenugreek seeds imported from Egypt. This emphasises both the potential for new strains to emerge and for global transport to play a role in public health incidents. Infected people would have released *E. coli* O104 to sewer and it could have conceivably appeared in sewage sludge. However, it would be reasonable to expect that the controls in place to protect the public from E. coli O157 would also provide protection from newly emergent bacteria via the application to land route.

The potential for the release of zoonotic bacteria into the sewerage network poses a hypothetical risk for the environmental spread of pathogens via sewage sludge. A

large outbreak of Q-fever caused by the bacterium *Coxiella burnetii* occurred between 2007 and 2010 in the Netherlands and affected 4000 people, most likely due to inhalation of aerosolised bacteria from goat farms (van der Hoek et al., 2011). Schets et al. (2013) detected *C. burnetii* DNA at sewage works receiving discharge from Q fever positive goat farms; 36% of activated sludge samples were positive for *C. burnetii* DNA but at low levels. The probability of *C. burnetii* surviving sludge treatment in sufficient numbers to expose workers and local residents as a result of aerosolisation of sewage sludge is unknown. The bacteria can exist as small cell variants and small dense cells (SDC). SDC have a greater physical stability (related to heat, pressure and chemical agents) and are believed to be the persistent forms in the host and environment (ACDP, 2007). Survival in soil has also been demonstrated (Evstigneeva et al., 2007).

Viruses

It is well known that viruses pathogenic to humans are released in human faeces. They can therefore be expected to be found in untreated sewage entering WWTPs and in raw sludge by consequence of attachment to faecal solids and biomass generated in the wastewater treatment process. Adenoviruses are commonly detected in wastewater effluent (Enriquez et al., 1995) and are more resistant to thermal treatment than other viruses (Gerba et al., 2002). However, the infectivity of adenoviruses in sewage sludges can be effectively eliminated by lime treatment (Wei et al., 2009), although this practice does have implications for generation of malodour (Section 3.1).

Hepatitis A and E have been detected in raw sewage (Casas & Suñén, 2002) and in treated wastewater and sewage sludge (Clemente-Casares et al., 2009). It was reported that Hepatitis A RNA was significantly degraded after 60 days at 20°C in sewage sludge (Wei et al., 2010) and the Hepatitis A virus is rapidly inactivated at high pH brought about by lime treatment (Wei et al., 2010; Katz & Margolin, 2007). Hepatitis E virus (HEV) is an RNA virus that causes liver inflammation in humans, predominantly in developing countries. HEV genotype 3 was detected in 17% of samples studied from the Meuse River in the Netherlands which was inferred to have originated from sewage (Rutjes et al., 2009). This may in turn indicate a connection between piggery wastewater and sewage catchments, and incomplete wastewater treatment of the viral load. In Canada, Brassard et al. (2012) detected HEV of animal origin, and norovirus and rotavirus of human origin, on strawberries irrigated with river water. Whilst samples of irrigation water proved to be negative, the authors inferred that wastewater and animal faeces contaminating the river were the likely source and indicating a cause for concern regarding the environmental survival of these viruses.

Rotavirus and norovirus are the most common causes of acute gastroenteritis (Sidhu & Toze, 2009) especially in children and immunocompromised individuals (Pepper et al., 2006). As a result, these pathogens can be expected to be present at large sludge treatment facilities. Norovirus were found at a level of 105 norovirus L^{-1} in raw

sludge and remain at a relatively high level of 103 L^{-1} even after treatment (van der Berg et al., 2005). Wei et al. (2010) studied the infectivity of murine norovirus spiked into sludge over time and found that the virus can maintain some infectivity after 60 days in both 4°C and 20°C conditions. However, this needs to be put into the context of the quantitative risk assessment by Gale (2005) which considered the risk of infection from enteroviruses to be very low.

Protozoa

Several species of protozoa cause disease in humans, including *Giardia* and *Cryptosporidium* (Straub et al., 1993). Large quantities of *Cryptosporidium* oocysts and *Giardia* cysts are frequently found in treated sewage sludge (Robertson et al., 1992). They are known to be environmentally resistant, with cysts remaining viable for almost 2 months at 0 - 2°C (deRegnier et al., 1989) and oocysts for nearly 6 months at 4°C (Robertson et al., 1992). Gavaghan et al. (1993) assessed the inactivation of *Giardia* cysts during anaerobic digestion and showed that 99.9% of the cysts were inactivated within an 18-hour exposure period at 37°C. Risks associated with *Cryptosporidium parvum* and *Giardia* were assessed by Gale (2005) and were considered low if the multiple barriers built into schemes such as the Safe Sludge Matrix were applied. Since then, Amorós et al. (2016) have detected *Cryptosporidium* oocysts and *Giardia* cysts in digested sludge in Spain, but there was no update on the risks of exposure or health outcomes.

Antibiotic resistance

There is widespread concern that the effectiveness of antibiotics is in such rapid decline that their future utility is under threat in the short term (Wellington et al., 2013). Soil is a reservoir of naturally-occurring resistance genes, but there is growing interest in the way that anthropogenic activities, such as the application to land of animal and human faecal material, can contribute to the evolution of antibiotic resistance in the environment (Wellington et al., 2013). Sewage sludge contains antibiotics, antibiotic-resistant bacteria (ARBs) and antibiotic resistance genes (ARGs), which can be released into the environment via application to land (Bondarczuk et al., 2016). Pruden, (2014) stated that “It is now clear that human activities, including WWTPs, have a strong influence on the distribution of ARGs in the aquatic environment”. There is no evidence to suggest that ARBs present in soil to which sewage sludge has been applied are more likely to infect an exposed person than a susceptible bacterium, but the concern is that the soil becomes a reservoir for the development and spread of ARGs (Wellington et al., 2013). The principal concern is that the conditions may be created in the wastewater-land application system by which ARBs may develop as a result of the transfer of ARGs and/or the selection and proliferation of ARBs due to the presence of co-selecting agents in sewage sludge and sludge-amended soils such as biocides and metals (Wellington et al., 2013; Tezul & Pavlostathis, 2011; Ashbolt et al., 2013). Gaze et al.

(2011) investigated the prevalence of mobile genetic elements known as integrons carrying antibiotic and quaternary ammonium compound (QAC) resistance genes that confer resistance to detergents and biocides. Studies of class 1 integron prevalence in sewage sludge amended soil showed measurable differences compared with controls, although prevalence dropped sharply after a month. This study concluded that by selecting for class 1 integrons, detergents and biocides co-select for antibiotic resistance in sewage sludge.

Compared with observations in clinical settings, antibiotic resistance profiles are often detected at low percentages in wastewaters. Current knowledge on the prevalence and types of antibiotic resistance in wastewater and sewage sludge is limited (Rizzo et al., 2013). Biosolids samples were reported in several studies to contain a high concentration of ARBs in a range between 6.78 × 105 -4.46 × 10^8 CFU g^{-1} (Munir et al., 2011; Brooks et al., 2007; Auerbach et al. 2007; Munir & Xagoraraki, 2011). Prado et al. (2008) studied the presence of ESBL (extended-spectrum β-lactamase)-producing Klebsiella pneumoniae in the effluents and sludge of a hospital sewage treatment plant, evaluating the plant's potential to remove these microorganisms. They found antibiotic resistant Klebsiella penumoniae, some of which were multiple drug resistant, at all stages of sewage treatment including in sludge. Galvin et al., (2010) also found that ESBL-producing E. coli survived treatment in a modern secondary treatment facility although they took no samples from sludge. Munir et al. (2011) found that ARBs and ARGs were reduced by wastewater and sludge treatment processes, with significant differences observed in ARGs and ARB concentrations between anaerobic digestion/lime stabilization and dewatering and gravity thickening methods. Ju et al. (2016) reported that anaerobic digestion can achieve a 20–52% removal efficiency of total ARGs.

Burch et al. (2014) demonstrated that ARGs in sewage sludge decay relatively slowly following their application to agricultural soils (half-lives > 2 weeks). Although the removal efficiency is moderate, it was found that when treated with aerobic/anaerobic digestion, air-drying etc, ARGs decay in soil after land application at much faster rates of <1 week (Burch et al., 2013a; Burch et al., 2013b).

Prions – Risks from sewage sludge spreading to land

Prion diseases or transmissible spongiform encephalopathies (TSEs) are progressive neurodegenerative disorders that affect both humans and animals. The causative agents of TSEs are prions, which are abnormal, transmissible agents capable inducing abnormalities in normal cellular proteins called prion proteins found most abundantly in the brain. The UK public is most familiar with the prion disease Variant Creutzfeldt-Jakob disease (vCJD). There is strong evidence to suggest that the agent responsible for a prion disease in cows, bovine spongiform encephalopathy (BSE or 'mad cow' disease), is the same agent responsible for vCJD in humans, and that in the UK cross-species transmission occurred in the 1980s due to the entry of bovine offal into the food chain (Gale, 2006). BSE has been reduced

to extremely rare and isolated cases in the Scottish herd and there is no known risk of spread of this prion via any route including through sewage sludge.

Hypothetically, if a country had cases of prion disease, the transmissible agents could enter WWTPs from slaughterhouses, laboratories, or landfill leachate containing infected carcasses and other materials (Hinckley et al., 2008; Miles et al., 2011). Prions are insoluble in water and many detergents and are resistant to chemical and thermal degradation (Taylor, 2000). There are a few studies that have investigated prion survival in sewage sludge. Hinckley et al. (2008) found no significant degradation of prions during activated sludge treatment of the WWT process. Kirchmayr et al. (2006) found no reduction of prion survival under mesophilic conditions, but 20-40% reduction after 302 hours of incubation under thermophilic conditions. These studies strongly suggest that if prions were to enter the wastewater treatment system, most could survive mesophilic anaerobic digestion, and be present in treated sewage sludge.

With respect to the historical case of BSE in UK, Gale and Stanfield (2001) assessed the risk of prion-diseases from land application of sewage sludge and found it to be very low. The study adopted a Source-Pathway-Receptor approach to quantify the risk to humans through consumption of vegetable crops grown in sludge-amended soil and found it to be acceptably low, at 1.32×10^{-9} persons infected $year^{-1}$. More recently, assessments have been completed using similar approaches for TSE risks associated with the land spreading of mammalian meat and bone meal which support the reinstatement of this practice subject to a range of controls (Cummins & Adkin, 2007). Adkin et al. (2013) reappraised the TSE risk posed by the irrigation to pasture land of wastewater from facilities processing livestock. The results indicated that the predicted number of new TSE infections arising from the spreading of wastewater on pasture over one year would be low, with a mean of one infection every 1,000 years for BSE in cattle, and one infection every 30 years and 33 years for classical and atypical scrapie, respectively.

In conclusion, the risks to humans from prions in sewage sludge are negligible.

4. Conclusions

Under good practice (e.g. adherence to the Safe Sludge Matrix) and normal circumstances, none of the potentially hazardous agents assessed posed significant health risks. Under realistic worst-case circumstances, a number of potentially hazardous agents were estimated to pose risk to health for human receptors. These are listed in Table 4-1 along with possible mitigation suggestions. It should be noted that these potential risks have been identified as part of a theoretical mathematical exercise with any mitigation (e.g. by taking a precautionary approach to minimise or eliminate potential risks) only necessary if the predicted exposures and associated risks were found to be real.

Table 4-1 – List of potentially hazardous agents for which a (semi-)quantitative estimate of risk was possible

Potentially hazardous agent	Outome	Magnitude of risk (worst case)	Uncertainty	Possible mitigation
Malodour	Annoyance	Medium	High	• Distance to residents from spreading operations >3km • Avoid weather conditions that are conducive for exposure (wind speed >6 m s^{-1}; 50% night time cloud cover; partial daytime solar radiation) • Application rate <20 t ha^{-1} • Avoid the use of lime treated sludge
Nonylphenol	HQ>1	Low	Medium	• AD may reduce concentrations but evidence compounded by ready transformation of NP2EO to NP
Nonylphenol diethoxylate	HQ>1	Low	High	
PBDE-99	HQ>1	Low	Medium	• Partially broken down by AD
PBDE-209	HQ>1	Low	Medium	
Benzothiazole	HQ>1	Low-Medium	High	• Should be removed during aerobic treatment
Triclocarban	HQ>1	Low	Medium	• Partial removal by thermal hydrolysis
Cyclomethicone 5	HQ>1	Low	Medium	• Cycolmethicone 5 readily degraded by aerobic treatment and AD • Cyclomethicone 6 more likely to remain in sludges
Cyclomethicone 6	HQ>1	Low	Medium	
Atenolol	HQ>1	Low	High – Very High	• Aerobic waste water treatment followed by anaerobic digestion

Chemical exposures (general)	HQ>1	Low - Medium	Medium – Very High	• Any risks could be further attenuated by restricting sewage sludge use to pasture/forage crops • Extremely precautionary approach

This analysis has focussed on emerging contaminants and issues, and as such it was not possible to assess a proportion of the potentially hazardous agents (semi-)quantitively in a meaningful way. There is very little known about microplastics in the terrestrial environment, and while they are likely present in sewage sludge, it is not possible to determine if people will end up being exposed to them or what the consequences of this exposure might be. Similarly, while the majority of pathogens potentially present in sewage sludge have been shown to pose minimal risk to human receptors, the significance of sewage sludge as a reservoir of antibiotic resistance is poorly understood.

Overall, the impacts of sewage sludge (when used correctly) on physical health outcomes is likely to be minimal and not significantly different to other organic soil amendments (e.g. animal manures). Impacts associated with quality of life and well-being (such as annoyance from malodour) can, however, be much more acute. These impacts are not exclusive to sewage sludge but are associated with a range of industrial and agricultural practices. In our opinion, physical health risks have been by and large managed through improvements in sludge treatment processes as well as best practice; however, the impacts on well-being have been largely ignored.

It should be noted that ‘emerging’ potentially hazardous agents is an ever-changing situation. The main hazards of concern today are likely to be superseded in the future. Since the 2008 report (SNIFFER 2008) we have seen the focus move from inorganic contaminants towards organics and pharmaceuticals, as well as ‘novel’ pathogens and antimicrobial resistance. Over the next 10 years, a different set of hazards are likely to become priority. Protecting public and environmental health is an on-going process.

5. References

Aalders, I., Hough, R.L. & Towers, W. 2011. Risk of erosion in peat soils – an investigation using Bayesian belief networks. *Soil Use and Management* **27:**538-549.

Aatamila, M., Verkasalo, P.K., Korhonen, M.J., Suominen, A.L., Hirvonen, M-R., Viluksela, M.K. & Nevalainen, A. 2011. Odour annoyance and physical symptoms among residents living near waste treatment centres. *Environmental Research* **111:**164-170.

Aatamila, M., Verkasalo, P.K., Korhonen, M.J., Viluksela, M.K., Pasanen, K. Tiittanen, P. & Nevalainen, A. 2010. Odor annoyance near waste treatment centres: A population-based study in Finland. *Journal of the Air & Waste Management Association* **60:**412-418.

Abbott, J. 1993. Enclosed wastewater treatment plants – health and safety considerations. Foundation for Water Research Report FR/W0001

ACDP. 2007. Occupational Exposure to Coxiella burnetii (Q fever) in the Agriculture and Food Sector. Advisory Committee on Dangerous Pathogens, HSE, ACDP/85/P8.

Adams, G., Hargreaves, R., Witherspoon, J., Ong, H., Burrowes, P., Easter, C., et al. 2003. Identifying and controlling odour in the municipal wastewater environment phase 1: literature search and review. IWA publishing 2003.

ADAS. 2001. The Safe Sludge Matrix: Guidelines for the Application of Sewage Sludge to Agricultural Land. 3rd Edition, ADAS, Gleadthorpes.

Adkin, A., Donaldson, N. & Kelly, L. 2013. A Quantitative Assessment of the Prion Risk Associated with Wastewater from Carcass-Handling Facilities. *Risk Analysis* **33:**1212–1227.

Ambrose, D., Ellender, J.H., Sprake, C.H.S. & Townsend, R. 1975. Thermodynamic properties of fluorine compounds. Part 15. – Vapour pressures of the three tetrafluorobenzenes and 1,3,5-trichloro-2,4,6-trifluorobenzene. *Journal of the Chemistry Society, Faraday Transactions 1: Physical Chemistry of Condensed Phases* **71:**35-41.

Amorós, I., Moreno, Y., Reyes, M., Moreno-Mesonero, L. & Alonso, J.L. 2016. Prevalence of Cryptosporidium oocysts and Giardia cysts in raw and treated sewage sludges. *Environmental Technology* **37:**2898–2904.

Andrady, A.L. 2011. Microplastics in the marine environment. *Marine Pollution Bulletin* **62:**1596-1605.

Arnold, D.L., Bryce, F., Stapley, R., McGuire, P.F., Burns, D., Tanner, J.R. & Karpinski, K. 1993a. Toxicological consequences of Aroclor 1254 ingestion by

female Rhesus (*Macaca mulatta*) monkeys, Part 1A: Prebreeding phase – clinical health findings. *Food and Chemical Toxicology* **31:**799-810.

Arnold, D.L., Bryce, F., Karpinski, K., Mes, J., Farnie, S., Tryphonas, H., Truelove, J., McGuire, P.F., Burns, D., Tanner, J.R., Stapley, R., Zawidzka, Z.Z. & Basford, D. 1993b. Toxicological consequences of Araclor 1254 ingestion by female Rhesus (*Macaca mulata*) monkeys, Part 1B: prebreeding phase – clinical and analytical laboratory findings. *Food and Chemical Toxicology* **31:**811-824.

Ashbolt, N.J., Amézquita, A., Backhaus, T., Borriello, P., Brandt, K.K., Collignon, P., Coors, A., Finley, R., Gaze, W.H., Heberer, T., Lawrence, J.R., Larsson, D.G., McEwen, S.A., Ryan, J.J., Schofield, J., Silley, P., Snape, J.R., Vab den Eede, C. & Topp, E. 2013. Human Health Risk Assessment (HHRA) for environmental development and transfer of antibiotic resistance. *Environmental Health Perspectives* **121:**993–1001.

Ashton, K., Holmes, L. & Turner, A. 2010. Association of metals with plastic production pellets in the marine environment. *Marine Pollution Bulletin* **60:**2050-2055.

Astrom, L. 2016. Shedding of Synthetic Microfibres from Textiles. Thesis, University of Gothenburg, Sweden.

Auerbach, E.A., Seyfried, E.E. & McMahon, K.D. 2007. Tetracycline resistance genes in activated sludge wastewater treatment plants. *Water Research* **41:**1143–1151.

Avdeef, A. 1993. pH-metric log *P*. II: Refinement of partition coefficients and ionization constants of multiprotic substances. *Journal of Pharmaceutical Sciences* **82:**183-190.

Avdeef, A. 1987. Seminar on Ionization & Lipophilicity. Log P values measured by pION Inc. In: Brookline, MA. Avdeef A, Berger C, eds (1987)

Bakir, A., Rowland, S.J. & Thompson, R.C. 2012. Competitive sorption of persistent organic pollutants onto microplastics in the marine environment. *Marine Pollution Bulletin* **64:**2782-2789.

Barnes, G. & Dourson, M. 1988. Reference Dose (RfD): Description and use in health risk assessment. *Regulatory Toxicology and Pharmacology* **8:**471-486.

Bashari H, Smith C, Bosch OJH. 2009. Developing decision support tools for rangeland management by combining state and transition models and Bayesian belief networks. *Agricultural Systems* **99:**23-34.

Besseling, E., Wegner, A., Foekema, E.M., van den Heuvel-Greve, M.J. & Koelmans, A.A. 2013. Effects of microplastic on fitness and PCB bioaccumulation by

the lugworm *Arenicola marina* (L.). *Environmental Science & Technology* **47:**593-600.

Bhhatarai, B. & Gramatica, P. 2011. Prediction of aqueous solubility, vapor pressure and critical micelle concentration for aquatic partitioning of perfluorinated chemicals. *Environmental Science & Technology* **45:**8120-8128.

Bidleman, T.F. & Renberg, L. 1985. Determination of vapor pressures for chloroguaiacols, chloroveratroles, and nonylphenol by gas chromatography. *Chemosphere* **14:**1475-1481.

Black, S., Grieve, D. & Llewellyn A. 2016. Biosolids Assurance Scheme. 21st European Biosolids and Organic Resources Conference and Exhibition. Edinburgh, November 2016.

Blanes-Vidal, V., Bælum, J., Nadimi, E.S., Løfstrøm, P. & Christensen, L.P. 2014. Chronic exposure to odorous chemicals in residential areas and effects on human psychosocial health: Dose-response relationships. *Science of the Total Environment* **490:**545-554.

Bondarczuk, K., Markowicz, A. & Piotrowska-Seget, Z. 2016. The urgent need for risk assessment on the antibiotic resistance spread via sewage sludge land application. *Environment International* **87:** 49–55.

Bonnin, C., Laborie, A. & Paillard, H. 1990. Odor nuisances created by sludge treatment: problems and solutions. *Water Science & Technology* **22:**65-74.

Boon, A.G. 1995. Septicity in sewers: causes, consequences and containment. *Water Science & Technology* **31:**237-253.

Bosker, T., Behrens, P. & Vijver, M.G. 2017. Determining global distribution of microplastics by combining citizen science and in-depth case studies. *Integrated Environmental Assessment and Management* **13:**536-541.

Bouwmeester, H., Hollman, P.C.H. & Peters, R.J.B. 2015. Potential health impact of environmentally released micro- and nanoplastics in the human food production chain: Experiences from nanotechnology. *Environmental Science & Technology* **49:**8932-8947.

Brandli, R.C., Kupper, T., Bucheli, T.D., Zennegg, M., Huber, S., Ortelli, D., Muller, J., Schaffner, C., Iozza, S., Schmid, P., Berger, U., Edder, P., Oehme, M., Stadelmann, F.X. & Tarradellas, J. 2007. Organic pollutants in compost and digestate. Part 2. Polychlorinated dibenzo-p-dioxins, and -furans, dioxin-like polychlorinated biphenyls, brominated flame retardants, perfluorinated alkyl substances, pesticides, and other compounds. *Journal of Environmental Monitoring* **9:**465–472.

Brassard, J., Gagné, M-J., Généreux, M. & Côté, C. 2012. Detection of human-borne and zoonotic viruses on irrigated, field-grown strawberries. *Applied and Environmental Microbiology* **78:**3763-3766.

Brennecke, D., Duarte, B., Paiva, F., Caçador, I. & Canning-Clode, J. 2016. Microplastics as a vector for heavy metal contamination from the marine environment. *Estuarine, Coastal and Shelf Science* **178:**189-195.

Brennan, B. 1993. Odour nuisance. *Water and Waste Treatment* **36:**30-33.

Bright, D.A. & Healey, N. 2003. Contaminant risks from biosolids land application: Contemporary organic contaminant levels in digested sewage sludge from five treatment plants in Greater Vancouver, British Columbia. *Environmental Pollution* **126:**39-49.

Brooks, J.P., Maxwell, S.L., Rensing, C., Gerba, C.P. & Pepper, I.L. 2007. Occurrence of antibiotic-resistant bacteria and endotoxin associated with the land application of biosolids. *Canadian Journal of Microbiology* **53:**616-622.

Browne, M.A., Crump, P., Niven, S.J., Teuten, E., Tonkin, A., Galloway, T. & Thompson, R. 2011. Accumulation of microplastic on shorelines worldwide: Sources and sinks. *Environmental Science & Technology* **45:**9175-9179.

Browne, M.A., Dissanayake, A., Galloway, T.S., Lowe, D.M. & Thompson, R.C. 2008. Ingested microscopic plastic translocates to the circulatory system of the mussel, *Mytilus edulis* (L.). *Environmental Science & Technology* **42:**5026-5031.

Burch, T.R., Sadowsky, M.J. & LaPara, T.M. 2014. Fate of Antibiotic Resistance Genes and Class 1 Integrons in Soil Microcosms Following the Application of Treated Residual Municipal Wastewater Solids. *Environmental Science & Technology* **48:**5620–5627.

Burch, T.R., Sadowsky, M.J. & LaPara, T.M. 2013a. Air-Drying Beds Reduce the Quantities of Antibiotic Resistance Genes and Class 1 Integrons in Residual Municipal Wastewater Solids. *Environmental Science & Technology* **47:**9965–9971.

Burch, T.R., Sadowsky, M.J. & LaPara, T.M. 2013b. Aerobic digestion reduces the quantity of antibiotic resistance genes in residual municipal wastewater solids. **12:**4;17.

Carballa M. Omil F., Lema J.M., Llompart M., Garcia-Jares C., Rodriguez I., Gomez M. & Ternes T. 2004. Behaviour of pharmaceuticals, cosmetics and hormones in a sewage treatment plant. *Water Research*. **38:**2918-2926.

Carr, S.A. 2017. Sources and dispersive modes of micro-fibers in the environment: Environmental Microfiber Sources. *Integrated Environmental Assessment & Management* **13:**466-469.

Carr, S.A., Liu, J. & Tesoro, A.G. 2016. Transport and fate of microplastic particles in wasterwater treatment plants. *Water Research* **91:**174-182.

Carr, K.E., Smyth, S.H., McCullough, M.T., Morris, J.F. & Moyes, S.M. 2012. Morphological aspects of interactions between microplastics and mammalian cells: intestinal uptake and onward movement. *Progress in Histochemistry and Cytochemistry* **46:**185-252.

Carrington, E.G. 2001. Evaluation of sludge treatments for pathogen reduction. European Commission. Report No.: CO 5026/1.

Carter, L.J., Ryan, J.J. & Boxall, A.B.A. 2016. Effects of soil properties on the uptake of pharmaceuticals into earthworms. *Environmental Pollution* **213:**922–931

Casas, N. & Suñén, E. 2002. Detection of enteroviruses, hepatitis A virus and rotaviruses in sewage by means of an immunomagnetic capture reverse transcription-PCR assay. *Microbiological research* **157:**169–175.

Castañeda, R.A., Avlijas, S., Simard, M.A. & Ricciardi, A. 2014. Microplastic pollution in St. Lawrence River sediments. *Canadian Journal of Fisheries and Aquatic Sciences* **71:**1767-1771.

Cauwenberghe, L.V., Vanreusel, A., Mees, J. & Janssen, C.R. 2013. Microplastic pollution in deep-sea sediments. *Environmental Pollution* **182:**495-499.

CEC. 1986. Directive 86/278/EEC - Soil protection when sewage sludge is used in agriculture.

Cervinka, R. & Neudorfer, E. 2004. Odour control at a municipal sewage collector: psychological perspectives. *VDI Bericht* **1850:**13-25.

Cevasco, A., Urbatzka, R., Bottero, S., Massari, A., Pedemonte, F., Kloas, W., Mandich, A. 2008. Endocrine disrupting chemicals (EDC) with (anti)estrogenic and (anti)androgenic modes of action affecting reproductive biology of *Xenopus laevis*: II. Effects on gonad histomorphology. *Comparative Biochemistry and Physiology, Part C* **147:**241-251.

Charlton, A., Sakrabani, R., Tyrrel, S., Casado, M.R., McGrath, S.P., Crooks, B., Cooper, P. & Campbell, C.D. 2016. Long-term impact of sewage sludge application on soil microbial biomass: An evaluation using meta-analysis. *Environmental Pollution* **219:**1021–1035.

Chemicals Inspection and Testing Institute. 1992. Biodegradation and bioaccumulation data of existing chemicals based on the CSCL Japan. Japan Chemical Industry Ecology - Toxicology and Information Center. ISBN 4-89074-101-1

Cheremisinoff, P.N. 1988. Industrial Odour Control. Butterworth-Heinemann Ltd., Oxford, UK.

Clarke, B.O. & Smith, S.R. 2011. Review of 'emerging' organic contaminants in biosolids and assessment of international research priorities for the agricultural use of biosolids. *Environment International* **37:**226-247.

Clarke, R., Healy, M.G., Fenton, O. & Cummins, E. 2016. A quantitative risk ranking model to evaluate emerging organic contaminants in biosolids amended land and potential transport to drinking water. *Human and Ecological Risk Assessment* **22:**958-990.

Clegg, D., Sakai, C. & Voytek, P. 1986. Assessment of reproductive risks. *Biology of Reproduction* **34:**5-16.

Clemente-Casares, P., Rodriguez-Manzano, J. & Girones, R. 2009. Hepatitis E virus genotype 3 and sporadically also genotype 1 circulate in the population of Catalonia, Spain. *Journal of Water and Health* **7:**664-673.

Cole, M., Lindeque, P.K., Fileman, E., Halsband, C. & Galloway, T. 2015. The impact of microplastics on feeding, function and fecundity in the copepod *Calanus helgolandicus*. *Environmental Science & Technology* **49:**1130-1137.

Cole, M., Lindeque, P., Fileman, E., Halsband, C., Goodhead, R., Moger, J. & Galloway, T.S. 2013. Microplastic ingestion by zooplankton. *Environmental Science & Technology* **47:**6646-6655.

Cooper, D.A. & Corcoran, P.L. 2010. Effects of mechanical and chemical processes on the degradation of plastic beach debris on the island of Kauai, Hawaii. *Marine Pollution Bulletin* **60:**650-654.

Coover, M.P. & Sims, R.C. 1987. The effect of temperature on polycylic aromatic hydrocarbon persistence in an unacclimated agricultural soil. *Hazardous Waste and Hazardous Materials* **4:**69-82.

Cornu, S., Neal, C., Anbrosi, J.P., Whitehead, P., Neal, M., Sigolo, J. & Vachier, P. 2001. The environmental impact of heavy metals from sewage sludge in ferralsols (São Paulo, Brazil). *Science of The Total Environment* **271:**27–48.

COT COM. 2001. Committees on Toxicity Mutagenicity Carcinogenicity of Chemicals in Food, Consumer Products and the Environment. Annual Report 2001.

Crump, K.S. 1984. A new method for determining allowable daily intakes. *Fundamental and Applied Toxicology* **4:**851-871.

Cummins, E. & Adkin, A. 2007. Exposure Assessment of TSEs from the Landspreading of Meat and Bone Meal. *Risk Analysis* **27:**1179–1202.

Dal Pozzo, A., Donzelli, G., Rodriquez, L. & Tajana, A. 1989. “In vitro” model for the evaluation of drug distribution and plasma protein-binding relationships. *International Journal of Pharaceutics* **50:**97-101.

Daubert, T.E. & Danner, R.P. 1989. Physical and Thermodynamic Properties of Pure Chemicals Data Compilation. Washington, D.C.: Taylor and Francis, 1989.

Defra. 2017. Sewage sludge on farmland: code of practice for England, Wales and Northern Ireland.

Defra 2002. Guidelines for Environmental Risk Assessment and Management. http://www.defra.gov.uk/publications/2011/11/07/green-leaves-iii-pb13670/ Last accessed 16/04/12

De Bruijn, J., Busser, F., Seinen, W. & Hermens, J. 1989. Determination of octanol/water partition coefficients for hydrophobic organic chemicals with the “slow-stirring” method. *Environmental Toxicology and Chemistry* **8:**499-512.

DeFoe, D.L., Holcombe, G.W., Hammermeister, D.E. & Biesinger, K.E. 1990. Solubility and toxicity of eight phthalate esters to four aquatic organisms. *Environmental Toxicology and Chemistry* **9:**623-636.

De Maagd, P.G-J., ten Hulscher, D.Th.E.M., van den Heuvel, H., Opperhuizen, A. & Sijm, D.T.H.M. 1998. Physicochemical properties of polycylic aromatic hydrocarbons: Aqueous solubilities, *n*-pctanol/water partitian coefficients, and Henry’s law constants. *Environmental Toxicology and Chemistry* **17:**251-257.

deRegnier, D.P., Cole, L., Schupp, D.G. & Erlandsen, S.L. 1989. Viability of Giardia cysts suspended in lake, river, and tap water. *Applied and Environmental Microbiology* **55:**1223–1229.

Dietrich, S., Ploessl, F., Bracher, F., Laforsch, C. 2010. Single and combined toxicity of pharmaceuticals at environmentally relevant concentrations in *Daphnia magna* – A multigenerational study. *Chemosphere* **79:**60-66.

Dorn, C.R., Reddy, C.S., Lamphere, D.N., Gaeuman, J.V. & Lanese, R. 1985. Municipal sewage sludge application on Ohio farms: health effects. *Environmental Research* **38:**332–359.

Dris, R., Gasperi, J., Rocher, V., Saad, M., Renault, N. & Tassin, B. 2015. Microplastic contamination in an urban area: a case study in Greater Paris. *Environmental Chemistry* **12:**592-599

Duis, K. & Coors, A. 2016. Microplastics in the aquatic and terrestrial environment: sources (with a specific focus on personal care products), fate and effects. *Environmental Sciences Europe* **28:**2.

ECHA. 2015a. Search for Chemicals. Bis(pentabromophenyl) ether (CAS 1163-19-5) Registered Substances Dossier. European Chemical Agency. Available from, as of Mar 24, 2015: http://echa.europa.eu/

ECHA. 2015b. Search for Chemicals. Decamethylcyclopentasiloxane (CAS 541-02-6) Registered Substances Dossier. European Chemical Agency. Available from, as of Aug 12, 2015: http://echa.europa.eu/

ECHA. 2013. Member state committee support document for the identification of 4-Nonylphenol, branched and linear, ethoxylated. European Chemicals Agency 2013 https://echa.europa.eu/documents/10162/9af34d5f-cd2f-4e63-859c-529bb39da7ae

EFSA. 2016. Presence of microplastics and nanoplastics in food, with particular focus in seafood. EFSA Panel on contaminants in the food chain (CONTAM). *EFSA Journal* **14(6):**4501.

Eggen, T., Asp, T.N., Grave, K. & Hormazabal, V. 2011. Uptake and translocation of metformin, ciprofloxacin and narasin in forage- and crop plants. *Chemosphere* **85:**26-33.

Ellington, J.J. & Floyd, T.L. 1996. Octanol/water partition coefficients for eight phthalate esters. EPA/600/S-96/006, Sept. 1996; Athens, GA: USEPA, National Exposure Research Lab (1996)

Elliott, S., Cole, D., Krueger, P., Voorberg, N. & Wakefield, S. 1999. The power of perception: health risk attributed to air pollution in an urban industrial neighbourhood. *Risk Analysis* **19:**621-634.

Emel'yanenko, V.N. & Verevkin, S.P. 2008. Thermodynamic properties of caffeine: Reconciliation of available experimental data. *Journal of Chemical Thermodynamics* **40:**1661-1665.

Endo, S., Takizawa, R., Okuda, K., Takada, H., Chiba, K., Kanehiro, H., Ogi, H., Yamashita, R. & Date, T. 2005. Concentration of polychlorinated biphenyls (PCBs) in beaches resin pellets: variability among individual particles and regional differences. *Marine Pollution Bulletin* **50:**110-114.

Engler, R. 2012. The complex interaction between marine debris and toxic chemicals in the ocean. *Environmental Science & Technology* **46:**12302-12315.

Enriquez, C.E., Hurst, C.J. & Gerba, C.P. 1995. Survival of the enteric adenoviruses 40 and 41 in tap, sea, and waste water. *Water Research* **29:**2548–2553.

Environment Canada. 2010. Ecological State of the Science Report on Decabromodiphenyl Ether (decaBDE), Bioaccumulation and Transformation (August 2010). Available from, as of Mar 24, 2015: http://www.ec.gc.ca/lcpe-cepa/documents/substances/decabde/ess_report_decabde-eng.pdf

EPA Victoria. 2004. Guidelines for Environmental Management; Biosolids Land Application. EPA Victoria, Southbank, Victoria, Australia.

Erhard, H.W. & Rhind, S.M. 2004. Prenatal and postnatal exposure to environmental pollutants in sewage sludge alters emotional reactivity and exploratory behaviour in sheep. *Science of the Total Environment* **332:**101-108.

Eriksen, G. S., Amundsen, C. E., Bernhoft, A., Eggen, T., Grave, K., Halling-Sørensen, B., Källqvist, T., Sogn, T. & Sverdrup, L. 2009. Risk assessment of contaminants in sewage sludge applied on Norwegian soils. Opinion of the panel on contaminants in the Norwegian Scientific Committee for Food Safety. Norwegian Scientific Committee for Food Safety (VKM). 05/511-22-final.

Eriksen, M., Lebreton, L.C.M., Carson, H.S., Thiel, M., Moore, C.J., Borerro, J.C., Galgani, F., Ryan, P.G. & Reisser, J. 2014. Plastic pollution in the world's oceans: more than 5 trillion plastic pieces weighing over 250,000 tons afloat at sea. *PLoS ONE* **9:**e111913.

Eriksen, M., Mason, S., Wilson, S., Box, C., Zellers, A., Edwards, W., Farley, H. & Amato, S. 2013. Microplastic pollution in the surface waters of the Laurentian Great Lakes. *Marine Pollution Bulletin* **77:**177-182.

European Commission. 2012. Occurrence and Levels of Selected Compounds in Europe Sewage Sludge Samples. Results of a Pan-European Screening Experience (FATE SEES).

European Commission. 2005. Proposal for a Guideline on the definition of a Potential Serious Risk to Human or Animal Health or for the Environment. Brussels, 08.02.2005

European Union. 2001. Diphenyl ether, pentabromo derivative (pentabromodiphenyl ether). European Union Risk Assessment Report. Luxembourg: Office for Official Publications of the European Committees, p. 1-124 (2001)

Eurostat. 2016. Sewage sludge production and disposal. https://ec.europa.eu/eurostat/data/database

Evstigneeva, A.S., Ul'yanova, T. Yu. & Tarasevich, I.V. 2007. The survival of Coxiella burnetii in soils. *Eurasian Soil Science* **40:**565–568.

Fendall, L.S. & Sewell, M.A. 2009. Contributing to marine pollution by washing your face: microplastics in facial cleansers. *Marine Pollution Bulletin* **58:**1225-1228.

Fini, A., Fazio, G. & Rabasco, A.M. 1986./ Partición 1-octanol/agua de sales de Diclofenac. *Acta Technologiae et Legis Medicamenti* **4:**33-44. [In Italian]

Fowler, P.A., Dora, N.J., McFerran, H., Amezaga, M.R., Miller, D.W., Lea, R.G., Cash, P., McNeilly, A.S., Evans, N.P., Cotinot, C., Sharpe, R.M. & Rhind, S.M. 2008.

In utero exposure to low doses of environmental pollutants disrupts fetal ovarian development in sheep. *Molecular Human Reproduction* **14:**269-280.

Free, C.M., Jensen, O.P., Mason, S.A., Eriksen, M., Williamson, N.J. & Boldgiv, B. 2014. High-levels of microplastic pollution in a large, remote, mountain lake. *Marine Pollution Bulletin* **85:**156-163.

Fries, E., Dekiff, J.H., Willmeyer, J., Neulle, M-T., Ebert, M. & Remy, D. 2013. Identification of polymer types and additives in marine microplastic particles using pyrolysis-GC/MS and scanning electron microscopy. *Environmental Science: Processes & Impacts* **15:**1949-1956.

Fujisawa, T., 2002. Model of the uptake of pesticides by plant. *Journal of Pesticide Science* **27:**279-286.

Gale, P. 2006. The infectivity of transmissible spongiform encephalopathy agent at low doses: the importance of phospholipid. *Journal of Applied Microbiology* **101:**261–274.

Gale, P. 2005. Land application of treated sewage sludge: quantifying pathogen risks from consumption of crops. *Journal of Applied Microbiology* **98:**380–396.

Gale, P. & Stanfield, G. 2001. Towards a quantitative risk assessment for BSE in sewage sludge. *Journal of Applied Microbiology* **91:**563–569.

Galvin, S., Boyle, F., Hickey, P., Vellinga, A., Morris, D. & Cormican, M. 2010. Enumeration and Characterization of Antimicrobial-Resistant Escherichia coli Bacteria in Effluent from Municipal, Hospital, and Secondary Treatment Facility Sources. *Applied and Environmental Microbiology* **76:**4772–4779.

Gaze, W.H., Zhang, L., Abdouslam, N.A., Hawkey, P.M., Calvo-Bado, L., Royle, J., Brown, H., Davis, S., Kay, P., Baxall, A.B. & Wellington, E.M. 2011. Impacts of anthropogenic activity on the ecology of class 1 integrons and integron-associated genes in the environment. *The ISME Journal* **5:**1253–1261.

Gavaghan, P.D., Sykora, J.L., Jakubowski, W., Sorber, C.A., Sninsky, A.M., Lichte, M.D. & Keleti, G. 1993. Inactivation of Giardia by Anaerobic Digestion of Sludge. *Water Science and Technology* **27:**111-114.

Gerba, C.P., Pepper, I.L. & Whitehead, L.F. 3rd. 2002. A risk assessment of emerging pathogens of concern in the land application of biosolids. *Water Science and Technology* **46:**225-230.

Gerba, C.P., Wallis, C. & Melnick, J.L. 1975. Fate of wastewater bacteria and viruses in soil. *Journal of Irrigation and Drainage Engineering – ASCE* **101:**157-174.

Gerhartz, W. (exec ed.). 1985. Ullmann's Encyclopedia of Industrial Chemistry. 5th ed.Vol A1: Deerfield Beach, FL: VCH Publishers, 1985 to Present., p. VA2 322

Gibbs, H. & Jones, V. 2017. CIP2 Sludge Investigations, UKWIR use only.

Gibson, R., Wang, M-J., Padgett, E. & Beck, A.J. 2005. Analysis of 4-nonylphenols, phthalates, and polychlorinated biphenyls in soils and biosolids. *Chemosphere* **61:**1336-1344.

Golet E.M., Xifra I., Siegrist H., Alder A., Giger W. 2003. Environmental Exposure Assessment of Fluoroquinolone Antibacterial Agents from Sewage to Soil. *Environmental Science & Technology* **37:**3243-3249.

Golovko, O., Kumar, V., Fedorova, G., Randak, T. & Grabic, R. 2014. Seasonal changes in antibiotics, antidepressants/psychiatric drugs, antihistamines and lipid regulators in a waste water treatment plant. *Chemosphere* **111:**418-426.

Gomez M.J., Martinez Bueno M.J., Lacorte S., Fernandez-Alba A.R. & Aguera A. 2007. Pilot survey monitoring pharmaceuticals and related compounds in a sewage treatment plant located on the Mediterranean coast. *Chemosphere* **66:**993-1002.

González, M., Martin, J., Santox, J., Aparicio, I. & Alonso, E. 2010. Occurrence and risk assessment of nonylphenol and nonylphenol ethoxylates in sewage sludge from different conventional treatment processes. *Science of the Total Environment* **408:**563-570.

Gostelow, P., Parsons, S. & Stuetz, R. 2001. Odour measurements for the sewage treatment works. *Water Research* **35:**579-597.

Gouin, T., Avalos, J., Brunning, I., Brzuska, K, de Graaf, J., Kaumans, J., Koning, T., Metberg, M., Rettinger, K., Schlatter, H., Thomas, J., van Welie, R. & Wolf, T. 2015. Use of micro-plastic beads in cosmetic products in Europe and their estimated emissions to the North Sea environment. *SOFW-Journal* **141:**40-46.

Grad, F.P. 2002. The preamble of the constitution of the World Health Organization. *Bulletin of the World Health Organization* **80:**982.

Guo, H., Feddes, J., Dehod, W., Laguë, C. & Edeogu, I. 2006. Monitoring of odour occurrence in the vicinity of swine farms by resident-observers – Part II: Impact of weather conditions on odour occurrence. *Canadian Biosystems Engineering* **48:**6.23-6.29.

Gurke, R., Rößler, M., Marx, C., Diamond, S., Schubert, S., Oertel, R. & Fauler, J. 2015. Occurrence and removal of frequently prescribed pharmaceuticals and corresponding metabolites in wastewater of a sewage treatment plant. *Science of the Total Environment* **532:**762-770.

Habib, D., Locke, D.C. & Cannone, L.J. 1998. Synthetic fibres as indicators of municipal sewage sludge, sludge products, and sewage treatment plant effluents. *Water, Air Soil Pollution* **103:**1-8.

Halkjaer-Nielsen, P., Raunkjaer, K. & Hvitved-Jacobsen, T. 1998. Sulphide production and wastewater quality in pressure mains. *Water Science & Technology* **37:**97-104.

Hansch, C., Leo, A. & Hoekman, D. 1995. Exploring QSAR - Hydrophobic, Electronic, and Steric Constants. Washington, DC: American Chemical Society., 1995., p. 154

Hardy, M.L. & Smith, R.L. 1999. In American Chemical Society, Division of Environmental Chemistry, Preprint Extended Abstracts, 217th ACS National Meeting, V39: 191-4 (1999)

Harrison, E.Z. & Oakes, S.R. 2002. Investigation of alleged health incidents associated with land applied sewage sludges. *New Solutions* **12:**387-408.

Harrison, E.Z., Oakes, S.R., Hysell, M. & Hay, A. 2006. Organic chemicals in sewage sludges. *Science of the Total Environment* **367:**481-497.

Harrison, E.Z., McBride, M.B. & Bouldin, D.R. 1999. Land application of sewage sludges: an appraisal of the US regulations. *International Journal of Environment and Pollution* **11:**1-36.

Hayes, J.E., Stevenson, R.J. & Stuetz, R.M. 2014. The impact of malodour on communities: A review of assessment techniques. *Science of the Total Environment* **500-501:**395-407.

Healy, M.G., Fenton, O., Cummins, E., Clarke, R., Peyton, D., Fleming, G., Wall, D., Morrison, L. & Cormican, M. 2017. Health and Water Quality Impacts Arising from Land Spreading of Biosolids. EPA Research Report 200.

Heskett, M., Takada, H., Yamashita, R., Ito, M., Geok, Y.B., Ogata, Y., Kwan, C., Heckhausen, A., Taylor, H., Powell, T., Morishige, C., Young, D., Patterson, H., Robertson, B., Bailey, E. & Mermoz, J. 2012. Measurement of persistent organic pollutants (POPs) in plastic resin pellets from remote island: toward establishment of background concentrations for International Pellet Watch. *Marine Pollution Bulletin* **64:**445-448.

Higgins, C. P., Sharp, J. O., Sepulvado,J. G., Littrell, B., O'Connor, G., Snyder, E. & McAvoy, D. 2010. Trace Organic Chemicals in Biosolids-Amended Soils. State-of-the-Science Review. Water Environment Research Foundation, IWA Publishing. SRSK5T09.

Hinckley, D.A., Bidleman, T.F. & Foreman, W.T. 1990. Determination of vapor pressures for nonpolar and semipolar organic compounds from gas chromatographic retention data. *Journal of Chemical & Engineering Data* **35:**232-237.

Hinckley, G.T., Johnson, C.J., Jacobson, K.H., Bartholomay, C., McMahon, K.D., McKenzie, D., Aiken, J.M. & Pedersen, J.A. 2008. Persistence of Pathogenic Prion

Protein during Simulated Wastewater Treatment Processes. *Environmental Science & Technology* **42:**5254–5259.

Hine, J. & Mookerjee, P.K. 1975. The intrinsic hydrophilic character of organic compounds. Correlations in terms of structural contributions. *Journal of Organic Chemistry* **40:**292-298.

Hirai, H., Takada, H., Ogata, Y., Yamashita, R., Mizukawa, K., Saha, M., Kwan, C., Moore, C., Gray, H., Laursen, D., Zettler, E.R., Farrington, J.W., Reddy, C.M., Peacock, E.E. & Ward, M.W. 2011. Organic micropollutants in marine plastics debris from the open ocean and remote and urban beaches. *Marine Pollution Bulletin* **62:**1683-1692.

Holling, C.S., Bailey, J.L., Heuvel, B.V. & Kinney, C.A. 2012. Uptake of human pharmaceuticals and personal care products by cabbage (Brassica campestris) from fortified and biosolids-amended soils. *Journal of Environmental Monitoring* **11:**3029-3036.

Holmes, L.A., Turner, A. & Thompson, R.C. 2012. Adsorption of trace metals to plastic resin pellets in the marine environment. *Environmental Pollution* **160:**42-48.

Hoogenboom, L.A.P., Bovee, T.H.F., Kloet, D., DeWaal, E., Kleter, G., Van Leeuwen, S.P.J., Pieters, H. & De Boer, J. 2003. Contaminanten in vis- en visproducten. Mogelijke risico's voor de consument en adviezen voor monitoring. RIKILT Rapportnr [in Dutch]

Horton, A. (2017). Microplastics in the Freshwater Environment. A Review of Current Knowledge. Foundation for Water Research FR/R007 http://www.fwr.org/environw/frr0027.pdf

Hosseini Koupaie, E. & Eskicioglu, C. 2015. Health risk assessment of heavy metals through the consumption of food crops fertilized by biosolids: A probabilistic-based analysis. *Journal of Hazardous Materials* **300:**855–865.

Hough, R.L., Booth, P., Avery, L.M., Rhind, S., Crews, C., Bacon, J., Campbell, C.D. & Tompkins, D. 2012. Risk assessment of the use of PAS100 green composts in sheep and cattle production in Scotland. *Waste Management* **32:**117-130

Hough, R.L., Towers, W. & Aalders, I. 2010a. The risk of peat erosion from climate change: land management combinations – an assessment with Bayesian Belief Networks. *Human and Ecological Risk Assessment* **16:**962-976.

Hough, R.L., Fletcher, T., Leonardi, G.S., Goessler, W., Gnagnarella, P., Clemens, F., Gurzau, E., Koppova, K., Rudnai, P., Kumar, R. & Vahter, M. 2010b. Lifetime exposure to arsenic in residential drinking water in Central Europe. *International Archives of Occupational and Environmental Health* **83:**471-481.

Hough, R.L., Breward, N., Yound, S.D., Crout, N.M.J., Tye, A.M., Moir, A.M. & Thornton, I. 2004. Assessing potential risk of heavy metal exposure from consumption of home-produced vegetables by urban populations. *Environmental Health Perspectives* **112:**215-221.

Hough, R.L., Young, S.D. & Crout, N.M.J. 2003. Modelling of Cd, Cu, Ni, Pb and Zn uptake, but winter wheat and forage maize, from a sewage disposal farm. *Soil Use and Management* **19:**19-27.

Howard, P.H., Banerjee, S. & Robillard, K.H. 1985. Measurement of water solubilities, octanol/water partition coefficents and vapor pressures of commercial phthalate etsers. *Environmental Toxicology and Chemistry* **4:**653-661.

Hoyer, H. & Peperle, W. 1958. Dampfdruckmessungen an organischen substanzen und ihre sublimationswärmen. *Berichte der Bunsengesellschaft für Physikalische Chemie* **62:**61-66 [in German]

Hung, H. & Mackay, D. 1997. A novel and simple model of the uptake of organic chemicals by vegetation from air and soil. *Chemosphere* **35:**959-977.

Hussain, N., Jaitley, V. & Florence, A.T. 2001. Recent advances in the understanding of uptake of microparticulates across the gastrointestinal lymphatics. *Advanced Drug Delivery Reviews* **50:**107-142.

Intarakosit, E. 2010. GIS-based odor impact assessment from biosolids land application sites. Civil and environmental engineering. University of Maryland, p.174.

Itokawa, H., Totsuka, N., Nakahara, K., Maezuru, M., Takeya, K., Kondo, M., Inamatsu, M. & Morita, H. 1989. A quantitative structure-activity relationship for antitumor activity of long-chain phenols from *Ginkgo biloba* L. *Chemical and Pharmaceutical Bulletin* **37:**1619-1621.

Inoue, Y., Hashizume, N., Yakata, N., Murakami, H., Suzuki, Y., Kikushima, E. & Otsuka, M. 2012. Unique physicochemical properties of perfluorinated compounds and their bioconcentration in common carp *Cyprinus carpio* L. *Archives of Environmental Contamination and Toxicology* **62:**672-680.

Isidori, M., Lavorgna, M., Nardelli, A., Pascarella, L. & Parrella, A. 2005. Toxic and genotoxic evaluation of six antibiotics on non-target organisms. *Science of the Total Environment* **346:**87-98.

Janssens, D., Wets, G., Brijs, T., Vanhoof, K., Arentze, T. & Timmermans, H. 2006. Integrating Bayesian networks and decision trees in a sequential rule-based transportation model. *European Journal of Operational Research* **175:**16-34.

Jenkins, S.R., Armstrong, C.W. & Monti, M.M. 2007. Health effects of biosolids applied to land: available scientific evidence. NER Committee; 2007.

Jensen FV. 1996. An Introduction to Bayesian Networks. University College London Press, London, UK.

Jensen FV. 2001. Bayesian Networks and Decision Graphs. Springer, New York, USA.

Jensen, J., Ingvertsen, S.T. & Magid, J. 2012. Risk evaluation of five groups of persistent organic contaminants in sewage sludge. Environmental Project No, 1406 2012. Danish Environmental Protection Agency.

Jeong, C-B., Won, E-J., Kang, H-M., Lee, M-C., Hwang, D-S., Hwang, U-K., Zhou, B., Souissi, S., Lee, S-J. & Lee, J-S. 2016. Microplastic size-dependent toxicity, oxidative stress induction, and p-JNK and p-p38 activation in the monogonont rotifer (*Brachionus koreanus*). *Environmental Science & Technology* **50:**8849-8857.

JRC. 2012. Occurrence and Levels of Selected Compounds in European Sewage Sludge Samples – Results of a Pan-European Screening Exercise (FATE SEES). Joint Research Centre. ISBN 978-92-79-27391-9.

Ju, F., Li, B., Ma, L., Wang, Y., Huang, D. & Zhang, T. 2016. Antibiotic resistance genes and human bacterial pathogens: Co-occurrence, removal, and enrichment in municipal sewage sludge digesters. *Water Research* **91:**1–10.

Katz, B.D. & Margolin, A.B. 2007. Inactivation of hepatitis A HM-175/18f, reovirus T1 Lang and MS2 during alkaline stabilization of human biosolids. *Journal of Applied Microbiology* **103:**2225–2233.

Kelessidis, A. & Stasinakis, A.S. 2012. Comparative study of the methods used for treatment and final disposal of sewage sludge in European countries. *Waste Management* **32:**1186–1195.

Kidd, P.S., Domínguez-Rodríguez, M.J., Díez, J. & Monterroso, C. 2007. Bioavailability and plant accumulation of heavy metals and phosphorus in agricultural soils amended by long-term application of sewage sludge. *Chemosphere* **66:**1458–1467.

Kirchmayr, R., Reichl, H.E., Schildorfer, H., Braun, R. & Somerville, R.A. 2006. Prion protein: detection in ‘spiked’ anaerobic sludge and degradation experiments under anaerobic conditions. *Water Science & Technology* **53:**91-98.

Kochetkov, A., Smith, J.S., Ravikerishna, R., Valsaraj, K.T. & Thibodeaux, L.J. 2001. Air-water partition constants for volatile methyl siloxanes. *Environmental Toxicology and Chemistry* **20:**2184-2188.

Koe, L.C.C. 1989. Sewage odors quantification. In Encyclopedia of Environmental Control Technology, Wastewater Treatment Technology, Vol. 3, cd P.N. Cheremisinoff, pp. 423-446. Gulf Publishing Company, Houston, USA.

Koelmans, A.A., Bakir, A., Burton, G.A. & Janssen, C.R. 2016. Microplastic as a vector for chemicals in the aquatic environment: Critical review and model-supported reinterpretation of empirical studies. *Environmental Science & Technology* **50:**3315-3326.

Kolarova, D. 1999. Environmental risk perception, health concerns and attributes for change. 9th annual conference, Risk Analysis: facing the new millennium. Rotterdam; the Netherlands: Delft University Press, 1999.

Konradi, S. & Vogel, I. 2013. Fate of pharmaceuticals (PhCs) in sewage sludge and proposal of indicator substances for monitoring. https://www.umweltbundesamt.de/sites/default/files/medien/376/dokumente/konradi-presentation.pdf

Köster, E.P. 2002. The specific characteristics of the sense of smell. In: Rouby C., et al., eds. “Olfaction, taste and cognition”. London: Cambridge University Press; p.27-45.

Kotowska, U., Żalikowski, M. & Isidorov, V.A. 2012. HS-SPME/GC-MS analysis of volatile and semi-volatile organic compounds emitted from municipal sewage sludge. *Environmental Monitoring and Assessment* **184:**2893-2907.

Latare, A.M., Kumar, O., Singh, S.K. & Gupta, A. 2014. Direct and residual effect of sewage sludge on yield, heavy metals content and soil fertility under rice-wheat system. *Ecological Engineering* **69:**17-24.

Lechner, A. & Ramler, D. 2015. The discharge of certain amounts of industrial microplastic from a production plant into the River Danube is permitted by the Austrian legislation. *Environmental Pollution* **200:**159-160.

Lee, H., Shim, W.J. & Kwon, J.H. 2014. Sorption capacity of plastic debris for hydrophobic organic chemicals. *Science of the Total Environment* **470-471:**1545-1552.

Lee, K.W., Shim, W.J., Kwon, O.Y. & Kang, J.H. 2013. Size-dependent effects of micro polystyrene particles in the marine copepod *Tigriopus japonicas*. *Environmental Science & Technology* **47:**11278-11283.

Lei, Y.D., Wania, F. & Mathers, D. 2010. Temperature-dependent capor pressure of selected cyclic and linear polydimethylsiloxane oligomers. *Journal of Chemical & Engineering Data* **55:**5868-5873.

Leslie, H.A., Brandsma, S.H., van Velzen, M.J.M. & Vethaak, A.D. 2017. Microplastics en route: Field measurements in the Dutch river delta and Amsterdam canals, wastewater treatment plants, North Sea sediments and biota. *Environment International* **101:**133-142.

Lind, P.M., Gustafsson, M., Hermsen, S.A.B., Larsson, S., Kyle, C.E., Örberg, J. & Rhind, S.M. 2009. Exposure to pastures fertilised with sewage sludge disrupts bone tissue homeostasis in sheep. *Science of the Total Environment* **407:**2200-2208.

Lorber, M. & Cleverly, D. 2010. In: An Exposure Assessment of Polybrominated Diphenyl Ethers. EPA/600/R-08/086F. National Center for Environmental Assessment (NCEA), Office of Research and Development. Washington, DC (2010). Available from, as of Mar 24, 2015: https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=210404

Luginaah, I., Taylor, S., Elliott, S. & Eyles, J. 2002. Community reappraisal of the perceived health effects of a petroleum refinery. *Social Science and Medicine* **55:**47-61.

Luo, Y., Guo, W. & Ngo, H.H. 2014. A review on the occurrence of micropollutants in the aquatic environment and their fate and removal during wastewater treatment. *Science of the Total Environment* **473:**619–41.

Lusher, A.L., Burke, A., O'Connor, I. & Officer, R. 2014. Microplastic pollution in the Northeast Atlantic Ocean: Validated and opportunistic sampling. *Marine Pollution Bulletin* **88:**325-333.

Mackay, D. 2001. Multimedia Environmental Models: The Fugacity Approach. 2nd Edition. Lewis Publishers, London.

Magnusson, K. & Norén, F. 2014. Screening of microplastic particles in and downstream from a wastewater treatment plant. In Report to the Swedish Environmental Research Institute: C55

Mahon, A.M., O'Connell, B., Healy, M.G., O'Connor, I., Officer, R., Nash, R. & Morrison, L. 2017. Microplastics in sewage effluent: Effects of treatment. *Environmental Science & Technology* **51:**810-818.

Marcot, B.G., Steventon, J.D., Sutherland, G.D. & McCann, R.K. 2006. Guidelines for developing and updating Bayesian belief networks applied to ecological modelling and conservation. *Canadian Journal of Forest Research* **36:**3063-3074.

Marklund, A., Andersson, B. & Haglund, P. 2005. Organophosphorus flame retardants and plasticizers in Swedish sewage treatment plants. *Environmental Science & Technology* **39:**7423-7429.

Matos, J.S. & Sousa, E.R. 1992. The forecasting of hydrogen sulphide gas build-up in sewerage collection systems. *Water Science & Technology* **26:**915-922.

May, W.E., Wasik, S.P., Miller, M.M., Tewarl, Y.B., Brown-Thomas, J.M. & Goldberg, R.N. 1983. Solution thermodynamics of some slightly soluble hydrocarbons in water. *Journal of Chemical & Engineering Data* **28:**197-200.

May, W.E., Wasik. S.P. & Freeman, D.H. 1978. Determination of the solubility behaviour of some polycyclic aromatic hydrocarbons in water. *Analytical Chemistry* **50:**997-1000.

McBride, M., Richards, B.K., Steenhuis, T. & Spiers, G. 1999. Long-term leaching of trace elements in a heavily sludge-amended silty clay loam soil. *Soil Science* **164:**613-623.

McBride, M.B., Richards, B.K. & Steenhuis, T. 2004. Bioavailability and crop uptake of trace elements in soil columns amended with sewage sludge products. *Plant and Soil* **262:**71–84.

McCarthy, L.H., Loyo-Rosales, J.E., Raby, M., Payne, M., Huber, A., Lavery, J., Caputa, K., Taylor, L. & Falls, A. 2015. Risks associated with application of municipal biosolids to agricultural lands in a Canadian context. Literature Review prepared for Canadian Municipal Water Consortium. http://www.cwn-rce.ca/assets/resources/pdf/McCarthy-Risks-Biosolids-2015.pdf [accessed 01/03/18].

McCormick, A., Hoellein, T.J., Mason, S.A., Schluep, J. & Kelly, J.J. 2014. Microplastic is an abundant and distinct microbial habitat in an urban river. *Environmental Science & Technology* **48:**11863-11871.

McFarland, J.W., Avdeef, A., Berger, C.M. & Raevsky, O.A. 2001. Estimating the water solubilities of crystalline compounds from their chemical structures alone. *Journal of Chemical Information and Computer Sciences* **41:**1355-1359.

McFarland, J.W., Berger, C.M., Froshauer, S.A., Hayashi, S.F., Hecker, S.J., Jaynes, B.H., Jefson, M.R., Kamicker, B.J., Lipinski, C.A., Lundy, K.M., Reese, C.P. & Vu, C.B. 1997. Quantitative structure-activity relationships among macrolide antibacterial agents: In vitro and in vitro potency against *Pasteurella multocida*. *Journal of Medicinal Chemistry* **40:**1340-1346.

McGrath, S.P., Chang, A.C., Page, A.L. & Witter, E. 1994. Land application of sewage sludge: scientific perspectives of heavy metal loading limits in Europe and the United States. *Environmental Reviews* **2:**108–118.

McIntyre, A., Vincent, R.M., Perkins, A.C. & Spiller, R.C. 1997. Effect of bran, ispaghula, and inert plastic particles on gastric emptying and small bowel transit in humans: the role of physical factors. *Gut* **40:**223-227.

McLaughlin, H.P., Casey, P.G., Cotter, J., Gahan, C.G. & Hill, C. 2011. Factors affecting survival of Listeria monocytogenes and Listeria innocua in soil samples. *Archives of Microbiology* **193:**775–785.

Michielssen, M.R., Michielssen, E.R., Ni, J. & Duhaime, M.B. 2016. Fate of microplastics and other small anthropogenic litter (SAL) in wastewater treatment

plants depends on unit processes employed. *Environmental Science: Water Research & Technology* **2:**1064-1073.

Miles, S.L., Takizawa, K., Gerba, C.P. & Pepper, I.L. 2011. Survival of infectious prions in Class B biosolids. *Journal of Environmental Science and Health, Part A* **46:**364–370.

Miller, M.M., Wasik, S.P., Huang, G-L., Shiu, W-Y. & MacKay, D. 1985. Relationships between octanol-water partition coefficients and aqueous solubility. *Environmental Science & Technology* **19:**522-529.

Mamindy-Pajany, Y., Sayen, S., Mosselmans, F.W. & Guillon, E. 2014. Copper, Nickel and Zinc Speciation in a Biosolid-Amended Soil: pH Adsorption Edge, μ-XRF and μ-XANES Investigations. *Environmental Science & Technology* **48:**7237–7244.

Miyata, H., Takayama, K., Mimura, M., Kashimoto, T. & Fukushima, S. 1989. Specific congener profiles of polychlorinated dibenzo-p-dioxins and dibenzofurans in blue mussel in Osaka Bay in Japan: Aqueous solubilities of PCDDs and PCDFs. *Bulletin of Environmental Contamination and Toxicology* **43:**342-349.

Moffatt, S. & Pless-Mulloli, T. 2003. “It wasn’t the plague we expected.” Parents’ perceptions of the health and environmental impact of opencast coal mining. *Social Science and Medicine* **57:**437-451.

Munir, M., Wong, K. & Xagoraraki, I. 2011. Release of antibiotic resistant bacteria and genes in the effluent and biosolids of five wastewater utilities in Michigan. *Water Research* **45:**681–693.

Munir, M. & Xagoraraki, I. 2011. Levels of Antibiotic Resistance Genes in Manure, Biosolids, and Fertilized Soil. *Journal of Environment Quality* **40:**248-255.

Murphy, F., Ewins, C., Carbonnier, F. & Quinn, B. 2016. Wastewater treatment works (WwTW) as a source of microplastics in the aquatic environment. *Environmental Science & Technology* **50:**5800-5808.

Murray, J.J., Pottie, R.F. & Pupp, C. 1974. The vapor pressures and enthalpies of sublimation of five polycylic aromatic hydrocarbons. *Canadian Journal of Chemistry* **52:**557-563.

Nakashima, E., Isobe, A., Kako, S., Itai, T. & Takahashi, S. 2012. Quantification of toxic metals derived from macroplastic litter on Ookushi Beach, Japan. *Environmental Science & Technology* **46:**10099-10105.

Nallani, G.C., Paulos, P.M., Constantine, L.A., Venables, B.J. & Huggett, D.B. 2011. Bioconcentration of ibuprofen in fathead minnow (*Pimephales promelas*) and channel catfish (*Ictalurus punctatus*). *Chemosphere* **84:**1371-1377.

Nestrick, T.J., Lamparski, L.L. & Townsend, D.I. 1980. Identification of tetrachlorodibenzo-p-dioxin isomers at the 1-ng level by photolytic degradation and pattern recognition techniques. *Analytical Chemistry* **52:**1865-1874.

Neutra, R., Lipscomb, J., Satin, K. & Shusterman, D. 1991. Hypotheses to explain the higher symptom rates observed around hazardous waste sites. *Environmental Health Perspectives* **94:**31-38.

Nicell, J.A. 2009. Assessment and regulation of odour impacts. *Atmospheric Environment* **43:**196-206.

NITE. 2012. Chemical Risk Information Platform (CHRIP). Biodegradation and Bioconcentration. Tokyo, Japan: Natl Inst Tech Eval. Available from, as of Mar 27, 2012: http://www.safe.nite.go.jp/english/db.html

Nowara, A., Burhenne, J. & Spiteller, M. 1997. Binding of fluoroquinolone carboxylic acid derivatives to clay minerals. *Journal of Agricultural and Food Chemistry* **45:**1459-1463.

National Research Council (NRC). 2002. Biosolids Applied to Land: Advancing Standards and Practices. The National Academies Press, Washington, D.C.

O'Connor, A. 2012. An overview of the role of bread in the UK diet. *Nutrition Bulletin* **37:**193-212.

Ogata, Y., Takada, H., Mizukawa, K., Hirai, H., Iwasa, S., Endo, S., Mato, Y., Saha, M., Okuda, K., Nakashima, A., Murakami, M., Zurcher, N., Booyatumanondo, R., Zakaria, M.P., Dung, L.Q., Gordon, M., Miguez, C., Suzuki, S., Moore, C., Karapanagioti, H.K., Werts, S., McClurg, T., Burres, E., Smith, W., Van Velkenburg, M., Lang, J.S., Lang, R.C., Laursen, D., Danner, B., Stewardson, N. & Thompson, R.C. 2009. International Pellet Watch: global monitoring of persistent organic pollutants (POPs) in coastal water. 1. Initial phase on PCB, DDT and HCHs. *Marine Pollution Bulletin* **58:**1437-1446.

Okuda, T., Kobayashi, Y., Nagao, R., Yamashita, N., Tanaka, H., Tanaka, S., Fujii, S., Konishi, C. & Houwa, I. 2008. Removal efficiency of 66 pharmaceuticals during wastewater treatment process in Japan. *Water Science & Technology* **57:**65-71.

O'Neil, M.J. (ed.). 2006. The Merck Index - An Encyclopedia of Chemicals, Drugs, and Biologicals. Whitehouse Station, NJ: Merck and Co., Inc., 2006., p. 690

Pasquill, F. 1961. The estimation of the dispersion of windborne material. *Meteorology Magazine* **90(1063):**33-49.

Passuello, A., Mari, M., Nadal, M., Schuhmacher, M., Domingo, J.L., 2010. POP accumulation in the food chain: Integrated risk model for sewage sludge application in agricultural soils. *Environment International* **36:**577-583.

Paterson, S., Mackay, D., Mcfarlane, C., 1994. A Model of Organic-Chemical Uptake by Plants from Soil and the Atmosphere. *Environmental Science & Technology* **28:**2259-2266.

Paul, C., Rhind, S.M., Kyle, C.E., Scott, H., McKinnell, C. & Sharpe, R.M. 2005. Cellular and hormonal distruption of fetal testis development in sheep. *Environmental Health Perspectives* **113:**1580-1587.

Payne, J., Scholze, M., Kortenkamp, A. 2001 Mixtures of four organochlorines enhance human breast cancer cell proliferation. *Environmental Health Perspectives* **109:**391-397.

Pearl J. 1988. Probabilistic Reasoning in Intelligent Systems: Networks of Plausible Inference. Morgan Kaufmann Publishers, San Francisco, California, USA.

Pearlman, R.S., Yalkowsky, S.H. & Banerjee, S. 1984. Water solubilities of polynuclear aromatic and heteroaromatic compounds. *Journal of Physical and Chemical Reference Data* **13:**555-562.

Pepper, I.L., Brooks, J.P. & Gerba, C.P. 2006. Pathogens in Biosolids. *Advances in Agronomy* **90:**1–41.

Pollard, S.J.T., Hickman, G.A.W., Irving, P., Hough, R.L., Gauntlett, D.M., Howson, S.F., Hart, A., Gayford, P. & Gent, N. 2008. Exposure assessment of carcass disposal options in the event of a notifiable exotic animal disease: application to avian influenza virus. *Environmental Science & Technology* **42:**3145-3154.

Pomeroy, R.D. 1959. Generation and control of sulphides in filled pipes. *Sewage and Industrial Wastes* **31:**1082-1095.

Pomeroy, R.D. & Parkhurst, J.D. 1977. The forecasting of sulphide build up rates in sewers. *Progress in Water Technology* **9:**621.

Prado, T., Pereira, W.C., Silva, D.M., Seki, L.M., Carvalho, A.P. & Asensi, M.D. 2008. Detection of extended-spectrum β-lactamase-producing Klebsiella pneumoniae in effluents and sludge of a hospital sewage treatment plant. *Letters in Applied Microbiology* **46:**136-141.

Pritchard, D.L., Penney, N., McLaughlin, M.J., Rigby, H. & Schwartz, K. 2010. Land application of sewage sludge (biosolids) in Australia: risks to the environment and food crops. *Water Science & Technology* **62:**48-57.

Prosser, R.S. & Sibley, P.K. 2015. Human health risk assessment of pharmaceuticals and personal care products in plant tissue due to biosolids and manure amendments, and wastewater irrigation. *Environment International* **75:**223–233.

Pruden, A. 2014. Balancing Water Sustainability and Public Health Goals in the Face of Growing Concerns about Antibiotic Resistance. *Environmental Science & Technology* **48:**5–14.

Radjenović, J., Petrović, M. & Barceló, D. 2009. Fate and distribution of phamaceuticals in wastewater and sewage sludge of the conventional activated sludge (CAS) and advanced membrane bioreactor (MBR) treatment. *Water Research* **43:**831-841.

Rajapakse, N., Silva, E. & Kortenkamp, A. 2002. Combining xeno-estrogens at levels below individual no-observed-effect concentrations dramatically enhances steroid hormone action. *Environmental Health Perspectives* **110:**917-921.

Rappert, S. & Muller, R. 2005. Odor compounds in waste gas emissions from agricultural operations and food industries. *Waste Management* **25:**887-907.

Rein, A., Legind, C.N., Trapp, S., 2011. New concepts for dynamic plant uptake models. *Sar and Qsar in Environmental Research* **22:**191-215.

Remy, F., Collard, F., Gilbert, B., Compère, P., Eppe, G. & Lepoint, G. 2015. When microplastic is not plastic: the ingestion of artificial cellulose fibres by macrofauna living in seagrass macrophytodetritis. *Environmental Science & Technology* **49:**11158-11166.

Richards, S.M. & Cole, S.E. 2006. A toxicity and hazard assessment of fourteen pharmaceuticals to *Xenopus laevis* larvae. *Ecotoxicology* **15:**647-656.

Richardson, B.J., Lam, P.K.S. & Martin, M. 2005. Emerging chemicals of concern: Pharmaceuticals and personal care products (PPCPs) in Asia, with particular reference to Southern China. *Marine Pollution Bulletin* **50:**913–920.

Rios, L.M., Moore, C. & Jones, P.R. 2007. Persistent organic pollutants carried by synthetic polymers in the ocean environment. *Marine Pollution Bulletin* **54:**1230-1237.

Rizzo, L., Manaia, C., Merlin, C., Schwartz, T., Dagot, C., Ploy, M.C., Michael, I. & Fatta-Kassinos, D. 2013. Urban wastewater treatment plants as hotspots for antibiotic resistant bacteria and genes spread into the environment: A review. *Science of The Total Environment* **447:**345–360.

Robertson, L.J., Campbell, A.T. & Smith, H.V. 1992. Survival of Cryptosporidium parvum oocysts under various environmental pressures. *Applied and Environmental Microbiology* **58:**3494–500.

Rochman, C.M., Hentschel, B.T. & The, S.J. 2014. Long-term sorption of metals is similar among plastic types: Implications for plastic debris in aquatic environments. *PLoS One* **9:** e85433.

Rordorf, B.F. 1987. Prediction of vapour pressures, boiling points and enthalpies of fusion for twenty-nine halogenated dibenzo-p-dioxins. *Thermochimica Acta* **112:**117-122.

Rutjes, S.A., Lodder, W.J., Lodder-Verschoor, F., van den Berg, H.H.J.L., Vennema, H., Duizer, E., Koopmans, M. & de Roda Husman, A. 2009. Sources of Hepatitis E Virus Genotype 3 in the Netherlands. *Emerging Infectious Diseases* **15:**381–387.

Sangster J. 2005. LOGKOW Database. A databank of evaluated octanol-water partition coefficients (Log P). Available from, as of Dec. 27, 2005: re3data.org/search

Schets, F.M., de Heer, L. & de Rosa Husman, A.M. 2013. Coxiella burnetii in sewage water at sewage water treatment plants in a Q fever epidemic area. *International Journal of Hygiene and Environmental Health* **216:**698–702.

Schiffman, S.S. & Williams, K. 2005. Science of odor as a potential health issue. *Journal of Environmental Quality* **34:**129-138.

Schiffman, S.S., Walker, J.M., Dalton, P., Lorig, T.S., Raymer, J.H., Shusterman, D. & Williams, C.M. 2000. Potential health effects of odor from animal operations, wastewater treatment, and recycling of byproducts. *Journal of Agromedicine* **7:**7-78.

Schoof, R.A. & Houkal, D. 2005. The evolving science of chemical risk assessment for land-applied biosolids. *Journal of environmental quality* **34:**114–21.

Science for Environment Policy (2017) *Persistent organic pollutants: towards a POPs-free future*; Future Brief 19. Brief produced for the European Commission DG Environment. Bristol: Science Communication Unit, UWE.

Shargil, D., Gerstl, Z, Fine, P., Nitsan, I. & Kurtzman, D. 2015. Impact of biosolids and wastewater effluent application to agricultural land on steroidal hormone content in lettuce plants. *Science of the Total Environment* **505:**357-366.

Shiu, W.Y., Doucette, W., Gobas, F.A.P.C., Andren, A. & MacKay, D. 1988. Physical-chemical properties of chlorinated dibenzo-*p*-dioxins. *Environmental Science & Technology* **22:**651-658.

Shopp, G.M., White, K.L., Holsapple, M.P., Barnes, D.W., Duke, S.S., Anderson, A.C., Condie, L.W., Hayes, J.R. & Borzelleca, J.F. 1984. Naphthalene toxicity in CD-1 mice: general toxicity and immunotoxicology. *Fundamental and Applied Toxicology* **4:**406-419.

Shusterman, D. 2001. Odor-associated health complaints: Competing explanatory models. *Chemical Senses* **26:**338-343.

Shusterman, D. 1999. The health significance of environmental odour pollution: revisited. *Journal of Environmental Medicine* **1:**249-258.

Sidhu, J.P.S. & Toze, S.G. 2009. Human pathogens and their indicators in biosolids: A literature review. *Environment International* **35:**187–201.

Sims, R.C. & Overcash, M.R. 1983. Fate of polycyclic aromatic compounds (PNAs) in soil-plant systems. *Research Reviews* **88:**1-68.

Smith, S.R. 2009. Organic contaminants in sewage sludge (biosolids) and their significance for agricultural recycling. *Philosophical Transactions of The Royal Society* **367:**4005-4041.

SNIFFER. 2008. Human health and the environmental impacts of using sewage sludge on forestry and for restoration of derelict land. UKLQ09 August 2008.

Sonnefeld, W.J., Zoller, W.H. & May, W.E. 1983. Dynamic coupled-column liquid chromatographic determination of ambient temperature vapour pressures of polynuclear aromatic hydrocarbons. *Analytical Chemistry* **55:**275-280.

SRUC. 2013. Optimising the application of bulky organic fertilisers. Technical Note TN650. April 2013.

Stasinakis A. S., Gatidou G., Mamais D., Thomaidi N. S., Lekkas T. D. 2008. Occurrence and fate of endocrine disrupters in Greek sewage treatment plants. *Water Research* **42:**1796-1804.

Stewart, M., Northcott, G., Gaw, S & Tremblay, L.A. 2016. An update on emerging organic contaminants of concern for New Zealand with guidance on monitoring approaches for councils. Prepared by Streamlined Environmental Ltd, Northcott Research Consultants Ltd, University of Canterbury, Cawthron Institute and the University of Auckland for Auckland Council, Greater Wellington Regional Council and Environment Canterbury Regional Council. Auckland Council technical report, TR2016/006

Straub, T.M., Pepper, I.L. & Gerba, C.P. 1993. Hazards from pathogenic microorganisms in land-disposed sewage sludge. *Reviews of environmental contamination and toxicology* **132:**55–91.

Suk, W.A., Olden, K. & Yang, R.S.H. 2002. Chemical mixtures research: significance and future perspectives. *Environmental Health Perspectives* **110(S6):**891-892.

Sucker, K., Both, R., Bischoff, M., Guski, R. & Winneke, G. 2008a. Odor frequency and odor annoyance. Part I: assessment of frequency, intensity and hedonic tone of environmental odors in the field. *International Archives of Occupational and Environmental Health* **81:**671-682.

Sucker, K., Both, R., Bischoff, M., Guski, R., Kramer, U. & Winneke, G. 2008b. Odor frequency and odor annoyance Part II: dose-response associations and their modification by hedonic tone. *International Archives of Occupational and Environmental Health* **81:**683-694.

Takács-Novák, K., Józan, M., Hermecz, I. & Szász, G. 1992. Lipophilicity of antibacterial fluoroquinolones. *International Journal of Pharmaceutics* **79:**89-96.

Talvitie J, Heinonen M, Pääkkönen J-P, Vahtera E, Mikola A, Setälä O. & Vahala R. 2015. Do wastewater treatment plants act as a potential point source of microplastics? Preliminary study in the coastal Gulf of Finland, Baltic Sea. *Water Science & Technology* **72**:1495–1504.

Taylor, D.M. 2000. Inactivation of transmissible degenerative encephalopathy agents: a review. *The Veterinary Journal* **159:**10-17.

Teuten, E.L., Saquing, J.M., Knappe, D.R.U., Barlaz, M.A., Jonsson, S., Björn, A., Rowland, S.J., Thompson, R.C., Galloway, T.S., Yamashita, R., Ochi, D., Watanuki, Y., Moore, C., Viet, P.H., Tana, T.S., Prudente, M., Boonyatumanond, R., Zakaria, M.P., Akkhavong, K., Ogata, Y., Hirai, H., Iwsa, S., Mizukawa, K., Hagino, Y., Imamura, A., Saha, M. & Takada, H. 2009. *Philosophical Transactions of the Royal Society B* **364:**2027-2045.

Tezel, U. & Pavlostathis, S.G. 2011. Role of Quaternary Ammonium Compounds on Antimicrobial Resistance in the Environment. Antimicrobial Resistance in the Environment. First Edition. Edited by Patricia L. Keen and Mark H. M. M. Montforts. John Wiley & Sons, Inc., pp. 349–387.

Thomaidi, V.S., Stasinakis, A.S., Borova, V.L. & Thomaidis, N.S. 2016. Assessing the risk associated with the presence of emerging organic contaminants in sludge-amended soil: A country-level analysis. *Science of the Total Environment* **548-549:**280-288.

Thompson, R.C., Olsen, Y., Mitchess, R.P., Davis, A., Rowland, J.S., John, A.W.G., McGonigle, D. & Russell, A.E. 2004. Lost at sea: where is all the plastic? *Science* **304:**838.

Thomson P.D.R. & Montvale, N.J. 2007. Physicians Desk Reference 61st ed., p. 3314

Tindall, H. & Ashby, J. 2004. Sensitivity of the immature rat uterotrophic assay to mixtures of estrogens. *Environmental Health Perspectives* **112:**575-582.

Trapp, S., 2004. Plant uptake and transport models for neutral and ionic chemicals. *Environmental Science and Pollution Research* **11:**33-39.

Trapp, S., Matthies, M., 1995. Generic One-Compartment Model for Uptake of Organic-Chemicals by Foliar Vegetation. *Environmental Science & Technology* **29:**2333-2338.

Trapp, S., Legind, C.N., 2011. Uptake of organic contaminants from soil into vegetables and fruits. In: Swartjes, F.A. (Eds.), Dealing with contaminated sites - From theory towards practical application. Springer, pp. 369-408.

Troldborg, M., Aalders, I., Towers, W., Hallett, P.D., McKenzie, B.M., Bengough, A.G., Lilly, A., Ball, B.C. & Hough, R.L. 2013. Application of Bayesian Belief Networks to quantify and map areas at risk to soil threats: Using soil compaction as an example. *Soil and Tillage Research* **132:**56-68.

Tryphonas, H., Hayward, S., O'Grady, L., Loo, J.C.K., Arnold, D.L., Bryce, F. & Zawidzka, Z.Z. 1989. Immunotoxicity studies of PCB (Aroclor 1254) in the adult Rhesus (*Macaca mulatta*) monkey – preliminary report. *International Journal of Immunopharmacology* **11:**199-206.

Tryphonas, H., Luster, M.I., Schiffman, G., Dawson, L.L., Hodgen, M., Germolec, D., Hayward, S., Bryce, F., Loo, J.C.K., Mandy, F. & Arnold, D.L. 1991a. Effect of chronic exposure of PCB (Arochlor 1254) on specific and nonspecific immune parameters in the rhesus (*Macaca mulatta*) monkey. *Fundamental and Applied Toxicology* **16:**773-786.

Tryphonas, H., Luster, M.I., White, K.L., Naylor, P.H., Erdos, M.R., Burleson, G.R., Germolec, D., Hodgen, M., Mayward, S. & Arnold, D.L. 1991b., Effects of PCB (Aroclor 1254) on non-specific immune parameters in Rhesus (*Macaca mulatta*) monkeys. *International Journal of Immunopharmacology* **13:**639-648.

Umlauf, G., Christoph, E.H., Lanzini, L., Savolainen, R., Skejo, H., Bidoglio, G., Clemens, J., Goldbach, H. & Scherer, H. 2011. PCDD/F and dioxin-like PCB profiles in soils amended with sewage sludge, compost, farmyard manure, and mineral fertilizer since 1962. *Environmental Science and Pollution Research International* **18:**461-470.

UKWIR 2012. Investigation of Organic Compounds in Sludge – Persistent Organic Pollutants; Steroid Oestrogens; and selected Pharmaceuticals. United Kingdom Water Industry Research 2012.

USEPA. 2012. Estimation Program Interface (EPI) Suite. Ver. 4.11. Nov, 2012. Available from, as of Nov 2, 2016: http://www2.epa.gov/tsca-screening-tools

US EPA. 2009a. Estimation Program Interface (EPI) Suite. Ver. 4.0. Jan, 2009. Available from, as of Jan 4, 2010: http://www.epa.gov/oppt/exposure/pubs/episuitedl.htm

USEPA 2009b. *Targeted National Sewage Sludge Survey – Sampling and Analysis Technical Report*. United States Environmental Protection Agency January.

USEPA. 2004. Estimation Program Interface (EPI) Suite. Ver.3.12. Nov 30, 2004. Available from, as of Jun 7, 2007: http://www.epa.gov/oppt/exposure/pubs/episuitedl.htm

USEPA. 1996. Guidelines for Toxicity Risk Assessment. EPA/630/R/008. 1996.

USEPA. 1993. Standards for the Use or Disposal of Sewage Sludge." Title 40, Code of Federal Regulations, Part 503.

USEPA. 1987. Health and Environmental Effects Profile for Benzo(k)fluoranthene. ECAO-CIN-P229. (1987) Cincinnati, OH: USEPA, Environ Criteria Assess Off. Available from, as of Aug 25, 2016: http://www.epa.gov

van den Berg, H., Lodder, W., van der Poel, W., Vennema, H & de Rosa Husman, A.M. 2005. Genetic diversity of noroviruses in raw and treated sewage water. *Research in Microbiology* **156:**532–540.

van der Hoek, W., Hunink, J., Vellema, P. & Droogers, P. 2011. Q fever in The Netherlands: the role of local environmental conditions. *International Journal of Environmental Health Research* **21:**441–451.

van Elsas, J.D., Chiurazzi, M., Mallon, C.A., Elhottova, D., Kristufek, V. & Salles, J.F. 2012. Microbial diversity determines the invasion of soil by a bacterial pathogen. *Proceedings of the National Academy of Sciences* **109:**1159–1164.

Varaprath, S., Frye, C.L. & Hamelink, J. 1996. Aqueous solubility of permethylsiloxanes (silicones). *Environmental Toxicology and Chemistry* **15:**1263-1265.

Verschoor AJ, de Porter L. & Roex, E. 2014. Quick Scan and Prioritization of Microplastic Sources and Emissions, RIVM Advisory Letter 250012001, National Institute for Public Health and Environment, Bilthoven, The Netherlands. re3data.org/search

Viau, E., Bibby, K., Paez-Rubio, T. & Peccia, J. 2011. Toward a Consensus View on the Infectious Risks Associated with Land Application of Sewage Sludge. *Environmental Science & Technology* **45:**5459–5469.

Vieno N. M., Tuhkanen T. & Kronberg L. 2006. Analysis of neutral and basic pharmaceuticals in sewage treatment plants and in recipient rivers using solid phase extraction and liquid chromatography–tandem mass spectrometry detection. *Journal of Chromatography A* **1134:**101-111.

Vincent, A. & Hobson, J. 1998. Odour control. CIWEM Monographs on Best Practics No. 2, Chartered Institute of Water and Environmental Management, Terence Dalton Publishing, London, UK.

Von Moos, N., Burkhardt-Holm, P. & Köhler, A. 2012. Uptake and effects of microplastics on cells and tissue of the Blue Mussel Mytilus edulis L. after an experimental exposure. *Environmental Science & Technology* **46:**11327-11335.

Walters, E., McClellan, K., Halden, R.U. 2010. Occurrence and loss over three years of 72 pharmaceuticals and personal care products from biosolids-soil mixtures in outdoor mesocosms. *Water Research* **44:**6011-6020.

Wang, L., Wang, X., Xu, O. & Tian, L. 1986. Determination of the *n*-octanol/water partition coefficients of polycyclic aromatic hydrocarbons by HPLC and estimation of their aqueous solubilities. *Huanjing Kexue Xuebao* **6:**491-497.

Watts, A.J.R., Urbina, M.A., Goodhead, R.M., Moger, J., Lewis, C. & Galloway, T.S. 2016. Effect of microplastic on the gills of the Shore Crab Carcinus maenas. *Environmental Science & Technology* **50:**5364-5369.

WEAO. 2010. Assessing the Fate and Significance of Microconstituents and Pathogens in Sewage Biosolids. Update of the 2001 WEAO Report on Fate and Significance. Water Environment Association of Ontario.

WEAO. 2001. Fate and Significance of Selected Contaminants in Sewage Biosolids Applied to Agricultural Land Through Literature Review and Consultation with Stakeholder Groups. Water Environment Association of Ontario.

Wegner, A., Besseling, E., Foekema, E., Kamermans, P. & Koelmans, A.A. 2012. Effects of nanopolystyrene on the feeding behaviour of the blue mussel (*Mytilus edulis* L.). *Environmental Toxicology and Chemistry* **31:**2490-2497.

Wei, J., Jin, Y., Sims, T. & Kniel, K.E. 2009. Survival of Human Adenovirus 41 in Land-Applied Manure and Biosolids. *Food and Environmental Virology* **1:**148–154.

Wei, J., Jin, Y., Sims, T. & Kniel, K.E. 2010. Survival of Murine Norovirus and Hepatitis A Virus in Different Types of Manure and Biosolids. *Foodborne Pathogens and Disease* **7:**901–906.

Wellington, E.M., Boxall, A.B., Cross, P., Gaze, W.H., Hawkwy, P.M., Johnson-Rollings, A.S., Jones, D.L., Lee, N.M., Otten, W., Thomas, C.M. & Williams, A.P. 2013. The role of the natural environment in the emergence of antibiotic resistance in Gram-negative bacteria. *The Lancet Infectious Diseases* **13:**155–165.

WHO 2012. *Pharmaceuticals in Drinking-Water*. Geneva: World Health Organisation.

Winneke, G. 2004a. The assessment of the impact of environmental odours in the community. *VDI Bericht* **1850:**5-7.

Winneke, G. 2004b. Population odour annoyance in influenced by the hedonic quality of industrial odours. *VDI Bericht* **1850:**9-12.

WRAP. 2016a. Risk assessment for the use of source-segregated composts in UK agriculture. Report OAV024-004. Waste and Resources Action Programme, Banbury, UK; available on request from risk.assessments@wrap.org.uk

WRAP. 2016b. Risk-based guidance for the use of source-segregated anaerobic digestates in GB agriculture. Report OAV036-008. Waste and Resources Action Programme, Banbury, UK; available on request from risk.assessments@wrap.org.uk

WRAP. 2016c. Risk assessment for the use of PAS100 green composts in Scottish livestock production. Report OAV021-004. Waste and Resources Action Programme, Banbury, UK; available on request from risk.assessments@wrap.org.uk

Wright, S.L., Thompson, R.C. & Galloway, T.S. 2013. The physical impacts of microplastics on marine organisms: a review. *Environmental Pollution* **178:**483-492.

Writer, J.H., Ferrer, I., Barber, L.B. & Thurman, E.M. 2013. Widespread occurrence of neuro-active pharmaceuticals and metabolites in 24 Minnesota rivers and wastewaters. *Science of the Total Environment* **461-462:**519-527.

Wu, C., Spongberg, A.L., Witter, J.D., Fang, M. and Czajkowski, K.P. 2010. Uptake of Pharmaceutical and Personal Care Products by Soybean Plants from Soils Applied with Biosolids and Irrigated with Contaminated Water. *Environmental Science and Technology* **44:**6157-6161.

Xu, S., Kozerski, G. & MacKay, D. 2014. Critical review and interpretation of environmental data for volatile methylsiloxanes: Partition properties. *Environmental Science & Technology* **48:**11748-11759.

Yalkowsky, S.H., He, Yan, & Jain, P. 2010. Handbook of Aqueous Solubility Data Second Edition. CRC Press, Boca Raton, FL 2010

Yalkowsky, S.H. & Dannenfelser, R.M. 1992. Aquasol Database of Aqueous Solubility. Version 5 (1992).

Yoo, J.W., Doshi, N. & Mitragotri, S. 2011. Adaptive micro and nanoparticles: temporal control over carrier properties to facilitate drug delivery. *Advanced Drug Delivery Reviews* **63:**1247-1256.

Young, P.J. 1984. Odours from effluent and waste treatment. *Effluent & Water Treatment Journal* **24:**189-195.

Zarietalabad, P., Siemens, J. Hamer, M. & Amelung, W. 2013. Perfluorooctanoic acid (PFOA) and perfluorooctanesulfonic acid (PFOS) in surface waters, sediments, soils and wastewater – A review on concentrations and distribution coefficients. *Chemosphere* **91:**725-732.

Zhang, X. & Gong, Z. 2013. Fluorescent transgenic zebrafish *Tg(nkx2.2a:mEGFP)* provides a highly sensitive monitoring tool for neurotoxins. *PLoS One* **8:**e55474.

Zhang, Z., Le Velly, M., Rhind, S.M., Kyle, C.E., Hough, R.L., Duff, E.I. & McKenzie, C. 2015. A study on temporal trends and estimates of fate of Bisphenol A in agricultural soils after sewage sludge amendment. *Science of the Total Environment* **515:**1-11.

6. Appendix A

6.1. Bayesian Network modelling

Bayesian belief networks (BBN) (also known as belief networks, causal nets, causal probabilistic networks, probabilistic cause effect models, and graphical probability networks) are graphical models consisting of nodes (boxes) and links (arrows) that represent system variables and their cause-and-effect relationships (Jensen 1996, 2001). BBNs consist of qualitative and associated quantitative parts. The qualitative part is a directed acyclic graph (cause-and-effect diagram consisting of various nodes and links) while the quantitative part is a set of conditional probabilities that quantify the strength of the dependencies between variables represented in the directed acyclic graph (Bashari et al. 2009). A directed acyclic graph can be defined as an ordered pair that consists of a finite set of vertices or nodes (V) and an adjacency relation E on V. The graph is denoted as (V,E). For each (A,B) $\in$ E (where A and B are nodes) there is a directed edge from node A to node B. In this representation, A is called a parent of B and B is called a child of A. In a graph, this is represented by a link (arrow) which is drawn from node A to node B. For any A $\in$ V, (A,A) $\in$ E, which basically means that a link cannot have a node as both its start and end point. Each node in a network corresponds to a particular variable of interest (Janssens et al. 2006).

Links in a BBN represent direct conditional dependencies between variables. For example, variables B and C are conditionally independent from variable A if and only if P(c,b|a) = P(c|a)P(b|a). Hence, as a consequence P(c|b,a) = P(c|a) for all values a, b and c of variables A, B and C. Variables B and C are also said to be independent conditional on A.

A BBN also represents distributions, in addition to representing statements of independence. A distribution is represented by a set of conditional probability tables (CPT). Each node A has an associated CPT that describes the conditional distribution of A given different assignments of values for its parents.

Structural learning

The process of structural learning determines the dependence and independence of variables within the network. This process is used to validate the structure of the conceptual model. The structure can then be amended and adjusted (i.e. learn) to better represent the system being studied (exposure to agents within sewage sludge).

Once the relationships between nodes were quantified, the influence that one node has on another was inferred. This is defined as the expected entropy reduction of one node due to a finding (observation) related to the other node. The dependent variable is called the query variable (Q), the independent variables are called

findings variables (F). The expected entropy reduction of Q due to a finding related to F can be calculated according to the following equation (Pearl 1988):

$$I(Q,F) = \sum_{q}\sum_{f} p(q,f)\log\left(\frac{p(q,f)}{p(q)p(f)}\right) \qquad \text{(Equation 6-1)}$$

Where p(q,f) is the posterior probability that a particular state (e.g. a node 'risk' could have two states: high or low) of Q(q) and a particular state of F(f) occur together; p(q) is the prior probability that a state q of Q will occur and p(f) is the prior probability that a state f of F will occur. The probabilities were summed across all states of Q and across all states of F.

6.2. Multi-media fugacity modelling

Exposure to organic agents and PPCPs via crop uptake presents various challenges. There are few data describing the uptake of organic contaminants by crop plants and therefore there is limited ability to evaluate the influence of soil chemistry on plant uptake. This is especially true for the majority of organic agents and PCPPs assessed in this study given the 'emerging' nature of our priority list. However, it is possible to derive a generic (i.e. land use:soil type – independent) exposure assessment based on partition coefficients that tend to be derived for most commercially-available chemicals and PCPPs.

Organic contaminants may enter crop plants through several pathways. The main uptake processes include i) uptake with soil water, ii) diffusion from soil or air, and iii) deposition of soil or airborne particles. The importance of the different pathways depends on both the contaminant-specific and plant-specific properties (Trapp and Legind, 2011). Experiments and model simulations have shown that persistent, polar (log octanol:water partition coefficient, Kow < 3) and non-volatile (dimensionless Henry's constant (the proportionality constant in Henry's gas law that states that the amount of dissolved gas is proportional to its partial pressure in the gas phase) < 10^{-6}) contaminants generally have the highest potential for accumulation from soil into plants. Concentrations in roots and leaves may even exceed the concentrations in soil (in some cases by several orders of magnitude), which among other things is because the water content in roots (up to 95%) usually is higher than in soils (about 30%). Volatile contaminants have a low potential for accumulation, because they quickly escape to air (Trapp and Legind, 2011).

The crop type is decisive for which uptake processes are more likely to be dominant. For example, the accumulation of contaminants from soil will be higher for root crops than for tree fruits, while the accumulation by uptake from air is higher for fruits. The degree to which physiological plant-specific parameters such as leaf area, transpiration rate, water and lipid contents as well as growth rate affect the uptake is highly dependent on the properties of the contaminant of interest. For example, will

water soluble contaminants be rapidly translocated from soil to leaves, and the accumulation in leaves will in this case almost entirely be decided by transpiration rate (Trapp and Legind, 2011).

The uptake of contaminants by plants can be estimated in different ways. A simple way of doing this is through bioconcentration factors (BCFs), which express the ratio of contaminant concentration in an organism (e.g., the crop plant) to contaminant concentration in the surrounding medium. Measurements of concentrations in plant tissues and concentrations in soil will yield a BCF plant to soil, given by:

$$BCF = \frac{C_{plant}}{C_{soil}} \qquad \text{(Equation 6-2)}$$

Where C_{plant} is the concentration in plant tissue and C_{soil} is the concentration in soil. BCFs (or regression equations relating BCF to contaminant-specific properties) are usually determined through controlled experiments in the laboratory or in the field. It is important to note that BCFs will only be valid for the exact conditions under which they are estimated, i.e. for the specific contaminant and soil type used for the determination.

A range of mechanistic models capable of simulating plant uptake of organic contaminants furthermore exists (e.g., Rein et al., 2011; Passuello et al., 2010; Fujisawa, 2002; Trapp, 2004; Hung and Mackay, 1997; Paterson et al., 1994; Trapp et al., 1994). These models vary in complexity and usually aim at determining either the steady-state or dynamic uptake for specific crop types. Many of these models are based on a multimedia modelling principle; where mass balances are set up and combined for the different compartments considered (e.g., soil, roots, and leaves). Assuming equilibrium partitioning, this leads to relatively simple ordinary differential equations that can be solved analytically. These models are then capable of simulating the partitioning, transfer, and fate of chemical pollutants within and between the different compartments. The actually occurring processes and their parameterization depend on the type of crop and the contaminant properties.

Soil characteristics (Table 6-1) were used to parameterize an equilibrium partitioning model (Level I fugacity model, MacKay, 2001) in order to predict the general partitioning behaviour and preferential partitioning in an agricultural soil amended with sewage sludge.

Table 6-1 Input data set for the standard model for the calculation of the Phase I fugacity equilibrium (Eqs. 6-3 – 6-6) and subsequent plant uptake (Eqs. 6-7 – 6-9); normalised to 1 m of soil). From Trapp and Legind (2011).

Symbol	Input [unit]	Value
Fugacity equilibrium		
R_A	Sewage sludge application rate [t ha^{-1}]	50
A	Unit area [m^2]	1
ρ_{ss}	Sewage sludge density [kg L^{-1}]	0.6
V_{ss}	Applied sewage sludge volume [m^3]	0.0083
pd	Plough depth [m]	0.25
V	Soil-air-water volume [m^3]	0.25
θ_a	Air content of soil	0.1
θ_w	Water content of soil	0.3
n	Total porosity	0.4
Vs	Soil volume [m^3]	0.15
$f_{oc,\ soil}$	Fraction of organic carbon in soil	0.05
$f_{oc,\ ss}$	Fraction of organic carbon in sludge	0.5
P_a	Partial/vapour pressure [Pa]	9900
T	Temperature [K]	298
R	Gas constant [J/(mol*K)]	8.313
S	Solubility in water [mg/l]	300[a]
ρ_b	Bulk density [kg/l]	1.6
K_{OW}	Octanal:Water partition coefficient [l/kg]	3.32[b]
K_{OC}	Organic carbon distribution coefficient [l/kg] $=10^{\wedge}(1.04\ \log K_{OW} - 0.84)$	410.02
f_{OC}	Fraction of organic carbon in mixed system $=(Vs\ f_{OC,\ soil} + V_{SS}\ f_{OC,\ SS})/(V_S + V_{SS})$	0.068

K_d	Distribution coefficient in soil [l/kg] $=K_{OC}\ f_{OC}$	27.88
H	Henry's constant [dimensionless] $=(P_a\ MW/(RT)/S)$	1.75
Roots		
W_r	Water content of roots [L/kg]	0.89
L_r	Lipid content of roots [L/kg ww]	0.025
Q	Transpiration stream [L/d]	1
M_r	Root mass [kg ww]	1
k_r	First-order growth rate [1/d]	0.1
Leaves/grains		
A_p	Area of leaves [m^2]	5
	Area of grains [m^2]	1
W_p	Water content of leaves [L/kg]	0.8
	Water content of grains [L/kg]	0.15
L_p	Lipid content of leaves/grains [L/kg ww]	0.02
M_p	Mass of leaves/grains [kg ww]	1
ρ_p	Density of leaves/grains [kg ww/L]	1
g_p	Conductance of leaves/grains [m/d]	86.4
k_p	First-order growth rate for leaves/grains [1/d]	0.035
Q_p	Transpiration stream for leaves [L/d]	1
	Transpiration stream for grains [L/d]	0.2

While it was not possible to validate the results of this model with actual measurements, it does provide an indication of how the organic and PPCPs of interest might 'behave' in the soil environment. A three-compartment soil matrix (Hough et al., 2012) consisting of: air ('A', pore space), water ('W', soil pore water) and sewage sludge amended soil ('SS'). If these compartments are assumed to be

in equilibrium, the total mass of a specific agent in the system (T, mol) is described by:

$$T_x = V_A C_A + V_W C_W + V_{SS} C_{SS} \quad \text{(Equation 6-3)}$$

Where T_x is the total mass of the agent of interest in the system, V represents the volume of each compartment (m^3), and C represents the concentration of the agent of interest in each compartment (mol m^{-3}). If the total mass of the agent of interest, T_x, is known, thus Equation 6-4 is used to estimate the partitioning of the agent of interest between the three phases (A, W, SS) within the soil matrix.

To quantify the equilibrium between the compartments, the relationships between C_A, C_W, and C_{SS} were estimated by deriving partition coefficients (Equation 6-4; Equation 6-5).

$$C_A = HC_W \quad \text{(Equation 6-4)}$$

$$C_{SS} = k_d \rho_b C_W \quad \text{(Equation 6-5)}$$

Where H is the Henry's Law constant, k_d is the distribution coefficient in soil, L kg^{-1}, and ρ_b is the bulk density, kg L^{-1}.

The partition coefficients can be used to characterise the distribution of the agent of interest within the system (Equation 6-6).

$$T_x = V_A HC_W + V_W C_W + V_{SS} \rho_b k_d C_w \quad \text{(Equation 6-6)}$$

Subsequently, the fractions of the agent of interest in water (W_x), air (A_x), and sludge-amended soil (SS_x) can be derived from Equation 6-7 - Equation 6-9:

$$W_x = \frac{V_W}{(V_W + HV_A + \rho_b k_d V_{SS})} \quad \text{(Equation 6-7)}$$

$$A_x = \frac{HV_A}{(V_W + HV_A + \rho_b k_d V_{SS})} \quad \text{(Equation 6-8)}$$

$$SS_x = \frac{\rho_b k_d V_{SS}}{(V_W + HV_A \rho_b k_d V_{SS})} \quad \text{(Equation 6-9)}$$

To parameterise Equation 6-3, the volumetric composition of the sewage sludge-amended agricultural soil was derived by assuming a unit area of 1 m^2 and a plough depth of 0.25 m in order to calculate a total working volume of 0.25 m^3. All other parameter values required are detailed in Table 6-1.

An important question is whether the agent(s) of interest present in the sludge-amended soil has the propensity to be taken up into plants, and hence enter the (human) food chain. Plants take up water through their roots, and if agent(s) of

interest is present in the water in the plants' root zone, then the agent(s) may be taken up by the plants. This question was investigated using steady-state plant uptake models previously published by Trapp and Legind (2011). Estimated concentrations of agents of interest in the three phases of the soil matrix (air, water, sewage sludge-amended soil) as derived using the Level I fugacity model (Equation 6-3 - Equation 6-9) were used as input into the plant uptake model. This model assumes that no removal of contaminants occurs due to degradation, infiltration and volatilization, and as such can be considered a precautionary approach. Finally, deposition of particles on the surfaces of leaves or grains is neglected and uptake from air is assumed solely by diffusive exchange in the gas phase. The steady-state expressions are given by:

$$C_{roots} = \frac{Q}{\frac{Q}{K_{rw}} + k_r M_r} C_W \qquad \text{(Equation 6-10)}$$

$$C_{plant} = \frac{\frac{Q_p}{K_{pw}} C_{roots} + A_p g_p C_A}{\frac{H}{K_{pw}} A_p g_p + k_p M_p} \qquad \text{(Equation 6-11)}$$

Where C_{roots} and C_{plant} are the concentrations in the roots and plant (here: leaves or grains), Q and Q_p are the transpiration stream (L d^{-1}) and the transpiration stream for leaves/grains (L d^{-1}) respectively, M_r and M_p are the root mass (kg ww) and leaf/grain mass (kg ww), respectively, A_p is the area of leaves or grains (m^2), g_p is the conductance of leaves/grains (m d^{-1}), and k_r and k_p are the first-order growth rate coefficients for the roots and leaves/grains, respectively. K_{rw} and K_{pw} are the equilibrium partition coefficients (L kg^{-1}) between roots and water and between leaves/grains and water, respectively. These can be determined through the following empirical expression:

$$K_{xW} = W_x + 1.22 L_x (K_{OW})^b \qquad \text{(Equation 6-12)}$$

Where W_x and L_x are the water and lipid content of roots, leaves or grains and b is a correction factor for differences between solubility in octanol and sorption to plant lipids. Based on previous studies, b can be assumed to be 0.77 for roots and 0.95 for leaves/grains (Trapp and Legind, 2011).

As seen from the above equations, the concentrations in soil water and air are needed in order to estimate the accumulated concentrations in roots and leaves/grains. Here, fugacity (based on adjusted pressures and tendency to move as liquid or volatile phase) modelling (Mackay 2001) was used to estimate the propensity of various organic compounds to partition between the various phases of the soil matrix (i.e. air, water, soil mineral matter, soil organic matter). The fugacity modelling is, like the plant uptake model, based on an equilibrium assumption.

The SOIL model (Mackay, 2001) comprises four environmental compartments: air, water, soil mineral matter and soil organic matter. Densities, volumes, areas and

depths of soil are user-specified, enabling total volumes and masses to be calculated, from which individual fugacities and environmental concentrations are derived (Mackay, 2001). The SOIL model has been modified to account for the mixing of the soil with SSGW compost due to ploughing. Table 6-1 presents the soil specific parameters used for the fugacity calculation. The modified SOIL model was used to estimate the propensity of each organic contaminant to enter the different phases of the soil matrix and the output was subsequently used as input for the plant uptake model.

Although more sophisticated plant uptake models capable of simulating the dynamic behaviour of the soil-plant system exist, we consider the above approach for estimating the uptake of organic contaminants into crop plants appropriate for the current purpose. The chosen plant uptake model is well-accepted; it is simple and relies on relatively few inputs. This is important given the limited data available for the 'emerging' contaminants covered by this study. The use of the steady-state solution is likely to overpredict the concentrations in the crops by orders of magnitude, which is in line with the precautionary approach used throughout this project. In reality the source (concentrations in soil) is better described as a pulse injection.

It should be noted that the plant uptake predictions are uncertain due to the large variations in both environmental and plant physiological conditions. Because of the limited data available, the calculations carried out here are therefore by necessity rather crude. Where available, the calculations will be supported by results and conclusions from the literature such as published bioconcentration factors

The final step of this exposure assessment was to estimate the Average Daily Dose (ADD; mg kg^{-1} d^{-1}) of a specific agent (e.g an organic contaminant) to a specific receptor (human). In general terms the exposure model can be simplified to the following equation:

$$ADD = \left(\frac{C_s R_{in}}{BW} \right) F \qquad \text{(Equation 6-13)}$$

Where C_s is the concentration (mg kg^{-1}) of the specific agent in the exposure medium (e.g. surface of soil/crop); R_{in} is the rate of ingestion (kg d^{-1}) of the exposure medium, BW is the body weight (kg) of the receptor (e.g. juvenile, adult), and F is the fractional time of exposure.

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