Final report

Composts derived from catering wastes containing meat: Assessment of residual pathogen risks to livestock

The report updates a risk assessment undertaken in 2002, examining the potential risks to livestock from composts derived from inputs including catering wastes. New assumptions and data have been used to estimate individual risks to grazing livestock (as per animal per year) and number of years between infections (or number of infected livestock per year) in GB. The importance of the current grazing ban has also been re-examined

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Front cover photography: Grazing cattle

Executive Summary

This report summarises the findings of a series of risk assessments that examine the fates during composting and subsequent land application of various pathogens of animal health interest that might be present in kitchen ('catering') waste sent for composting.

Overall, the conclusions of this study are that there is no need to tighten current composting process parameters or amend the length of the grazing bans, and that prevention of process by-pass remains a critical control point in preventing pathogens from re-entering the food chain.

The risk assessment is based on worst-case assumptions to overcome uncertainty. Many of these assumptions are deliberately unrealistic, but the estimation of risks that results should provide confidence to users of catering-waste composts as they overstate the likely actual risk (i.e. they are absolute worst-case results). This risk assessment assumes that:

- All the meat discarded in GB through catering waste over a period of one year goes to composting;
- All of the resulting compost is spread on agricultural land;
- Where compost is applied to grassland, livestock graze that land every day for a year;
- In the case of surface-applied compost, livestock ingest compost to the complete exclusion of soil, every day for a period of one year. There is thus no dilution of the compost in the soil for surface-applied compost.

The predicted impact of these assumption for incidence of a range of diseases is presented in Table 1, below:

Table 1: Predicted number of years between each infection in livestock in GB

	Surface applied (no dilution in soil)			Minimum tillage to 10 cm depth	
Grazing ban	**None**	**3 week**	**2 month**	**None**	**3 week**
Classical Swine Fever	3.4	48	4,110	256	3,579
African Swine Fever	29,574	123,400	1,858,000	2,218,000	9,254,000
Foot & Mouth Disease (sheep)	7,773	80,771	5,392,000	583,000	6,058,000
Swine Vesicular Disease	8.6×10^{8}	8.6×10^{8}	8.6×10^{8}	6.4×10^{10}	6.4×10^{10}
Toxoplasma gondii[‡]	0.019	0.335	0.1	1.4	2.5
BSE	7	7	7	511	511
Classical scrapie (95th percentile)	14.7	14.7	14.7	0.195	0.195
Atypical scrapie (95th percentile)	9.6	9.6	9.6	0.13	0.13

[‡]abortion in pregnant sheep for UK

Specific points to note:

- Predicted risks to individual livestock from FMDV, ASFV and SVDV are negligible at less than one per billion ($<10^{-9}$) per animal per year, even with no grazing ban and for surface application of compost (such that livestock ingest compost with no soil for a year).
- For surface applied compost, it is recommended that the grazing ban remain 2 months for pigs, as the model predicts one case of CSF every 48 years with just a 3 week grazing ban, reducing to one case every 4,110 years with the current 2 month grazing ban.
- Although parvoviruses and circoviruses are more resistant to heat inactivation processes than the other viruses considered here, there is evidence to suggest that significant inactivation (>3-log for parvovirus, and perhaps up to 2.6-log for porcine circovirus) may be achieved through composting over 48 h at 60°C. A second composting step (as required under National regulations for composting of catering waste containing meat) would give additional inactivation. Although the risks of transmission of PPV and PCV2 to pigs through composted catering waste may be low relative to other routes, it cannot at this stage be demonstrated that the risks are negligible. This is because quantitative risk assessments are impossible at present, due to lack of information.
- Avian viruses, namely AIV and NDV, are enveloped and are inactivated by heat. Commercial free-range and housed poultry are unlikely to be exposed to compost. The risks posed to backyard poultry from NDV and AIV in compost are likely to be insignificant compared to the risks from other routes.
- Even allowing for regrowth in the meat and compost, the levels of pathogens such as salmonellas, *E. coli* O157 and campylobacters present in composted catering waste would be well below those of stored manure, which is currently used on 78% of farms.
- Risks of BSE transmission to cattle through composted catering waste are remote.
- Compared to the 67,619 scrapie-infected sheep (classical and atypical) estimated to be entering the food chain annually in GB, the extra infections (classical and atypical combined) per year predicted from compost are insignificant.
- Predicted risks of abortion in pregnant sheep from *Toxoplasma gondii* from cat litter contamination of kitchen waste are very low compared to reported abortion rates in the UK.
- The risks to grazing livestock from *Clostridium botulinum* in compost which has been tilled into a depth of 10 cm are low.
- It is concluded that further consideration of the risks of MRSA through compost is not required.
- Composting process by-pass significantly increases any risks and must be minimized by ensuring the compost process operators continue to adopt and implement robust HACCP plans, and comply with the requirements of the Animal By-Products Regulations.
- Although outside of the scope of this risk assessment, the complete prevention of direct feeding of catering waste to livestock is extremely important.

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1.0 Introduction

1.1 Project Background

While safe for human consumption when cooked, some uncooked meats may contain pathogens that impact on animal health. For example, illegally imported meats could have viable titres of foot and mouth disease virus (FMDV), swine vesicular disease virus (SVDV), African swine fever virus (ASFV), classical swine fever virus (CSFV) and avian influenza virus. In 2001, Defra commissioned an independent assessment of the risks from such pathogens in compost produced from catering waste containing meat (Gale, 2002; Gale, 2004). That risk assessment considered the risks to livestock grazing or livestock housed on land to which the compost had been applied. In total 14 pathogens were considered in the original risk assessment and are listed in Table 1-1.

Table 1-1 A list of the pathogens considered in the 2002 composting risk assessment (Gale, 2002)

Pathogen	Nature of agent	Livestock receptors considered
BSE	Prion protein	Cattle
Scrapie	Prion protein	Sheep
Foot and Mouth disease virus	Virus	Cattle, sheep, pigs
Classical swine fever virus	Virus	Pigs
Swine vesicular disease virus	Virus	Pigs
African swine fever virus	Virus	Pigs
Newcastle disease virus	Virus	Poultry
Toxoplasma gondii	Protozoan	Sheep
Cryptosporidium	Protozoan	
Salmonellas	Bacterium	
Campylobacters	Bacterium	
Escherichia coli O157:H7	Bacterium	
Clostridium botulinum	Bacterium	
Trichinella spiralis	Nematode	Pigs

As part of that risk assessment, the operational conditions were defined for the composting of catering waste, namely that a time/temperature combination of 48 h at 60°C (to ensure 4h at 56°C in the centre of a 40 cm joint of meat) should be obtained for each composting step. Where the catering waste included meat, then the risk assessment determined that two composting steps would be required to ensure adequate mixing and heating. Where meat was excluded from the catering waste, then the risk assessment determined that one composting step was required, followed by an 18 day stockpiling period. These operational process conditions were incorporated into the Animal By-Products (ABP) Regulations (2003) (SI 1482) and (2005) (SI 2347) and are currently specified in the guidance to the Animal By-Products (Enforcement) (England) Regulations 2011, Animal By-Products (Enforcement) (Scotland) Regulations 2011, and Animal By-Products (Enforcement) (Wales) Regulations 2011.

The original risk assessment identified many data gaps which led to the use of assumptions and uncertainty associated with the estimates of risk. For example, it was assumed that 10,000 viraemic pig carcasses infected with CSFV were illegally imported, and that the high infectivity bone marrow accounts for 10% weight by weight (w/w) of the carcass. Other assumptions included that England and Wales comprised of a homogenous single field to which compost was randomly applied where livestock had access. Despite these necessary assumptions, the fundamental risk assessment approach remains valid and indeed has been

applied, in part, in subsequent risk assessments for livestock pathogens in the environment; notably transmission of FMDV through water (Schijven *et al.*, 2005), and the risks of bovine spongiform encephalopathy (BSE) through application of meat and bone meal to land (Cummins and Adkin, 2007).

Since 2002, new information and data have become available that could be used to update the original risk assessment. For example, quantitative estimates of the amount of illegally imported meat products have been produced (Hartnett *et al.* 2004). Furthermore an extensive study of food waste (WRAP 2008a) has enabled better estimates of the amount of meat discarded to catering waste at a livestock species level. It is therefore timely and appropriate to revisit some of the data sources, and the assumptions, to determine whether the 2002 risk assessment conclusions are still valid and appropriate.

It should be noted that this report does not consider the risks to human receptors from those zoonotic pathogens under consideration. An assessment of any such risks forms part of a separate risk assessment, also supported by WRAP (project OAV025-004).

1.2 Scope of revision

The aim of this project is to revise the original risk assessment (Gale 2002) given the availability of new information, and subsequently to review the validity of the key assumptions made. Specifically, the objectives are to use new assumptions and data:

- To estimate individual risks to grazing livestock (as per animal per year) and number of infected livestock per year in GB for three compost soil-incorporation scenarios, namely plough depth, minimum tillage, and surface application to grass; and
- To assess the importance of the grazing ban by calculating the risks with 0, 3 week and 2 month grazing ban periods.

The pathogens included in this risk assessment were selected to cover those of most importance to British livestock and to include a wide range of pathogen groups. No pathogen/microbiological hazards have been excluded because of limitations from the scope or due to lack of information.

In addition to the pathogens listed in Table 1-1, brief examinations of the emerging risks from porcine circoviruses, porcine parvoviruses, avian influenza virus and methicillin-resistant *Staphylococcus aureus* are presented in this updated study.

Discussions have been held on whether other emerging pathogens from other parts of the world should be considered and whether in a changing world, those pathogens may become of greater importance in Britain in the future. Climate change, for example, could affect soil moisture, decay rates of pathogens in soil and the balance of various microbes.

The emergence of new diseases in other parts of the world, together with globalisation and changes in land use, together with climate change may be the key drivers. An interesting example would be the shift in food production to the less developed world in order to meet protein demand in the future. Rather than undertake an extensive horizon scanning exercise, it may be better to identify the characteristics of the "cell from hell", i.e. heat resistant, stable in environment, highly infectious through the oral route, and potentially present in meat, to rule out certain pathogens which could present a high risk. For example, viruses such as Crimean-Congo haemorrhagic fever virus (present in infected livestock in the Balkans and Middle East) and Reston Ebola virus (pigs in the Philippines) are enveloped and would not tend to survive in soil for long periods. Furthermore, their envelopes would render them susceptible to heat and hence destruction by composting.

Perhaps of greater interest are the midge-borne reoviruses, namely bluetongue and African horse sickness virus, which are non-enveloped and could be transmitted through the oral route (e.g. African horse sickness virus to dogs through infected horse meat). The emergence of these viruses in Europe is related to globalisation and to climate change in some degree, although new routes of transmission such as maternal transmission are also important (as in the case of bluetongue virus serotype 8). Given the number of different factors which may affect future exotic disease incursions or the spread of various endemic diseases within Europe, this updated risk assessment needs to be viewed as a snap-shot in time, concerning the current diseases which are deemed to impact British livestock. As with the original risk assessment, this work may require updating in the future.

1.2.1 Scope of risk assessment

The risks for Foot and Mouth Disease Virus (FMDV), Classical Swine Fever Virus (CSFV), African Swine Fever Virus (ASFV), Swine Vesicular Disease Virus (SVDV) together with those for classical and atypical scrapie and BSE are calculated for GB, i.e. England, Wales and Scotland.

The risks for *Toxoplasma gondii* are calculated for the UK using data for number of cats in the UK.

Evidence from Germany has demonstrated that frozen duck meat can be a source of H5N1 HPAIV (Highly Pathogenic Avian Influenza Virus) infection in backyard poultry (Harder *et al.* 2009). Birds slaughtered for meat during disease episodes may represent an important source of NDV (Newcastle Disease Virus) (Alexander 1988). The risk assessment for NDV is based on a "What If" scenario for 10,000 NDV-infected chicken carcasses entering the GB food chain. Similarly the risks for H5N1 HPAI virus are calculated assuming 10,000 infected duck carcasses enter the food chain.

In general, non-enveloped viruses, such as parvoviruses and circoviruses are more resistant to physicochemical inactivation procedures than enveloped viruses. Full quantitative risk assessments for porcine circoviruses, porcine parvoviruses, and MRSA are not undertaken in this work. Instead, WRAP requested brief examinations to investigate whether further work should be commissioned to assess any risks from these pathogenic agents. Further work could include laboratory experimentation or more detailed risk analysis.

Although of interest to stakeholders (particularly those who specify the use of composts on land where livestock might be grazed) ascarids are not considered here because in Quarter 3 of 2008, helminthiasis was diagnosed only twice in outdoor pigs across the whole of GB (VLA and SAC 2008), and furthermore there is evidence that eggs from the helminth *Ascaris suum* are inactivated rapidly at temperatures from 49 to 55°C (Aitken *et al.* 2005). These temperatures are below the minimum required during composting of catering waste.

Key new sources of information

The key new available data reviewed and used in the updated risk assessment are set out in Table 1-2. In order to facilitate a comparison of where new data have been used in the risk assessment, the structure of this report is broadly similar to the report for the original risk assessment (Gale 2002).

Table 1-2: New sources of information used in the revised risk assessment

Variable	Updated values (based on current evidence)	Source of updated values
Estimate of percentage of raw meat discarded	Poultry (2.82%) Pig meat (1.39%) Beef (0.8%) Lamb (1.09%)	Calculated from data on the amount of food wasted in the UK (WRAP 2008a) and Defra data on meat production in the UK
Bone marrow weight in pigs	0.546 kg estimated as 1% (w/w) of the dressed carcass	Sellers (1971) reports bone marrow in pig femur to be 0.011 kg. Pig bone marrow in long bones estimated at 0.315 kg (Table 2-9). Below 0.546 kg total viral loading estimated in carcass is little affected by amount of bone marrow (see Section 2.3.1)
Amount of infected meat illegally imported to GB per annum (95th percentile)	(kg / year) 565 (FMDV) 794 (CSFV) 0.14 (ASFV) 0.021 (SVDV)	APHA risk assessment of the amounts of illegally imported meats which are imported in to GB infected with exotic viruses (Hartnett *et al.* 2004)
Soil consumption	Maximum values found (kg/animal/day) 0.392 pigs 0.032 hens 0.69 sheep – Chronic data from James Hutton Institute green compost risk assessment 1.125 cattle – Chronic data from James Hutton Institute green compost risk assessment	Smith (1996); Hoffmann *et al.* (2002); Thornton and Abrahams 1983); Peterson *et al.* 1974; Commission of the European Communities (1996); van der Meulen *et al.* (2008); WRAP (2016).
Compost application rates	20 tds/ha	Defra Fertiliser Manual RB209 (2010)
Total compost produced from catering waste per year	6,522,000 tonnes wet weight (GB only), 3,913,200 tonnes dry weight	WRAP (2008a) for wet weight; assumes compost is 60% dry matter (as per RB209)
Total area of land to which compost is applied	195,660 ha (GB only)	Calculated as number of ha covered at 20 tds/ha by 3,913,200 tonnes dry weight
Total area of land to which compost is applied	195,660 ha (GB only) of which 58.2% (113,897 ha) is grassland for grazing	Calculated as number of ha covered at 20 tds/ha by 3,913,200 tonnes (dry weight), assuming 58.2% of land is grassland (Anon 2010)

Variable	Updated values (based on current evidence)	Source of updated values
Compost dilution in soil	Depth tilled into soil 0 cm – Surface application giving no dilution 10 cm – minimum tillage giving 75-fold dilution 25 cm – plough depth giving dilution of 187.5-fold	Tillage depths based on expert and stakeholder feedback, including Prof. Brian Chambers (pers. comm. 2008), discussion with WRAP, Gale and Stanfield (2001)
Livestock numbers exposed to compost-treated soil	GB, using England livestock densities for Scotland and Wales (see below) Cattle 181,096 Pigs 318,912 Sheep 580,875	Based on maximum livestock densities (see below) in England from Defra (2005, 2006a, 2006b) and application of compost to 195,660 ha in GB of which 58.2% used for grazing. Note that using the maximum density will provide an over-estimate for any risks, since for the purpose of calculating the number of livestock exposed, the arithmetic means should be used. These arithmetic mean data are not currently available.
Livestock densities for England	Cattle 1.59/ha Pigs 2.8/ha Sheep 5.10 /ha	Census for England - Defra (2005; 2006a,b). Note, these are the maximum values recorded.
BSE source term for GB	95th percentile of 260.23 bovine oral ID_{50} estimated to leave abattoir in GB to food chain in 2008	Adkin *et al.*, 2010
Number of cats in UK	9.2 million	Cats' Protection website
Percentage of cat litter discarded to green/catering waste	1% as a worst-case	Valorgas (2012) found no cat litter in 1,000 food waste collection bags sampled in UK
Virus destruction by composting	4.61 log	Gale (2004) and Section 4.0
Newcastle disease virus dose-response	Chicken oral ID_{50} = 80 EID_{50}	Unpublished data from APHA
Newcastle disease virus decay data	-0.095 log EID_{50} per day at 21 – 27°C -0.0125 log EID_{50} per day at 3 to 6°C	Analysis of data for decay on grain from Echeonwu *et al.* (2008) and soil survival times of Olesiuk (1951)
Avian influenza virus dose response	H5N1, Chicken oral ID_{50} = 1,000 EID_{50}	Unpublished data from APHA
AIV decay data	-0.006 log per day for virus in wildfowl wintering ground in winter	Breban *et al.* (2009)

Variable	Updated values (based on current evidence)	Source of updated values
H5N1 virus in meat	10^6 EID_{50} per gram	Thomas and Swayne (2007)

1.2.2 Transfer of disease/infection between animals is not considered

The risk assessment here considers the risk of incursion or the first infected animal (index case) through exposure to compost. Transfer of the disease to additional cases is beyond the scope of the risk assessment. Although some diseases, such as bluetongue virus serotype 8, can be transmitted through the placenta, this is a function of the disease itself and is not related to compost. With regard to exotic viruses, once the index case has been detected the country is required to take procedures to prevent further spread and eradicate. The exotic viruses generally have high R_0 or basic reproductive ratios. These are the average number of secondary cases produced by an average infectious individual livestock animal introduced into a fully-susceptible herd.

This means that a single animal infected with CSFV or FMDV can infect many other animals leading to spread and establishment of infection. Although environmental or maternal transmission is not a significant route of infection for BSE, transmission of scrapie may occur via these routes. This is not considered here. Animal-to-animal transmission could occur with faecal-oral pathogens particularly in cattle and pigs, and quantitative risk assessments for such exposures have not been undertaken here due to the lack of appropriate dose-response data. Commercial poultry operations are often "all-in/all-out", so all poultry in each batch are of the same generation and are slaughtered at the same time. There is therefore no scope for trans-generational transfer in such poultry.

1.2.3 Effects of time and seasonality

The risk assessment approach used here is based on the arithmetic mean for each of the source and pathway terms. As described by Gale (2003) the arithmetic mean accommodates the variation in each variable over the period of a year. Furthermore, the arithmetic means are used to model the effect of barriers such as decay and inactivation by compost. The basic equation is:

Equation 1 $$AM[Compost] = AM[Rawfeedstock] \times AM_{surv}$$

where AM_{surv} is the arithmetic mean of the proportion of pathogens surviving the composting process. The same equation is applied to removal of pathogens by decay in the soil over time.

A key assumption of the arithmetic mean approach used here in Equation 1 is that there is no association between pathogen loadings in the raw material and the efficiency of composting or the rate of decay in the soil (see below for discussion of seasonal effects of decay). Thus the arithmetic mean approach assumes that a high pathogen loading could equally experience a low or a high inactivation rate in composting or a high or a low decay rate in soil. Probabilistic Monte Carlo approaches (as used in other risk assessment approaches) also make the same assumption. If, for example, high pathogen loadings tended to occur when composting efficiency or decay rates in the soil were low then any risk assessment approach (be it arithmetic mean or Monte Carlo) would need to accommodate this such that the higher pathogen loadings would be correlated with the lower pathogen inactivation rates in Equation 1. However, there is no evidence that high pathogen loadings occur in meat when composting efficiency or decay rates are lower, and indeed there is no reason why this should occur. Furthermore, experiments to measure inactivation or decay of pathogens typically start with high counts or titres, so if there were some protective effect from high numbers of pathogen, this would be accommodated in the observed results. On the basis that loading and inactivation are not correlated, it is concluded that the arithmetic mean approach is acceptable and appropriate in terms of accommodating effects of seasonality and time.

The efficiency of the compost process in terms of achieving a 2.7-log removal (see below in Figure 10) should not be affected by season. Thus, although the temperatures, and in particular the extent of the "cold portion" of composting processes, may be affected by external environmental conditions and temperatures, the guidance to the Animal By-Products Regulations (2011) specifies a time/temperature combination of 60°C for 2 days with less than 0.2% by-pass which is independent of time of the year and the environment.

It is well documented that pathogen decay rates in the environment are higher during summer when the temperatures are higher. The approach taken here for NDV and *T. gondii* is to use decay rates for pathogen in the soil which were estimated for temperatures of 3 – 6°C. Similarly for the exotic viruses, decay data obtained at 4°C were used in the risk assessment, albeit in slurry and not soil. By using decay rates obtained at lower temperatures (representing winter conditions), a worst-case is adopted here which assumes, in effect, that pathogens in soil/compost experience all year round decay rates expected during the winter months when decay rates are low. It is worth noting that spreading of compost during winter months would not normally represent best practice, since the crop (be it arable or grass) would not be able to make best use of the benefits supplied by the material.

It is unlikely that there is any seasonal effect in loadings of exotic viruses, the TSE agents (BSE and scrapie), and *T. gondii* oocysts. Illegal meat could come into GB at any time of the year and could come from both northern and southern hemisphere countries, so that any seasonal effects in the country of origin may be balanced out.

It is well known that some bacterial species show seasonal variation. For example, *Campylobacter* counts in sewage showed peaks in May/June (Jones *et al.* 1990). Such peaks are accommodated in this risk assessment by using the arithmetic mean pathogen loadings. However, this raises the question as to whether all the barriers (namely composting, decay in the soil, and dilution in the soil) show any seasonal variation. As discussed above decay in the soil is greater at higher temperatures, and therefore a risk assessment using an annual average decay would be worst-case for *Campylobacter*. The argument here for faecal bacterial pathogens is academic because the approach (for *E. coli* O157 at least) is to compare loadings in compost with manure (Section 10.3.4) and not to take the risk assessment any further (i.e. it is assumed that any seasonal variation in bacterial pathogens in compost reflects that in manures – so as both experience the same decay rates).

In terms of the dose-response and livestock exposure, some receptors may be more vulnerable at certain stages of their life cycle. In particular, *T. gondii* infection may cause abortion in pregnant sheep. For this reason, the *T. gondii* risk assessment specifically focuses on this risk and adopts the worst-case assumption that all sheep exposed to compost are ewes which are pregnant all year round. For the TSEs, the exotic pig viruses (ASFV, CSFV, and SVDV), the avian viruses (NDV, AIV) and for FMDV, immunity is not an issue. Infected animals either die or are culled so as to maintain disease-free status. To the author's knowledge the dose-response for exotic viruses does not vary with the life cycle of the animal. However, it is interesting to note that some exotic pig viruses can be transmitted through skin abrasions and the degree of skin abrasion in an animal may vary over its life cycle depending on its behaviour.

In terms of underlying trends, the incidences of some pathogens in the UK, notably BSE and *Salmonella* are decreasing. In contrast, and of potential interest, is the emergence of ASFV in parts of Russia and Georgia. The sensitivity analysis (Section 9.7) specifically investigates the risks of an increasing amount of ASFV infected material entering GB.

2.0 Source Term

This section reviews the data available for characterizing the type and amount of meat entering catering waste and subsequently diverted to composting. Since the original risk assessment, new information has become available on the amount of meat from different livestock species discarded to waste in England and Wales and on the amount of illegally imported meat entering GB.

2.1 Amount of raw meat discarded for composting

For the original risk assessment there was little information on the amount of meat purchased by households and catering establishments that was discarded uncooked. Data available were based on a limited survey of staff at a private company (WRc-NSF Ltd) and a telephone survey of restaurants. From this information it was assumed that 1% of meat was discarded uncooked (Gale 2002).

2.1.1 Amounts of meat of different species discarded to waste in England and Wales

WRAP (2008a) estimated the amount of food wasted in the UK. The study commenced in July 2007 with a phase of doorstep interviews with householders within nine local authorities in England and two local authorities in Wales. It was not possible to carry out research in Scotland during 2007 and Northern Ireland was not covered by WRAP's work on food waste at the time of writing; however, WRAP extended the results to cover Northern Ireland and Scotland in order to provide a complete UK picture, and assumed that the nature of food waste in Scotland and Northern Ireland is similar to that in England and Wales. In total, 2715 householders were interviewed and several weeks later the waste from 2138 of them was collected.

The survey concluded that UK households (i.e. England, Wales, Scotland, and Northern Ireland) waste 6.7 million tonnes of food every year, around one third of the 21.7 million tonnes purchased. Of this, 560,100 tonnes was fish and meat waste (Table 2-1). Using the data in Table 2-1, estimates are made of the amounts of poultry, pig, beef and lamb that are discarded (Table 2-2).

Meat in sandwich spread, unidentified meat/offal, unidentified/mixed bones, cured meats, and other meat and fish are not included in the estimation as they add uncertainty. This uncertainty is modelled in Section 9.8, which addresses the sensitivity of the predicted risk to the parameters relating the amount of meat going to compost.

In Table 2-2, it is assumed that minced meat and meatballs contain beef, lamb, pork and turkey in the proportions of total amounts of those meats which are purchased annually (Table 2-3). It is also assumed that all 1,500 tonnes per year of hot dogs and 1,100 tonnes per year of black pudding were pig (Table 2-2) which may also be an over-estimate.

Table 2-1: Breakdown of fish and meat waste estimated to be discarded to waste in the UK (WRAP, 2008a)

Components of waste	Amount of meat waste (tonnes per year)
Poultry	312,400
Pig	102,200
Beef/burgers	(37,800 + 6,300) = 44,100
Lamb	27,100
Fish	23,800
Sandwich spread	14,300
Unidentified meat/Offal	9,100
Shellfish	8,700
Unidentified & bones	7,900
Minced meat	3,700
Meatballs	1,700
Hot dogs	1,500
Cured meats	1,400
Other meat and fish	1,300
Black pudding	1,100
Total	560,100

Table 2-2: Estimation of total amounts of poultry, pig, beef and lamb in the UK which are discarded (WRAP, 2008a)

Components of waste	Amount of meat waste (tonnes per year)			
	Poultry	Pig	Beef	Lamb
Clearly identified	312,400	102,200	44,100	27,100
Minced meat[#]	1,529	1,047	777	346
Meatballs[#]	703	481	357	159
Hotdogs		1,500		
Black pudding		1,100		
Total	314,632	106,329	45,234	27,606

[#]Totals 3,700 tonnes of minced meat, and 1,700 tonnes of meatballs assuming meat species in proportion of total amounts supplied annually (see Table 2-3).

2.1.2 Percentages of meat from different species discarded raw

The report estimated that 103,800 tonnes of the fish and meat waste is fresh, raw or minimally processed (WRAP, 2008a). With a total of 560,100 tonnes discard per year (Table 2-1), 18.5% of meat waste each year is estimated as the percentage of meat discarded raw or minimally processed, i.e. uncooked. Using this proportion, the total amounts of poultry, pig, beef and lamb that are discarded raw is estimated in Table 2-3, using the total meat production in the UK (Defra, 2009c). These meat production values include home-fed production plus imports from rest of world and imports from the EU.

Table 2-3: Estimation of percentages of purchased meats by livestock species which are discarded raw

Components of waste	Meat waste (tonnes per year for UK)			
	Poultry	Pig	Beef	Lamb
Total from Table 2-2	314,632	106,329	45,234	27,606
Estimated raw*	58,308	19,705	8,382	5,115
Total purchased in UK annually†	2,070,000	1,418,000‡	1,052,000	469,000
Proportion of meat purchased which is discarded raw	2.82%	1.39%	0.80%	1.09%

*18.5% of the total meat and fish discarded in the UK (WRAP, 2008a)
† Includes home-fed production plus imports from rest of world and imports from EU (Defra, 2009c)
‡ Includes pork, ham and bacon

The percentages of meat that are discarded raw range from 0.8% to 2.8% depending on the species. The upper estimate of this range is slightly greater than the value of 1% assumed in the original risk assessment. For the revised risk assessment, therefore, the percentages and amounts from Table 2-3 are used according to the species of meat to be composted. These percentages represent the amount of waste that could be diverted to composting if food collections were rolled-out on a national basis and achieved a 100% uptake. These figures do not consider the potential impact of government support for recovery of food wastes through anaerobic digestion nor the impacts that could result from initiatives to reduce household food waste (such as www.lovefoodhatewaste.com) both of which are very likely to reduce the amount of material available for composting. It is assumed that similar proportions are discarded by catering establishments which in the interest of maximising profits would tend to minimise waste of raw meat. In Section 9.8, a sensitivity analysis is undertaken to assess the impact of higher percentages of meat being composted.

2.2 Estimates of amounts of meat imported illegally into GB

In 2002, there was no information on the amount of meat illegally imported into GB, let alone on the amounts infected with exotic viruses. Therefore, for the purposes of the original risk assessment, worst-case assumptions were made that 10,000 carcasses infected with CSFV, SVDV and ASFV entered the food chain, together with 1,000 ASFV-infected carcasses (Gale, 2002). Estimates of the quantities of illegally imported meat made in recent risk assessments (Hartnett *et al.,* 2004), suggest that these assumptions were excessively pessimistic.

Hartnett *et al.* (2004) estimated that the total amount of illegal meat entering GB each year was, on average, 11,875 tonnes, with 90% certainty that this was between 4,398 and 28,626 tonnes per year (Hartnett *et al.*, 2004). To put this into context, 11,875 tonnes of illegal meat is equivalent to 191,000 pig carcasses, given that a pig carcass weighs, on average, 62 kg. It should be noted that the amount of illegally imported meat was not entirely domesticated pig meat, but included bush meat (wild animals). The top five contributors to the total flow were Eastern Asia, Near & Middle East, Eastern Europe, Southern Africa, and Western Africa, which together account for 77% of the total flow of illegally imported meat. These estimates of the amount of illegally imported meat are not directly used here in the revised risk assessment. Instead estimates by Hartnett *et al.* (2004) of the amounts of illegally imported meat infected with CSFV, ASFV FMDV and SVDV are used because they take into account the prevalence of each disease in the different

countries of origin, albeit in 2003. The revised values for the amounts of infected meat are detailed in the relevant section for each virus.

Hartnett *et al.* (2004) estimated the flow of illegal meat into GB based upon seizure data available from enforcement authorities. The primary source of data used to derive the flow of illegal meat was the Illegal Animal Product Seizures (ILAPS) database. That database was set up at Defra in April 2001 as a result of agreement between Defra, the Food Standards Agency, HM Customs and Excise (HMCE), Local Authorities Coordinators of Regulatory Services (LACORS) and Association of Port Health Authorities (APHA). The ILAPS database holds data collected on seizures of products of animal origin (POAO) where the importers have sought to evade correct importation procedures. Information is gathered on smuggled products in freight; both air and sea, mail and passenger baggage. Data are also collected where smuggled products are seized inland from trading premises. Data are collected on meat (including poultry), fish, dairy products and other goods, for example honey.

The ILAPS seizure data utilized in the study of Hartnett *et al.* (2004) covered a period of approximately 29 months (to 30th September 2003) and must therefore be considered as a "sample" rather than a precise observation.

2.3 Animal tissue composition

The risk assessment approach used is to estimate the pathogen loading in the carcass of a viraemic animal. Pathogen loadings differ between tissues within an infected animal. For example, the bone marrow contains high loadings of ASFV in viraemic pigs. Therefore information is needed on the weights of the different tissues that make up a pig carcass in order to estimate the overall amount of virus loading in the entire carcass. This section presents the weights of animal by-products per carcass. This information is used in the source term for estimating the loadings of viruses going into compost. This is done by multiplying the virus loading per unit weight of tissue by the total weight of tissue per carcass for each tissue type and summing (as described in the relevant sections for each virus). This approach was adopted in the original risk assessment (Gale 2002) and is a more refined approach than simply identifying the part of each carcass with the highest titre and applying that loading to the whole carcass weight.

The source term in the risk assessment for exotic viruses focuses on loadings in pig meat. This is because all of the exotic viruses considered here infect pigs and indeed, ASFV, CSFV and SVDV only infect pigs. In the case of FMDV, the data for viral titres in tissues taken from Farez and Morley (1997) are for pigs. Therefore weights of tissues from pig carcasses are used here in the risk assessment and are presented in Table 2-4.

In the case of scrapie (which infects sheep), data for weights of sheep tissues are given in Section 8.7.1.5. The only risk assessment here based on bovine products is for BSE. The source term for BSE is based on the amount of SRM material which could be in the human food chain and specific information on weights of tissues in cattle carcasses is not required.

For the purposes of the risk assessment for FMDV, consideration is not given as to which livestock species the meat originates from and it is therefore implicitly assumed that the FMDV loadings per gram of pig meat are representative of those for FMDV titres per gram of beef and sheep meat. Data from Sellers (1971) demonstrate that the titres of FMDV in tissues of pigs are typically higher than those from cattle and sheep (Table 2-5). In this sense the risk assessment presented here for FMDV is worst case. Only for faeces are the titres for pigs lower than for cattle (Table 2-5). Faeces are not considered in this risk assessment for exotic viruses in meat because they would make an insignificant contribution compared to the tissues. It should be noted that it is the average viral titre per gram of meat which is important for risk assessment and not the fact that cow carcasses are bigger

than pig carcasses. This is because the weight of illegally imported meat infected with FMDV (as provided by Hartnett et al. 2004) is used here (see Section 2.2).

Table 2-4: Pig by-products (data provided by Meat and Livestock Commission from Gale, 2002)

Category	Weight (kg)
Carcass (incl. head, feet, kidneys and flare)	62.9
Carcass (excl. head, feet, kidneys and flare)	54.64
Flare fat	1.00
Kidneys	0.26
Feet	2.00
Head, tongue	5.00
Gut contents	8.40
Intestinal fat	0.84
Caul fat	0.11
Intestines	2.70
Stomach (maw)	0.55
Category	**Weight (kg)**
Heart	0.26
Lungs	0.90
Trachea	0.04
Heart, lungs, trachea	1.20
Liver, gall bladder	1.50
Pancreas	0.06
Spleen	0.11
Blood	3.40
Cerebro-spinal fluid	
Skirt	0.35
Hair scrapings & hooves	0.84
Bladder	0.04
Reproductive organs	0.15
Lymph nodes	
Waste	0.75

Table 2-5: Titres of FMDV in tissues and excretions of cattle, pig and sheep (from Sellers 1971)

	Maximum concentration of virus		
	Cattle	Pig	Sheep
Blood	$10^{5.6}$/ml	$10^{7.2}$/ml	$10^{5.0}$/ml
Epithelium	$10^{9.0}$/g (tongue)	$10^{9.6}$/g (foot)	Not given
Bone marrow	$10^{5.9}$/g	$10^{6.1}$/g	$10^{5.0}$/g
Liver	$10^{3.6}$/g	$10^{5.6}$/g	Not given
Faeces	$10^{5.5}$/g	$10^{2.9}$/g	$10^{2.7}$/g

Farez and Morley (1997) describe the various tissues in which pig viruses replicate (in the live animal). Pork and pork products are comprised principally of skeletal muscle, bone and fat. The blood, respiratory, GI and reproductive tracts, the head, the respective lymph nodes of these parts and the tonsils are not defined as pork tissues.

2.3.1 Weight of bone marrow in pigs

In the original risk assessment, Gale (2002) made the worst-case assumption that bone marrow represents 10% (weight/weight) of the porcine carcass, i.e. that each carcass had 5.46 kg of bone marrow. The objective of this section is to establish a more realistic estimate of the weight of bone marrow in a pig.

Bone marrow is an important component of the carcass as the tissue can contain high titres of viruses such as African swine fever virus (ASFV) and Classical swine fever virus (CSFV) in infected pigs. Farez and Morley (1997) note that bone marrow, blood within the capillaries of skeletal muscles and lymph nodes (prepectoral, presternal, precrural, superficial inguinal, politeal, iliac, lumbar and renal) amount to a very small fraction of the swine carcass. With respect to pork portions, lymph nodes and bone marrow may not be present as a result of trimming and deboning of the cut. Many of the lymph nodes are removed through carcass trimming due to their fat-embodied location on the carcass.

It has proved difficult to obtain information on the weight of bone marrow in any vertebrate species. Pig anatomy books provide no information and at the time of writing, no expert opinion had been received from bone marrow transplant units and researchers. One way forward, therefore, is to estimate the internal volume of the bones of a pig. Before attempting this, however, it is of interest to determine how accurate the estimate needs to be in terms of its impact on the risk estimate. The ASFV infectivity loadings used in the original risk assessment are set out in Table 2-6. (ASFV titres are measured in haem absorbing 50% units (HAd_{50})).

Table 2-6: ASFV loadings in an infected pig (adapted from Farez and Morley, 1997)

Tissue	Weight (kg)	HAd_{50} / g or / ml	Total loading in pig (HAd_{50})
Flare fat	1.00	$10^{5.4}$	2.5×10^{8}
Intestinal fat	0.84	$10^{5.4}$	2.1×10^{8}
Caul fat	0.11	$10^{5.4}$	2.7×10^{7}
Heart	0.26	$10^{6.6}$	1.0×10^{9}
Drained blood	3.40	$10^{7.9}$	[d] 2.7×10^{11}
Lymph nodes	0.04[c]	$10^{8.5}$	1.3×10^{10}
Bone marrow	5.464[a]	$10^{9.5}$	1.7×10^{13}
Skeletal muscle	43.712[b]	$10^{6.6}$	1.7×10^{11}
Blood in muscle	5.464[a]	$10^{7.9}$	4.3×10^{11}
Total (bone-in)	(62.0)		[d] 1.8×10^{13}
Total (bone-out)	(56.6)		[d] 6.4×10^{11}

[a] assumes 10% of carcass weight (54.64 kg)
[b] assumes 80% of carcass weight (54.64 kg)
[c] value for sheep
[d] risk assessment assumes 5% of high titre blood remains in the carcass (e.g. in blood clots)

The impact of changing the estimated amount of bone marrow in a pig carcass on the overall amount of ASFV and CSFV infectivity in the carcass is shown in Table 2-7 and Table 2-8, respectively.

Table 2-7: Effect of decreasing the assumed amount of bone marrow in a pig carcass on the calculated total loading of ASFV infectivity in the carcass

Assumed % bone marrow in carcass (weight contribution)	Assumed % muscle in carcass (weight contribution)	Calculated total ASFV titre in pig (bone-in)
10% (5.464 kg)	80% (43.7 kg)	1.8×10^{13}
1% (0.546 kg)	89% (48.6 kg)	2.3×10^{12}
0.1% (0.055 kg)	89.9% (49.1 kg)	1.1×10^{12}
0.01% (0.005 kg)	89.99% (49.2 kg)	0.9×10^{12}

Table 2-8: Effect of decreasing the assumed amount of bone marrow in a pig carcass on the calculated total loading of CSFV infectivity in the carcass

Assumed % bone marrow in carcass (weight contribution)	Assumed % muscle in carcass (weight contribution)	Calculated total CSFV titre in pig (bone-in)
10% (5.464 kg)	80% (43.7 kg)	2.3×10^{11}
1% (0.546 kg)	89% (48.6 kg)	3.6×10^{10}
0.1% (0.055 kg)	89.9% (49.1 kg)	1.6×10^{10}
0.01% (0.0055 kg)	89.99% (49.2 kg)	1.4×10^{10}

As shown in Table 2-7, the magnitude of the weight of the bone marrow has a much greater impact on the calculated ASFV loading in the whole pig, if it is greater than 0.546 kg. Thus, above 0.546 kg, increasing the bone marrow by 10-fold, roughly increases the ASFV loading by 10-fold. Lowering the bone marrow by 10-fold (to 0.055 kg) or even by 100-fold (to 0.0055 kg) however, does not decrease the calculated ASFV loading by 10-fold or 100-fold respectively (Table 2-7). In fact it only halves it. This is due to the contributions from other tissues relative to the diminishing amounts of bone marrow. The same is apparent for CSFV as shown in Table 2-8. Thus, decreasing the amount of bone marrow below 1% of the weight of the pig has little effect on the predicted total loading. Indeed, establishing an accurate estimate between 0.005 kg and 0.546 kg will only affect the risk by approximately 2.6-fold in the case of both ASFV and CSFV.

Sellers (1971) reported that the bone marrow in the femur of a pig is 11 g. However, it is not clear that all the bone marrow was extracted from the femur and there are other bones in a pig to consider. In Table 2-9, it is estimated that the total internal volume of the five long bones comprising both fore legs and both hind legs of a pig is 315 cm^3 which is 0.315 kg – assuming bone marrow has a similar density to water. The volume is calculated as $V = l \times \pi \times \left(\frac{d}{2}\right)^2$ where *l* is the length of the bone and *d* is its internal diameter; hence the internal radius, *r*, is given by d/2, and the area of circle is πr^2. The sum of the volumes for each bone is multiplied by 2 to account for there being two forelegs and two hind legs per pig.

Table 2-9: Estimation of internal volume of long bones in a pig

Bone	Length (cm)	Internal diameter (cm)	Volume (cm^3)
Femur	20.2[a]	1.5	35.7
Humerus	19.5[a]	1.5	34.5
Tibia	17.8[b]	1.5	31.5
Ulna	18.3[b]	1.5	32.3
Radius	13.3[b]	1.5	23.5
Total			157.5
Total x 2 for whole animal			314.9

[a]Dikic et al. (2007)
[b]Legge, A.J. (2009)

Therefore, as a worst case, it is assumed here for the purpose of risk assessment that the total bone marrow in a pig is 0.546 kg. This would allow for any additional bone marrow in those bones not included in Table 2-9. The 0.546 kg represents 1% of the weight of a (bone-out) pig's carcass (Table 2-7 and Table 2-8). Any value less than 0.546 kg does not have a major impact on the predicted viral loading in the whole carcass, as discussed above.

2.4 Amount of compost used in agriculture produced from catering waste in GB

In the original risk assessment, it was assumed that all of the infectivity in meat discarded in catering waste was disposed of into 500,000 tonnes of compost. This was based on the State of Composting report for 1999 (Slater *et al.* 1999).

2.4.1 Compost production from catering waste in UK and GB

According to the State of Composting report for 2005/06 (The Compost Association 2006) a total of 2,073,000 tonnes of compost products were produced in the 05/06 year from source segregated feedstock in the UK. The 2,073,000 tonnes of compost produced in 2005/06 will increase considerably over the coming years as more source segregated feedstock (including catering waste) is composted. Assuming the total catering waste in the UK is 6.7 million tonnes (Section 2.1.1), then the total compost produced from catering waste would also be around 6.7 million tonnes wet weight. This is calculated on the basis that it is mixed 1:1 (w/w) with green feedstock, and that composting reduces the mass by one half. Assuming 60% dry matter, the dry weight would be 4,020,000 tonnes of compost produced annually in the UK.

As set out in Table 4 of WRAP (2008a), "The Food We Waste", Northern Ireland only contributes 178,000 tonnes of food waste. Therefore, the total for England, Wales and Scotland is estimated to be 6,522,000 tonnes of compost per year (assuming 50:50 mix with green waste and reduction in mass by one half). Assuming 60% dry matter, the estimated dry weight is 3,913,200 tds of compost produced annually in GB.

2.4.2 Proportion of compost to agriculture

A total of 2,073,000 tonnes of compost products were produced in the 05/06 year from source segregated feedstock (The Composting Association 2006). Of that, 1.0 million tonnes, or approximately 50%, went to agriculture, mainly on arable and cereal crops. Of those agricultural sites taking compost, 88% of sites applied to arable/cereal, with 12% of sites applying to grassland. According to the 2006 Farm Practices Survey (Defra 2006c), only 1% of all farm types applied compost from municipal sources. This compared to 78% of farms which used farmyard manure and 6% which used sewage sludge. Only 2% of cereal farm holdings and 3% of general cropping and horticulture farms used compost from municipal sources.

Although compost is supplied to numerous markets, agriculture is the most significant (The Composting Association, 2006). As a worst case, it is assumed here that all 3,913,200 tds of compost produced from catering waste in GB per year (Section 2.4.1) are applied with an average application rate of 20 tds/ha (Section 3.1.1) to agricultural land. If all catering waste were captured, composted and applied to agricultural land, the total area of agricultural land covered in GB would be 195,660 ha per year. This is discussed fully in Section 3.1.3.

3.0 Modelling the pathways

This section of the risk assessment investigates the partitioning of the pathogen in the compost through various event pathways, and estimates the impact in terms of viability of the pathogen at each step. From reviewing the data in the original risk assessment no changes were made on the growth of pathogenic bacteria in catering waste, or the decay rates of the exotic viruses or BSE prions in the soil. However, the dilution of compost in soil, livestock soil consumption and livestock density have been revisited and are described here, with consideration given to re-growth of bacterial pathogens in compost or composted-amended soils as discussed in Section 10.2.

3.1 Application and integration of compost into the soil

The dilution factor for compost in the soil is dependent on the application rate and the degree of mixing with soil. Both these parameters have been updated based on additional information.

3.1.1 Compost application rates provided by experts

Application rates to land tend to be limited by nitrogen loading rates of 250 kg/ha total N, which is a legislative limit in Nitrate Vulnerable Zones and good practice elsewhere (Fertiliser manual (RB209) 2010). The application rate for green compost which typically contains 7.5 kg total N/tonne fresh weight (60% dry matter) would be 33 t/ha fresh weight and 20 t/ha for dry solids. The application rate for green/food-waste-derived compost containing 11 kg total N/tonne fresh weight (60% dry matter) would be 23 t/ha fresh weight or 14 t/ha for dry solids. The higher figure is used for subsequent calculations (20 t/ha dry solids).

3.1.2 Degree of mixing with soil

Central to the degree of mixing with soil is the depth to which the compost is incorporated. The depths to which compost may be incorporated into soil are set out in Table 3-1.

Table 3-1: Depth of incorporation of compost into soil

Type of incorporation	Depth
Plough depth	25 cm is a good depth to select as typical and has been widely used in many other studies, e.g. sewage sludge risk assessment (Gale 2005a)
Minimum tillage	Reduced tillage involves cultivation of around half to two-thirds of a typical plough depth, and most commonly involves the use of heavy disc cultivators. 15 cm might reasonably be assumed (Prof. Brian Chambers ADAS, pers. comm.), but the more conservative value of 10 cm was used for the purposes of risk assessment, as agreed with WRAP
Surface application to grass	No immediate dilution – other studies have assumed 5 cm dilution depth after approximately 6 months application, due to mixing by soil fauna, etc. (Prof. Brian Chambers ADAS, pers.comm.).

Ploughing is a practice which involves inversion of the soil to a depth of around 10 inches (about 25 cm).

Minimum tillage is a low-impact system replacing ploughing in some systems (Daily Telegraph, 2008). Minimum tillage uses tines, discs, and presses to manipulate surface soils sufficiently to form a seed bed. Minimum tillage reduces the cultivation depth and can avoid the use of the plough i.e. relies on non-inversion of the soil (Scottish National Heritage, 2008). SAC (2003) note that there should be fewer or faster cultivation passes at a shallower depth than under normal ploughing. Interest in minimum tillage has been heightened in England by the publication of detailed guidelines by the Soil Management Initiative. However, according to SAC (2003), soil management practices set out by the UK Soil Management Initiative are not necessarily applicable in Scotland where the higher proportion of spring barley, the greater use of rotational cropping, the smaller scale of farm enterprises and the wetter climate can mitigate against the success of minimum tillage or direct drilling.

When considering surface application to grass, Prof. Brian Chambers (ADAS, pers. comm) has assumed dilution to a depth of 5 cm in the soil after about 6 months as shown in Table 3-1. The grazing bans after application of compost are in the order of 3 weeks to 2 months (EU control Regulation (1069/2009) and Animal By-Products Regulations 2011). To the author's knowledge, there is no information on how compost applied to the surface of grasslands will disperse into the soil over a period of 3 weeks or 2 months. The worst-case, therefore, would be to assume zero dilution of the compost into the soil following surface application – and this assumption has been modelled here.

3.1.3 Calculating the proportion of surface area covered with compost

The objective of this section is to explore the proportion of the ground surface area of the field which would be covered with compost, assuming it is evenly spread to a certain height. This is an important consideration because if it only covered a small proportion of the field surface area, it would be reasonable to assume that grazing livestock would eat some soil in addition to compost. This is calculated in terms of volumes using the densities of compost and soil. The fresh weight bulk density (BD) of compost usually lies between 500 and 600 g/litre (Wallace, *et al*. 2004). The mean dry BD ranges from 298 – 383 g / litre and an average of 327 g/l is calculated as shown in Table 3-2. Within the literature, compost generally has a dry BD of below 400 g/l and of five composts studied at Reading University, dry bulk densities between 180 and 400 g/l were obtained (Wallace *et al.* 2004).

Table 3-2: Mean dry bulk densities of composts (Wallace *et al*., 2004)

Data source for compost	Mean dry bulk density (g/l)
The Composting Association	317
The Environment Agency	298
ReMaDe Project Analyses	310
HDRA report	383 (median)
Average	327

Using an application rate of 20 tonnes (dry solids) / ha (refer to Section 3.1.1), and an average dry bulk density of 327 g per litre of compost (Table 3-2), 20 tonnes equals 61,000 litres in volume. This volume, when spread at a 1 cm height, would cover 6,116 m^2, which is 61% of the surface of a hectare (10,000 m^2). The percentages of the field covered, assuming the compost is applied to different heights, are set out in Table 3-3.

Table 3-3: Percentage of field area covered by compost at different heights (at 20 tds/ha)

Height of compost (cm)	Area covered (cm^2)	% of 1 hectare
0.61	100,000,000	100
1	61,162,079	61.16%
2	30,581,039	30.58%
5	12,232,415	12.23%
10	6,116,207	6.12%

The calculations suggest that compost applied at a height of 0.61 cm, evenly over the field, would cover 100% of the surface area of one hectare. Even at a height of 2 cm, 30% of the soil area is covered. This raises the question as to what depth of soil cattle or sheep consume. If grazing livestock take soil from 1 cm depth, then each mouthful would comprise of 61% compost and 39% soil. The dilution factor is therefore 1.6-fold. If livestock only take compost from the top 0.5 cm, then each mouthful will be 100% compost, and there will be no dilution. It should be noted that livestock, particularly cattle, will selectively avoid disturbed land, such as land to which compost has been applied. However, surface-applied compost, in time, may stimulate new growth of grass that would make the amended area particularly attractive to grazing stock.

On the basis of these calculations, and for the purposes of this risk assessment, it is assumed as a worst case that there is no dilution in the soil for surface applied composts and that grazing livestock ingest compost (to the exclusion of soil) every day for one year.

3.1.4 Compost dilutions in soil for risk assessment should be calculated on a weight by weight basis

The original risk assessment calculated the dilution factors for surface application to grass using the weights of soil and compost (as opposed to the volumes, see Section 3.1.3). Let us assume that the compost is mixed with just the top 1 cm of soil. The top 1 cm of soil taken from an area of one hectare is 100 m^3 and weighs 150 tonnes (dry weight) assuming a soil density of 1.5 g/cm^3 (Rowell 1997). Adding in 20 tds of compost gives a dilution factor of 150/20 = 7.5-fold, which is more than the 1.6-fold calculated using volume. Thus 20 tds at a density of 327 g/l is 61,162 litres in volume (refer to previous section). Applying this to one hectare to a depth of 1 cm (i.e. 100 m x 100 m x 0.01 m = $100m^3$ volume = 100,000 litres) gives a dilution factor of 100,000/61,162 = 1.6-fold.

This raises the question of why the dilution is less if the compost volume is used instead of the weight. The density of compost at 327 g / l is 4.5-fold less than that of soil at 1,500 g / l. Therefore, the volume of compost (for a given mass) is effectively 4.5-fold the volume of soil, hence its ability to cover greater areas. This in turn raises the question of whether using the weight or the volume of compost is more appropriate in the dilution calculations. Since dry matter is the key parameter for ingestion of food in risk assessments for livestock feeds (Table 3-6), it is suggested that weight be used. Pathogen concentrations are also typically calculated as per unit dry weight.

3.1.5 Effect of application rate on proportion of field covered with compost

The percentage areas of a field covered by compost for application rates between 1 and 20 tds/ha are set out in Table 3-4. This assumes the compost is applied at a height of 1 cm.

Table 3-4: Percentage of field area covered by compost at different application rates, with height of 1 cm

Application Rate (tds/ha)	Area covered (cm^2)	% of 1 hectare
1	3,058,103	3%
5	15,290,519	15%
10	30,581,039	31%
14	42,813,455	43%
20	61,162,079	61%

3.1.6 Compost application rate used for the revised risk assessment

At 14 tds/ha and a height of 1 cm, the compost covers less than half of the field area (Table 3-4), and at 10 tds/ha less than a third of the field is covered. Therefore, for these lower application rates, the risk assessment could not assume that grazing livestock eat compost to the complete exclusion of soil for surface applied compost. If an application rate of 10 tds/ha were used, there would be a 69% probability of livestock's ingesting soil rather than compost. While we could simply assume a 69:31 fold dilution in the risk assessment, this is complicated by the fact that the height of compost may not always be applied to a height of 1cm.

An application rate of 20 tds/ha was the maximum assumed for compost (Section 3.1.1). At this application rate it is estimated, assuming a height of 0.61 cm to 1.0 cm, that at least 61% of the field is covered (Table 3-3), and for simplicity, dilution effects are ignored in the case of surface applied compost. If compost were applied at the usual (good practice) rate of 14 tds/ha (Section 3.1.1), a dilution factor would need to be applied, as livestock would be more likely to eat soil than compost.

For the purpose of this risk assessment, an application rate of 20 tds/ha is used. This is two-fold higher than the 10 tds/ha used in the original risk assessment, and higher than the typical application rate for green/food composts of 14 tds/ha. Using 20 tds/ha instead of 14 tds/ha results in the prediction of slightly higher individual risks, although the total number of infected livestock predicted in GB is unaffected (see sensitivity analysis in Section 9.0).

3.1.7 Calculation of soil dilution factors for the risk assessment

The dilution factors based on weight for weight ratios of compost and soil are presented in Table 3-5. While there is likely to be some incorporation, and hence dilution, of surface-applied compost into the soil during the 3 week or 2 month grazing ban, no incorporation is assumed here.

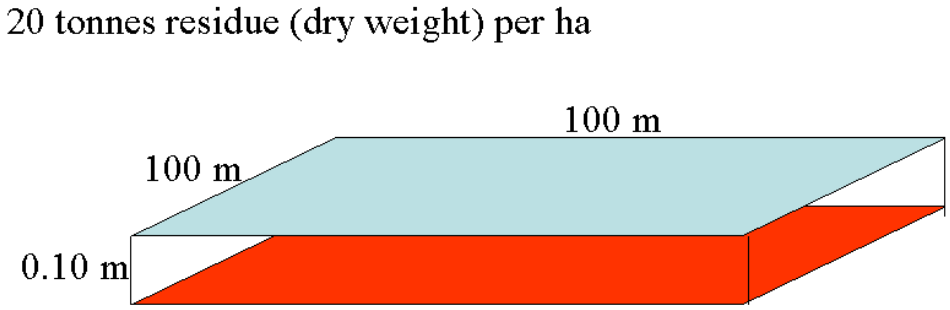
Table 3-5: Summary of dilution factors for compost in soil (calculated using weight for weight ratios)

Type of incorporation	Depth (cm)	Dilution factor*
Plough depth	25	187.5-fold
Minimum tillage	10	75-fold
Surface application	0 (worst-case)	0

*Based on application rate of 20 tds / ha

The calculation of the dilution factor for 20 tonnes residue (dry weight) tilled into a depth of 10 cm is set out in Figure 1.

Figure 1: Dilution of composted catering waste residues in soil. Based on assumption that compost is tilled in to a depth of 10 cm



Volume = 100 x 100 x 0.10 = 1,000 m^3
Density soil (dry weight) = 1.5 g/cc
Mass of soil = 1,500 tonnes
Dilution = 1,500 / 20 = 75-fold (w/w)

Each of these dilution factors can be considered in part worst-case, as they do not consider the leaching of any pathogens into lower soil levels with time due to weathering (rain) and other physical movement in the soil (e.g. due to mechanical disturbance or the action of soil fauna).

3.2 Receptor term – Soil consumption by grazing farm animals

The extent of soil ingestion by grazing animals is highly variable, with many influencing factors. These include soil properties, stocking rate and pasture availability, earthworms, plant type and sward characteristics, management practices, seasonal and climatic variations (cited by Smith, 1996). Smith (1996) assessed the extent of soil ingestion by grazing animals. In general soil intake occurs in one of two ways. The first is soil that is present on the surface of plant leaves which are eaten. The second is soil that it is ingested directly from the ground surface, from earthworm casts or attached to plant roots. Various studies have found that the intake of soil is dependent on the amount of herbage which is available for grazing (Smith, 1996).

Table 3-6: Maximum feed intakes (per day) for farm animals (adapted from Commission of the European Communities, 1996; Peterson *et al.* 1974)

	Chicken	Dairy Cattle	Beef Cattle	Sheep	Pig
Body weight	1.9 kg	550 kg	350 kg	-	75 kg
Daily feed (Dry matter)	120 g	20 kg	15 kg	Between 0.782 and 1.262 kg	3 kg

3.2.1 Sheep

One study found that soil ingestion by adult sheep increased from less than 0.5% of total dry matter (DM) intake in late May to 14% in mid-December (Smith, 1996). From a review of the literature on soil ingestion by sheep, it was determined that soil was normally only 1% to 2% of sheep's diet, but could reach 24% in "worst-cases" (Smith, 1996). Another estimate (cited by Smith 1996) is that ingestion of soil by sheep could typically be of the order of 10-15% DM of the total intake respectively on an annual basis. Thornton and Abrahams, cite adult sheep as consuming up to a maximum 30% of their dry matter as soil (Thornton and Abrahams, 1983). According to Hoffman *et al.* (2002), the lowest soil ingestion rates of sheep were as little as 1% or 2% of DM intake. When forage was sparse, during autumn or winter, intake of soil was estimated to be as great as 18% of the diet. This is because of the need to graze close to the ground.

The calculated yearly average soil intake was about 4.5% of total DM intake when animals in New Zealand grazed 365 days a year and pasture was the only feed source. This average is equivalent to intake of 45 g /day of soil by a 50 kg sheep consuming 1 kg/day DM (Hoffman *et al.*, 2002). Two management factors had a significant effect on the amount of soil consumed by sheep. These are supplementary feed and stocking density. Soil ingestion was reduced by about 50% when sheep were offered supplemental feed during the winter months. Increasing stocking density would increase soil intake because less forage would be available per animal (Hoffman *et al.*, 2002). The highest sheep intake reported by Hoffman *et al.* (2002) was 0.198 kg / sheep/ day in the Scottish study (Table 3-7).

Table 3-7: Results of representative studies of soil ingestion by sheep (Hoffman *et al.*, 2002)

Location	Season	Other feed	Soil intake (g/day)		
			Mean	High	Low
New Zealand	Winter	No	60	150	5
New Zealand	Spring to Autumn	No	4	10	0
New Zealand	Apr to Oct	No	63	108	1
New Zealand	Jul – Aug	Yes	>1	>1	-
New Zealand	Jul – Oct	No	90	-	-
New Zealand	Jul – Oct	Yes	35	-	-
New Zealand	Winter	No	83	125	43
New Zealand	Winter	Yes	48	68	26
New Zealand	Winter	No	30	41	21
Scotland	May – Dec	No	70	198	9
Ireland	Apr – Oct	No	40	159	7

3.2.2 Cattle

Fewer studies of soil ingestion have been carried out for cattle. Soil appears to comprise a lower proportion of total dry matter (DM) intake for cattle than that observed for sheep. One study cited in Smith (1996) observed that cattle ingest 1% to 11% of their dry matter intake as soil during the winter, decreasing to a maximum of 7% in May. Under good management conditions, soil ingestion by cattle rarely exceeds 3% DM intake (cited by Smith, 1996). Another estimate (cited by Smith 1996) is that ingestion of soil by cattle could typically be of the order of 5% to 10% DM of the total intakes respectively on an annual basis. Thornton and Abrahams cite that adult cattle intake between 1% and 18% of their dry matter as soil (Thornton and Abrahams, 1983).

Using the value from Smith (1996) that cattle ingest 5% to10% of dry matter as soil, and that the maximum daily intake is 20 kg DM (Table 3-6), it is calculated that dairy cattle ingest 1kg to 2 kg soil per bovine per day (Table 3-8). Hoffman *et al.* (2002) suggest that the average intake for dairy cattle in England is lower because they receive feed supplements. The % DM of 5% to 10% used in Table 3-8 may therefore be too high for dairy cattle. Hoffman *et al*. (2002) write that the soil ingested by a 500 kg dairy cow in New Zealand was estimated to average 900 g/day when it was assumed that dry matter intake was 15 kg/day and that cattle grazed 365 days per year and no supplement was offered. Hoffman *et al*. (2002) also report a mean value of 770 g/bovine/day for a New Zealand study (Table 3-9). The highest intake for cattle reported by Hoffman *et al.* (2002) was 2.4 kg / bovine/ day in the England study (Table 3-9).

Table 3-8: Estimation of intakes of soil by cattle

	Dairy Cattle		Beef Cattle	
*Daily maximum feed (dry matter) kg	20		15	
%DM intake as soil (Smith (1996))	5%	10%	5%	10%
Daily soil intake (kg)	1.0	2.0	0.75	1.5

*Data from Commission of the European Communities (1996) see Table 3-6

Table 3-9: Results of representative studies of soil ingestion by cattle (Hoffman *et al.*, 2002)

Location	Season	Soil intake (g/day)		
		Mean	High	Low
New Zealand	All Year	770	2070	260
England	Apr – Aug	310	2400	27
USA	Jun – Nov	400	1500	100
USA	May – Nov	113	146	83

3.2.3 Pigs

Pigs may ingest as much as 8% of their dry matter intake as soil because of their rooting habits (Hoffman *et al.*, 2002). Using the daily dry matter intake of 3 kg per pig, 8% equates to 240 g soil per pig per day (Table 3-11), which is in good agreement with the mean intake quoted by Hoffman *et al*. (2002) in Table 3-10. The highest intake for pigs reported by Hoffman *et al.* (2002) was 0.392 kg / pig / day in the England study (Table 3-9).

The value of 0.392 kg per pig per day is used in the revised risk assessment for pigs.

Table 3-10: Results of representative studies of soil ingestion by pigs (taken from Hoffman *et al.*, 2002)

Location	Season	Other feed	Soil intake (g/day)		
			Mean	High	Low
USA	Jun – Aug	Yes	197	392	37

Table 3-11: Estimation of intakes of soil by pigs

	Pig
*Daily maximum feed (dry matter) kg	3
%DM intake as soil (Smith (1996))	8%
Daily soil intake (g)	240

*Data from Commission of the European Communities (1996) see Table 3-6

3.2.4 Soil ingestion data used by the James Hutton Institute for a green compost risk assessment

For the green compost risk assessment, the James Hutton Institute (WRAP, 2016) used the cattle and sheep soil consumption data (provided by WRAP) set out in Table 3-12.

Table 3-12: Estimation of intakes of soil by cattle and sheep as used in the green compost risk assessment by the James Hutton Institute (WRAP, 2013)

	Grazing Cattle	Sheep
Daily maximum feed (dry matter) kg per day	12.5	4.32
Exposure	95th percentile	95th percentile
%DM intake as soil	9.0%	16%
Daily soil intake (kg per day)	1.125	0.69

The James Hutton Institute (JHI) chose to use the 95th percentile instead of the arithmetic mean, and to ensure consistency with the JHI green compost risk assessment, their approach is adopted here.

3.2.5 Poultry

Soil intake may be the most prominent source of environmental contaminants for free range and organic chickens (van der Meulen *et al.* 2008). According to van der Meulen *et al.* (2008), consumption of soil of 14g to 32 g a day can be estimated from the literature. A value of 32 g soil / chicken / day is therefore used here for the quantitative risk assessments for NDV and AIV.

3.3 Livestock densities

3.3.1 Densities of grazing cattle, pigs and sheep

The original risk assessment estimated the number of infected livestock by assuming that England and Wales was a single field to which compost was randomly applied and in which livestock randomly grazed (Gale, 2002). The total tillage and grassland for England and Wales was 9,537,300 ha in which it was assumed 8,165,500 cattle, 29,963,900 sheep and 6,374,200 pigs grazed or were housed. The calculated livestock densities are set out in Table 3-13. These are arithmetic mean livestock densities.

Table 3-13: Livestock densities used (indirectly) in the original risk assessment (Gale 2002)

	Cattle	Sheep	Pigs
Total head	8,165,200	29,963,900	6,374,200
*Density (head/ha)	0.86	3.14	0.67

*Assuming total tillage and grassland of 9,537,300 ha

Here that approach is refined by using livestock densities for livestock on farms. Livestock densities across England have been published by Defra (Defra, 2005, 2006a and 2006b). Local livestock densities will vary depending on the management system, and whether the farm is upland or lowland. Furthermore, densities may differ in Wales and Scotland to those estimated for England. The density map for pigs, for example, (Figure 2) shows densities ranging between <10 and >80 (max 280) pigs/km^2 in England. The maximum densities are summarised in Table 3-14.

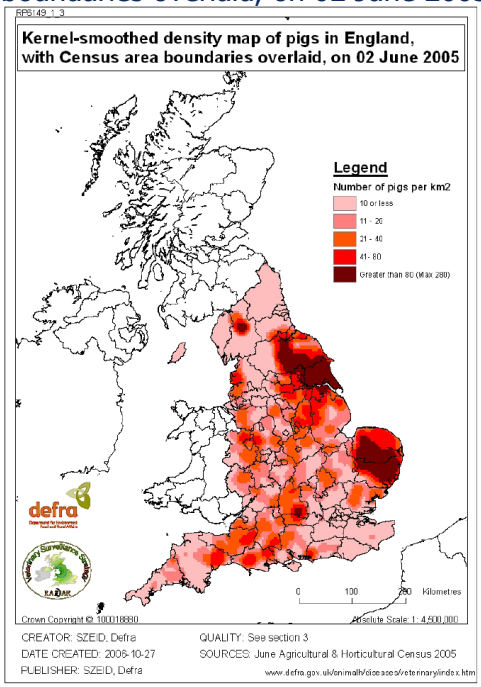
Table 3-14: Livestock density data for England recorded in 2005

	Census date	Maximum density (livestock per km^2)	Reference
Pigs	2 June 2005	280	Defra (2006a)
Cattle	1 April 2005	159	Defra (2005)
Sheep	2 June 2005	510	Defra (2006b)

The maximum densities for England in Table 3-14 are slightly higher than those used in the original risk assessment as estimated for England and Wales (Gale 2002) as shown in Table 3-13. This is not surprising as the densities used previously represented arithmetic means rather than maxima.

Density data for cattle and pigs have not (at the time of writing) been identified for Scotland. However, Dennis *et al*. (2008) assumed a commercial sheep stocking density of 2.7 ewes/ha in experiments performed in Perthshire. Their value is lower than the 5.1 sheep/ha used in the risk assessment here (Table 3-14).

Figure 2: Kernel-smoothed density map of pigs (per km^2) in England, with Census area boundaries overlaid, on 02 June 2005 (Defra 2006a)



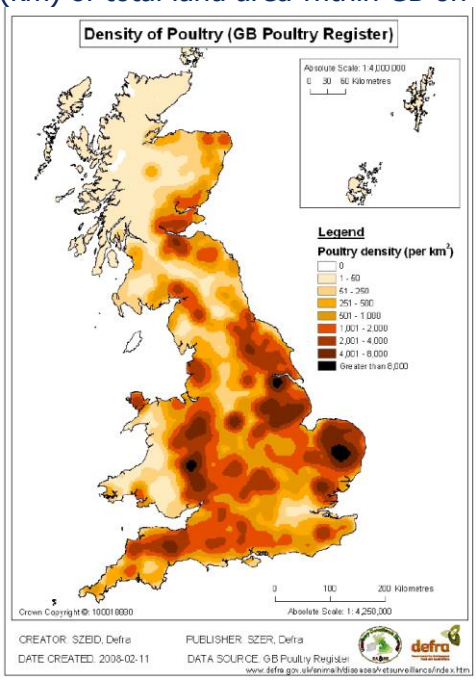
3.3.1.1 Field densities

The maximum pig density of 280 pigs/km^2 (2.8 /ha) in Figure 2 and Table 3-14 reflects all land in a given area. Clearly the pigs will be more concentrated within the boundaries of the farm itself. Cronin (1996) states that stocking rates for adult pigs in outdoor enterprises will vary according to rainfall and soil conditions. A stocking rate of 12-20 sows/ha has been suggested as a rule of thumb, with 15–18 sows/ha as the theoretical range (Cronin, 1996). However, it has been questioned whether this can be achieved in practice (Cronin, 1996). The Defra (2003) 'Code of Recommendations for Welfare of Livestock – Pigs' states a maximum pig density of 25 sows/ha. It is not, however, appropriate to use actual field densities for calculating the number of infections because the risk assessment would, in effect, be assuming that compost is being selectively applied to livestock fields to the exclusion of other types of land. Indeed, using a maximum stocking rate for pigs of 20 sows/ha in the model would in turn require the adoption of an assumption that pigs ingest 14.3% of the total compost applied to land. Similarly, sheep at 20/ha (English Beef and Lamb Executive, pers. comm. 12 June 2009) would consume 25% of the total compost produced annually. Clearly such assumptions are not realistic, and have not been used for modelling here.

3.3.2 Densities of poultry

Figure 3 shows a kernel-smoothed density map of registered poultry (birds) per square kilometre (km) of total land area within GB from data available on the 11 February 2008 (Defra 2008). The highest recorded densities are >8,000 per km^2 which is equivalent to 80 / ha.

Figure 3: Kernel-smoothed density map of registered poultry (birds) per square kilometre (km) of total land area within GB on 11th February 2008 (Defra 2008)



The structure of the poultry industry in GB is different to that of the pig, sheep and cattle industries and there is a significant difference between large commercial housed poultry operations, commercial free-range poultry operations and backyard (domestic) poultry. Defra (2007) have reported details of the statistics of the GB meat chicken population. These are set out in Table 3-15. Of 119 million chickens, 101 million (84.9%) were kept indoors and reared under commercial housed operations where the birds live in controlled environments. It is assumed for the purpose of risk assessment that indoor-housed birds are not exposed to compost. In contrast, the free range, organic and fewer than 50 bird backyard flocks (as well as the fewer than 1,000 bird flocks) are assumed to have outdoor access, where they could be exposed to compost.

The Defra (2007) report does not detail breeder broiler premises and birds, or layers. According to Defra (2008), however, there were a total of 24,269 premises (which kept a total of 251,913,661 poultry) in GB on 11th February 2008. This is over two-fold the stated number of GB meat chickens (Table 3-15). Poultry were defined in Defra (2008) as chickens (including bantams), turkeys, ducks, geese, guinea fowl, quail, partridges, pheasants, pigeons reared for meat, ostriches, emus, rheas, cassowaries and kiwis.

Assuming the same ratio of indoor birds as in Defra (2007), it is estimated that 15.1% of those 251,913,661 poultry (= 37,960,000 birds) are kept outdoors in GB, where they could be exposed to compost. It would be unrealistic to assume that all 37,960,000 birds are kept all year on the 195,660 ha of land to which all 3,913,200 tonnes dry weight of compost had been applied at 20 tds/ha. Indeed, 37,960,000 birds on 195,660 ha would give a density of 194 birds/ha, which is higher than the maximum of 80 birds/ha in Figure 3 (which includes commercial housed poultry). As with cattle and sheep assumptions, to calculate the number of poultry infections assuming 37,960,000 birds are kept on land to which all the compost

has been applied would be unrealistic. It is concluded that it is not possible to robustly estimate the number (or total population) of poultry which could be exposed to compost. Therefore, the risk assessment approach adopted here for NDV and AIV focuses on the individual risk (as a factor of compost application rate and dry matter ingestion by the bird), rather than the population risk.

Table 3-15: Details of the statistics of the GB meat chicken population (Defra 2007)

	Premises	Birds
Indoors	1,368	100,974,820
Free range	857	4,360,685
Organic	232	1,758,429
50 or less	527	12,024
Less than 1000	899	103,330
Total	2,158	118,890,235

4.0 Particle size, temperature and time for pathogen destruction by composting and biogas

The time-temperature combination and particle size criteria proposed for composting (and biogas treatment) of catering waste according to the Animal By-Product Regulations (2011) were set out and defined as part of the original risk assessment (Gale 2002). Here, a literature search has been undertaken to find any new information on inactivation of microbiological agents by the composting process. The new data are set out in Section 8.7.4 (data on scrapie inactivation in compost), Section 10.1 (data on effects of composting on genetic properties of bacteria) and Section 11.1 (impact of composting on bacterial spores). The conclusion is that there are no new data which suggest the time-temperature combination and particle size criteria recommended in the original risk assessment require amending. Therefore, the following section has been retained and updated for completeness, although the conclusions have not altered significantly since the original risk assessment.

4.1 Objectives

The objectives of this section are to:

- Review data for heat inactivation data of pathogens for different time/temperature combinations;
- Consider the effect of particle size in relation to surrounding temperature;
- On the basis of the most resistant pathogens, define a minimum temperature and time criterion;
- Identify a time/temperature combination to be achieved by composting; and
- Introduce the concept of by-pass of in-vessel and windrow composting processes as a method of calculating net pathogen inactivation.

4.2 Introduction

Discussions at the time of the original risk assessment noted the need to achieve a balance between pathogen kill, through the use of high temperatures, and obtaining stabilized compost. The use of high temperatures can lead to a loss of the soil-conditioning properties of compost, and the use of lower process temperatures has been advocated by some. Where lower temperatures are used, the indigenous microflora of the material being composted will be preserved, and competition for nutrients created by this natural microflora will tend to suppress growth of any (bacterial) pathogens in the meat which survive the composting process. For example, the indigenous microbes are important in preventing *Salmonella* regrowth in compost (Sidhu *et al.* 2001). Regrowth of faecal-oral bacterial pathogens in the compost is discussed in Section 10.2.

In general, bacteria are more rapidly inactivated by temperature than viruses. Moreover, while many faecal-oral bacterial pathogens are endemic in GB, the exotic viruses which cause such devastating diseases in livestock as FMD and CSF are, by definition, not present in GB. A major objective of this work is to ensure that the composting process is sufficient to prevent incursions of such exotic viruses in GB. The temperature-time criterion for the composting process is therefore based on the inactivation of the exotic viruses which may be present in meat. Here, the "hot part" of the compost process is defined as that material which reaches a minimum temperature of 56°C for a minimum time period of 4 h in each composting step, and represents the minimum condition achieved at the centre of a 40 cm diameter particle (see Section 4.0). In each composting step, the bulk medium is held at 60°C for 48 h where greater inactivation would be achieved than is required or modelled. Data on heat inactivation of exotic viruses and faecal bacteria in the hot part of composting are now reviewed.

4.3 Heat inactivation of exotic viruses and faecal bacterial pathogens in the "hot" part of composting

Turner *et al.* (2000) report data on decontamination of pig slurry containing exotic viruses of pigs (namely FMDV, Aujeszky's disease virus and CSFV) by heat inactivation. The work demonstrated the suitability of thermal treatment in ensuring the safety of pig slurry following a disease outbreak. Turner and Burton (1997) reviewed the inactivation of viruses in pig slurry. They concluded that the most suitable treatments were the use of heat at about 60°C for up to 30 minutes. At 55°C considerable destruction of pig viruses occurs within 10 – 15 minutes.

4.3.1 Foot and Mouth Disease Virus

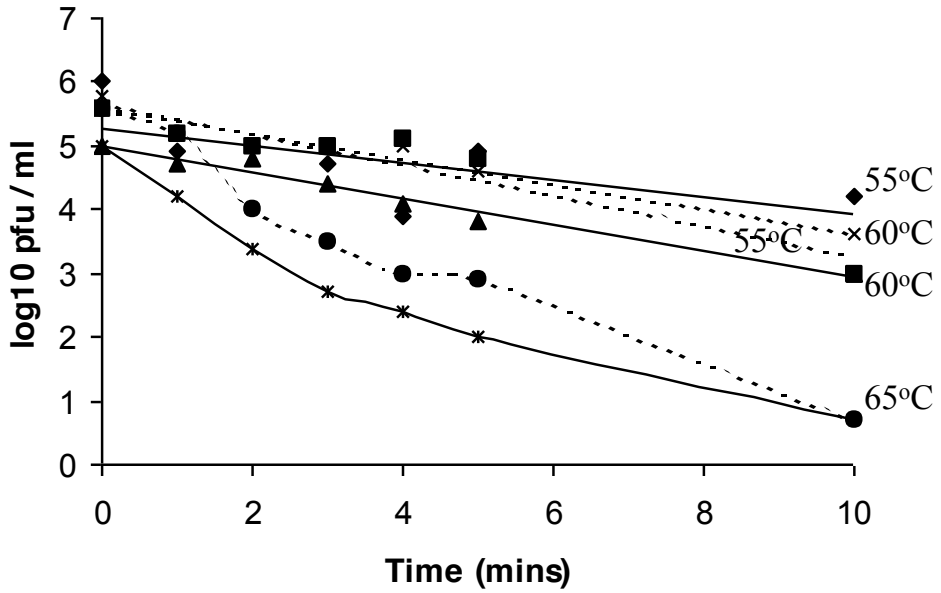
The inactivations of FMDV over a 10-min time period at three temperatures, namely 55°C, 60°C and 65°C, are shown in Figure 4. The net destructions, calculated on pooling both the slurry and culture medium data, are presented in Table 4-1. The data show that even at the lower temperatures of 55°C and 60°C considerable inactivation occurred over the 10 minute time period of the experiment. Thus even at 55°C a 1.92-log inactivation was measured over just 10 minutes.

Table 4-1: Net destruction of FMDV over 10 minutes (data from Turner *et al.,* 2000)

Temperature	0 minutes (pfu/ml)	10 minutes (pfu/ml)	Net destruction	log net destruction
55°C	699,053	8424	82.9	1.92
60°C	365,478	2490	146.7	2.16
65°C	249,053	5	49692.7	4.69

Data averaged for culture medium and pig slurry counts

Figure 4: Laboratory scale thermal treatment of FMDV incubated at different temperatures with time in pig slurry (solid line) and Glasgow Eagles medium (dashed line). Data from Turner *et al.* (2000)



Kamolsiripichaiporn *et al.* (2007) provide decimal reduction times for FMDV at a variety of temperatures. They conclude that the effective inactivating temperature is approximately 60°C. FMDV serotype O was found to be the most heat resistant at 60 to 80°C with a decimal reduction time (time for 1-log reduction) of 42 seconds at 60°C. At 50°C, around 3-log decay of FMDV was observed in the initial decay period of 13 minutes with decay still

continuing albeit at a slower rate after that (Figure 5). Overall, around 4 or 5-log decay of FMDV strain OPN was observed by Kamolsiripichaiporn *et al.* (2007) over the 2,200 s (37 min) for which they ran the experiment.

Figure 5: Thermal inactivation of FMDV strain OPN at 50°C showing the tailing effect. Figure from Kamolsiripichaiporn *et al.* (2007)

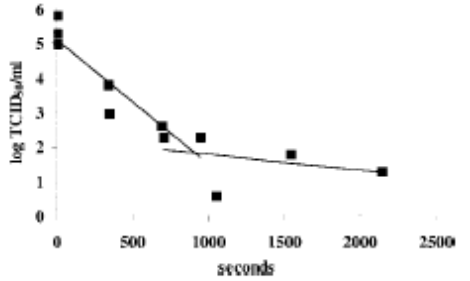


FIG. 3. Thermal inactivation of FMDV strain OPN in PBS at 50°C. The inactivation curve shows the tailing effect (biphasic curve).

At 50°C, the decimal reduction time was 1,275 seconds (22 minutes) for the ASI strain of FMDV (Kamolsiripichaiporn *et al.* 2007). Turner and Burton (1997) cite experiments where aerobic treatment processes as low as 40°C completely inactivated several animal viruses with starting titres of 10^7 to 10^8 IU. FMDV was undetectable after aeration at a pH of 8 at 50°C for 48 h.

Mateo *et al.* (2007) attempted to detect and select thermostable variants from different FMDV populations to explore whether FMDV may accept a substantial increase in thermostability without compromising its infectivity. The results indicate that the presence of thermostable virus variants – even in small proportions – is not a general feature of FMDV quasispecies. This suggests that no substantial increase in the thermostability of FMDV may readily occur without a negative effect on viral function. This is an important experimental finding for composting security and suggests that composting could not select for new thermoresistant variants.

There is some evidence that FMDV is inactivated by temperature less rapidly in meat compared with aqueous media and slurries (Donaldson et al. 2011). This is based on data from Masana *et al.* (1995), although the lowest temperature studied was 63°C at which a >4.1-log reduction was observed in 1.5 h (note the total time in the water bath was 4.73 h). Of interest is the finding that 6 of 6 cattle were infected by lymph node suspensions treated at 71°C for 1 h (total time in water bath 4.23 h), although a >4.1-log reduction in titre was achieved. Importantly, no cattle were infected for temperature/time combinations of 71°C/10.66 h, 75°C/4.00 h and 75°C/5.75 h. It should be noted that for the purpose of risk assessment it is the net reduction in infectivity which is important, i.e. the fact that >4.1-log of $TCID_{50}$ units are removed. These are for results from a single study of FMDV strain O1 Campos in beef muscle (Masana *et al.* 1995). For this reason, a sensitivity analysis is performed with an inactivation rate of just 1.92-logs in the "hot" part of the compost (Section 9.6.2). This is based on the inactivation of FMDV observed at 55°C after 10 min (Table 4-1), as it is the lowest reported in the literature, although no doubt greater inactivations would have been observed if the experiment had been continued beyond 10 min.

4.3.2 Classical Swine Fever Virus

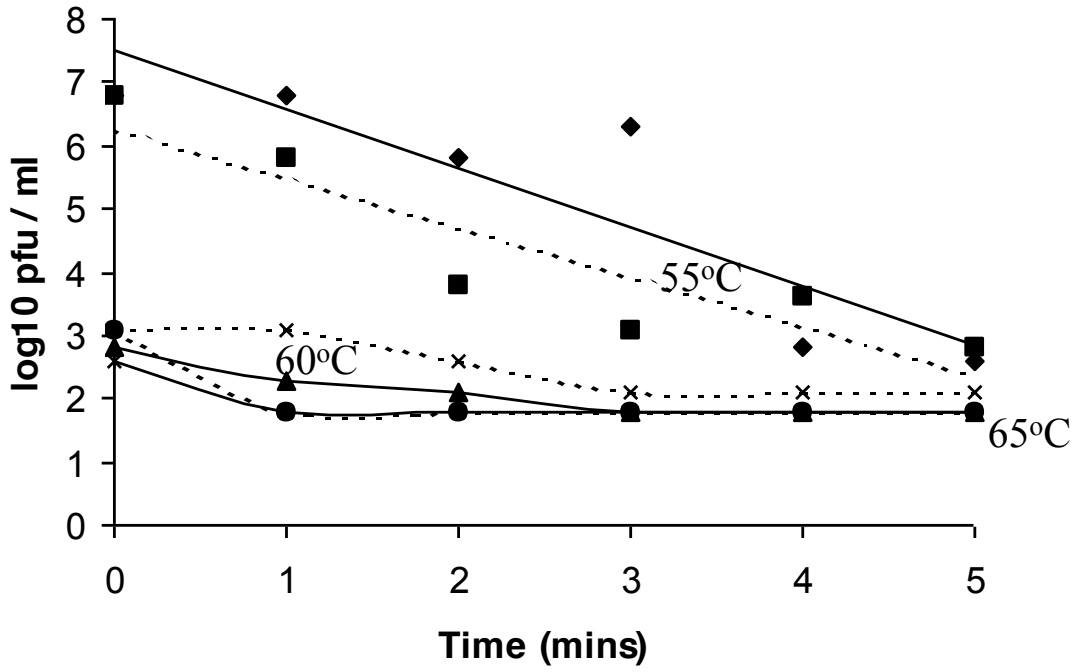
The heat inactivations of CSFV in pig slurry and culture medium at three temperatures are plotted in Figure 6 (data from Turner *et al.*, 2000). A destruction of over 4-logs in 5 minutes was reported for CSFV at 55°C (Table 4-2), with greater destructions apparent at higher temperatures.

Table 4-2: Net destruction of CSFV over 10 minutes (data from Turner *et al.,* 2000)

Temperature	0 minutes	10 minutes	Net destruction	log net destruction
55°C	6,309,573	514	12262	4.1
60°C	10,000,000	94	105826	5.0
65°C	10,000,000	63	158489	5.2

Assumes starting titre for experiments at 60°C and 65°C is 7.0 log. Data averaged for culture medium and pig slurry counts

Figure 6: Laboratory scale thermal treatment of Classical Swine Fever Virus incubated at different temperatures with time in pig slurry (solid line) and Glasgow Eagles medium (dashed line). Data from Turner *et al.* (2000). Starting titre for all experiments was measured at 7.0 log



4.3.3 African swine fever and swine vesicular disease

Plowright and Parker (1967) demonstrated rapid initial destructions of 7-log and 5-logs of ASFV at 56°C over periods of 60 and 90 minutes respectively. The rapid initial inactivation was followed by a period of slower decline in infectivity. Turner *et al.* (1999) designed a pilot plant to study the thermal inactivation of ASFV and SVDV in pig slurry. The plant maintained at least 99.99% of the slurry at the required temperature for a minimum of 5 minutes. ASFV was found to be more heat labile than SVDV. They reported that SVDV was inactivated in pig slurry to below detectable levels (at alkaline pH) at a temperature of between 50 and 55°C. ASFV was inactivated by operating the treatment plant at a temperature of 53°C at pH 8.

SVD is not inactivated at low temperatures and exhibits indefinite survival in frozen meat. Heat treatment of hams is effective if internal temperature reaches 70°C. Data for inactivation in milk gives values of 30 minutes at 56°C and 2 min at 60°C (Table 4-3). In slurry, however, a temperature of 56°C only achieved a 3-log reduction in 2 h (Table 4-4).

Table 4-3: The effect of heat on survival of SVD virus in milk (from Herniman *et al* 1973)

Time (min)	Log reduction in titre of virus				
	48°C	52°C	56°C	60°C	64°C
2	0	0.8	2.2	6.4	=>6.5
10	0.2	1.7	3.3	=>6.5	=>6.5
30	0.5	2.1	6.4	=>6.5	=>6.5
60	0.8	2.7	=>6.5	=>6.5	=>6.5
120	0.9	3.0	=>6.5	=>6.5	=>6.5

Table 4-4: The effect of heat on inactivation of SVD virus in slurry (from Herniman *et al.* 1973)

Time (min)	Log reduction in titre of virus				
	48°C	52°C	56°C	60°C	64°C
2	0	0	0.7	4.3	6.0
10	0	0	1.4	=>6.5	=>6.5
30	0.1	0.1	1.8	=>6.5	=>6.5
60	0	0.3	2.3	=>6.5	=>6.5
120	0.1	0.1	3.0	=>6.5	=>6.5

Turner and Williams (1999) demonstrated a >6.0 log destruction of SVDV after 4 h in Eagle medium Figure 7) and after 1 h in pig slurry at 50°C (Figure 8). Considerable inactivation even occurred at 40°C.

Figure 7: Thermal inactivation of SVDV in Eagle medium. Data from Turner and Williams (1999)

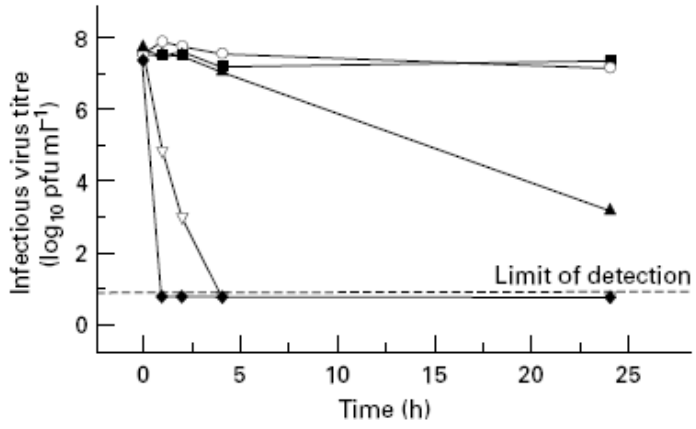


Fig. 3 Thermal inactivation of SVDV in EMEM after 24 h. Initial virus titre: $10^{7.7}$ pfu ml^{-1}. Titres that could not be detected are shown at just below the limit of detection. (■), 4 °C; (○), 22 °C; (▲), 40 °C; (▽), 50 °C; (◆), 60 °C

Figure 8: Thermal inactivation of SVDV in pig slurry. Data from Turner and Williams (1999). Note symbols for 50°C are hidden behind the 60°C

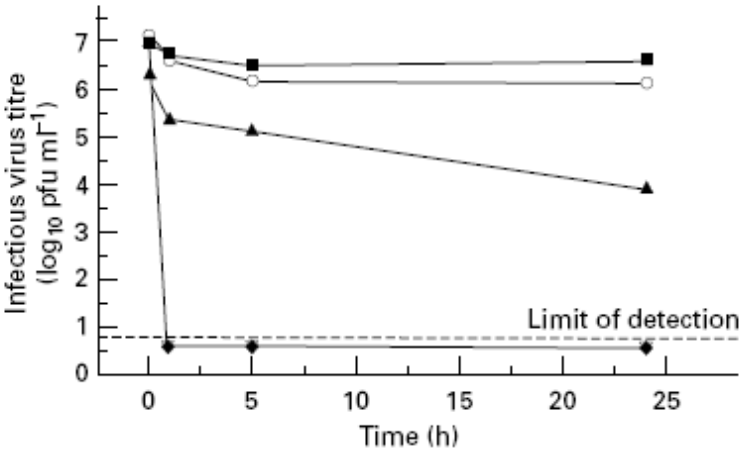


Fig. 4 Thermal inactivation of SVDV in pig slurry after 24 h. Initial virus titre: 10^7 pfu ml^{-1}. Titres that could not be detected are shown at just below the limit of detection. (■), 4 °C; (○), 22 °C; (▲), 40 °C; (▽), 50 °C; (◆), 60 °C

4.3.4 Exotic avian viruses

Senne *et al.* (1994) infected chickens with highly pathogenic avian influenza virus and adenovirus. Tissues were isolated from infected chickens and composted (in bags) with poultry carcasses. A two-stage composting process was used. At the end of the first 10 days of composting, avian influenza virus had been inactivated, as had 95% of the adenovirus. Both viruses were completely inactivated at the end of the second 10-day period of the two-stage composting process. Senne *et al.* (1994) also presented temperatures in their laboratory-scale composting process. They demonstrated differences between the different layers (i.e. alternating beds of oat straw, goat manure and chicken carcasses) in the compost. Peak temperatures for the upper layer during the first and second stages were 57.3°C and 58.3°C, but only 41.5°C and 42.8°C for the lower layer. Two-stage composting has been shown to be effective in destroying the viruses of Newcastle disease and infectious bursal disease (see Murphy (1990) in Senne *et al.* 1994).

4.3.5 Faecal bacterial pathogens

Lung *et al.* (2001) spiked *E. coli* O157:H7 and *Salmonella* Enteritidis into manure to determine the effect of a bench-scale composting system on their survival. At 45°C, a 7-log reduction of *E. coli* O157:H7 was observed after 72 h. For *Salmonella*, a 7-log reduction occurred in 48 h at 45°C. At room temperature, the composting process had no effect. Some pathogen removal data for laboratory scale compost 'windrows' are presented in Table 4-5.

Table 4-5: Effect of compost 'windrows' (at laboratory scale) on bacterial pathogens spiked into sewage sludge (data presented as log_{10} reductions, from Horan and Lowe, 2001)

Bacterium	55°C / 4 h	40°C / 5 d
E. coli	>6.2	>6.18
Listeria monocytogenes	2.5	3.2
Campylobacter jejuni	>5.7	>5.7
Salmonella Senftenberg	2.1	2.4
Salmonella Enteritidis	>5.7	>5.7
Salmonella Dublin	>5.6	>5.6

Tiquia *et al.* (1998) concluded that temperature was the main factor affecting the elimination of salmonellas in windrow composting of pig manure. However, their data question the efficiency of windrows. They demonstrated a decrease in number of faecal coliform numbers from 5-log to 2.27-logs over 91 days. This is only a 2.73-log decrease. Indeed, over the first 21 days, faecal coliform counts dropped by less than 1-log, despite the temperature being over 60°C. Furthermore, faecal streptococci numbers remained virtually unchanged at around 2.4 to 2.1-log over the 91 day period of the windrow experiment. In contrast, Deportes *et al.* (1998) reported a >7-log destruction of total and faecal coliforms in municipal solid waste (MSW) by a windrow composting process over 14 days. Counts of total streptococci decreased by 5-logs.

Hanajima *et al.* (2006) proposed that *E. coli* reduction during composting was related to the initial amount of easily digestible carbon as determined by the biological oxygen demand (BOD). They demonstrated that BOD values of >166 mg O_2/DM g brought about significant *E. coli* reduction in cattle faeces. The addition of organic waste (tofu residue, rice bran, rapeseed meal, or garbage) brought about maximum temperatures of >55ºC and reduced *E. coli* from 10^6 to $<10^2$ CFU/g wet weight after 7 days.

Chesshire and Ferry (2006) have reported on survival of faecal indicator bacteria in manure which was composted at operational scale. The results show a maximum log reduction of 5.7 for total coliform bacteria; a log reduction of 3.8 for *E. coli*; and a log reduction of 3.1 for *Enterococci*. However, there was a wide range in FIO kill between the different farms under study, and the results also show minimum log reductions that indicate an increase in faecal indicator organisms (FIO).

Larney *et al.* (2003) reported that more than 99.9% of total coliforms and *E. coli* bacteria were eliminated in the first seven days of composting of cattle manure when average windrow temperatures ranged from 33.5ºC to 41.5ºC. Coliform reductions of 5 to 7-log were measured at 14 days. These temperatures are 14ºC to 22°C lower than the thermal kill limit of 55°C in composting guidelines in Canada and the USA. Those guidelines specify maintenance of 55°C for at least 15 days. Desiccation probably only played a minor role in coliform elimination, since water loss was low in the first 7 days of composting. The total aerobic heterotrophic plate count, however, remained high throughout the composting period, and their competition for nutrients may have caused an antagonistic effect on pathogens.

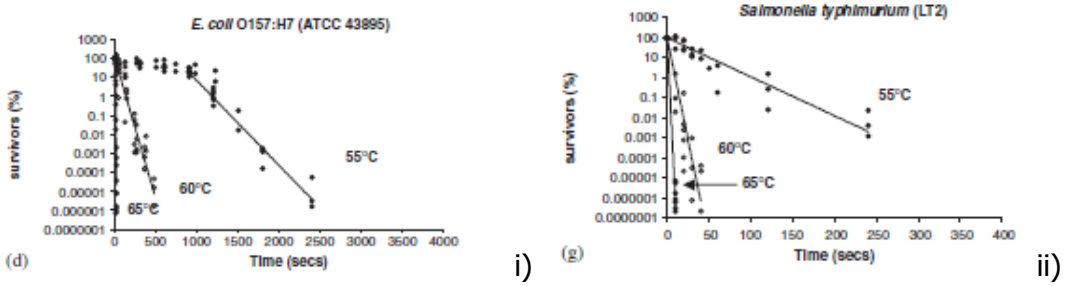
Briancesco *et al.* (2008) found differences in the removal of different microorganisms between different composting plants and feedstock compositions. Compost obtained from feedstock containing sewage sludge had a 'better' hygienic quality compared to compost derived from green discards and municipal solid waste.

Shepherd *et al.* (2007) conducted studies to determine the fate of *E. coli* O157:H7 in dairy manure-based compost in a field setting. Temperature stratification was observed, which

may have led to the variability in pathogen reduction. In some places 5-log reductions were observed, while *E. coli* O157:H7 survived at the heap's surface for up to 4 months. They conclude that composting, with periodic heap turning, can be a practical approach to inactivating *E. coli* O157:H7 in cattle wastes on farms, but note that they survive for months at the heap's surface when compost heaps are not turned.

Wichuk and McCartney (2007) have reviewed the effectiveness of the current time-temperature regulations according to the North American regulatory bodies on pathogen inactivation during composting. The North American regulatory bodies (the US Environmental Protection Agency and Canadian Council of Ministers of the Environment) specify composting at temperatures greater than 55ºC for at least three consecutive days.

Figure 9: Inactivation of *E. coli* O157:H7 (i) and *Salmonella* Typhimurium (ii) at temperatures of 55°C, 60°C to 65°C in water. Data from Spinks *et al.* (2006)



Spinks *et al.* (2006) demonstrate an 8 log removal of *E. coli* O157:H7 at 55°C in water in 42 minutes and a 4-5-log reduction of *Salmonella* Typhimurium (LT2) at 55°C in 4 minutes. Moreover, according to Spinks *et al.* (2006) 9-log inactivation occurred in under a minute at 60°C for *Salmonella* Typhimurium (LT2)

The findings here are consistent with the assumptions of the original risk assessment regarding destruction of *Enterobacteriaceae* by composting.

4.3.6 Conclusions

It is concluded here that a 6-log reduction in the exotic viruses and faecal bacterial pathogens can be achieved after 4 h at 56°C.

While a 6-log reduction has been demonstrated for SVDV (refer to Table 4-3 and Turner and Williams 1999) and AFSV (Plowright and Parker 1967) experiments have not used sufficiently high starting titres or have not been run for long enough time periods to demonstrate 6-log reduction for CSFV and FMDV (Turner and Williams 1999; Turner *et al.* 2000). However, this is not evidence that 6-log inactivation does not occur.

The maximum inactivation demonstrated for CSFV is >4.2-log after 5 min at 55°C (Turner *et al.* 1999) and >4.0 or >5.0-log for FMDV after 36 min at 50°C (Kamolsiripichaiporn *et al.* 2007). However, it is assumed that given the full 4h at 56°C that 6-log reduction would occur. This is the minimum condition at the centre of a 40 cm diameter particle. The bulk medium is at 60°C for 48h, where greater inactivation would be achieved. Furthermore, the composting process is repeated for the "meat fraction" and the bulk medium would therefore experience 60°C for 96 h. Due to the uncertainties, a sensitivity analysis has been performed to examine the impacts of lesser log-reductions on FMDV (Section 9.6.2).

4.4 Estimating net pathogen destruction by composting: By-pass of in-vessel composting stages

In terms of estimating the net inactivation by treatment barriers, defining the degree of both "within-batch" (spatial variation) and "between-batch" (temporal variation) is important (Gale 2005a). For example, a treatment process which typically provided a 6-log destruction, would only yield a 2-log reduction if it failed completely with a frequency of 1% (Table 4-6). Thus, if 1% of the raw material by-passed the process, it would effectively erase 4 of the 6-log removal. This illustrates the importance of minimizing "within-batch" and "between-batch" variation in the composting process. The effect of by-pass on the net inactivation achieved is less significant for the less efficient processes. Thus, 1% by-pass of a 2-log process only halves the net removal; from 100-fold to 50-fold (Table 4-6).

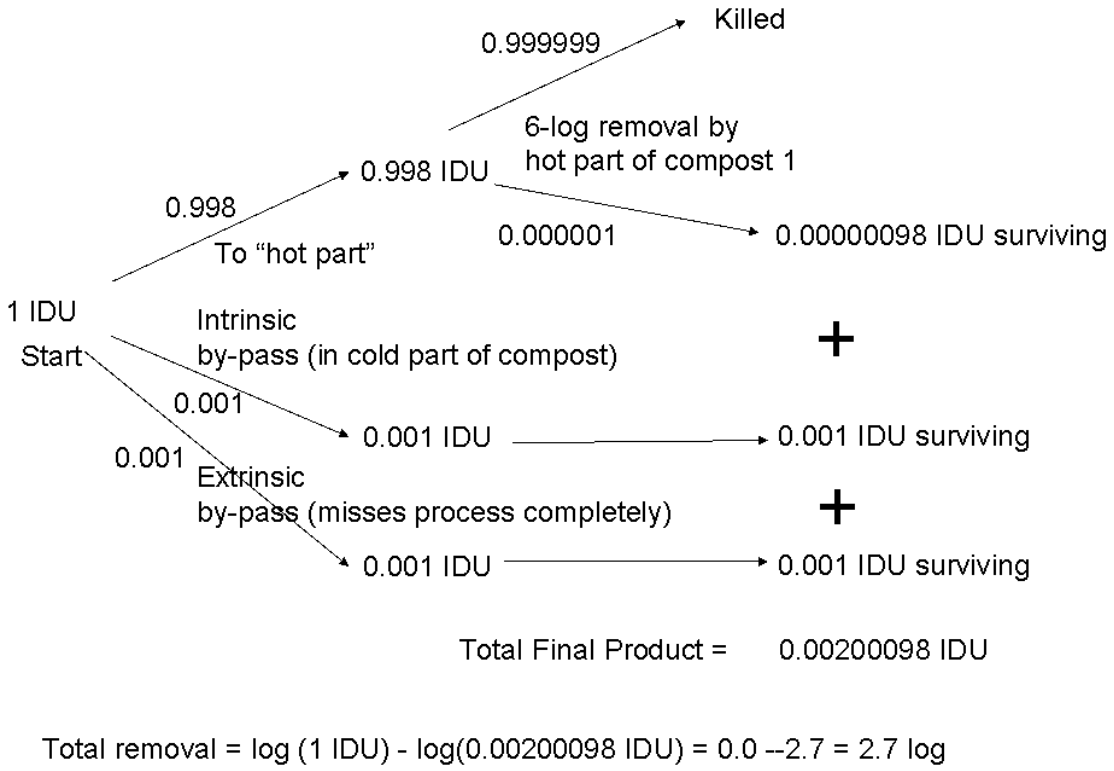
The key point for risk assessment, is that it is not so much whether a 2, 3, 4, 5, 6, or even 7-log destruction of pathogens can be achieved in the hot part of composting under laboratory conditions, but how much material by-passes the hot part of the compost process at operational scale. A worked example of by-pass is presented in Figure 10.

By-pass of each composting process is considered separately. It should be noted that this by-pass is either intrinsic or extrinsic. Intrinsic by-pass represents a cool part of the compost heap not circulating, while extrinsic by-pass represents, for example, a wheelbarrow not being cleaned out after removing raw material and subsequently used to carry final product. In Figure 10 both intrinsic and extrinsic by-pass of an "in-vessel" compost system are shown. There are no data to demonstrate the level of by-pass of an operational compost process. Indeed to demonstrate <0.2% by-pass would involve demonstrating that >99.8% of the input material is in the hot part all day every day. One approach to judging by-pass is to consider the volumes. Thus assuming 100,000 kg are processed by composting, 0.2% (w/w) represents 200 kg. At a bulk density of 0.6t/m^3 (600 kg/m^3) that is a volume of material occupying 0.33 m^3, i.e. 69 x 69 x 69 cm in a cube of length, wide and height of 5.5 m. This can be visualized as a "couple of wheel barrow loads" in a 5.5 m cube. Avoiding such occurrences should be readily achievable using HACCP. For the purposes of modelling, 0.1% of the by-pass is assumed to be intrinsic and 0.1% extrinsic in Figure 10.

0.2% by-pass means that a portion representing 0.002 of the infectivity is untreated and finds its way into the next process stage, together with the 10^{-6} of the infectivity surviving the hot part. Thus the total infectivity in the final product is 0.002001, representing a 1/002001 = 500-fold reduction. In log terms, this is 2.7-log. Therefore in this risk assessment, each "in vessel" composting step is modelled as causing a 2.7 log inactivation of the viral, bacterial and protozoan pathogens (Figure 10).

It should be noted that the regulations require all material to achieve the required time/temperature combination, having first been reduced to the required particle size. Composting processes are validated by APHA (Animal and Plant Health Agency) on this basis, and twelve consecutive tests of the final compost must pass the required Salmonella and either *E. coli* or Enterococcaceae limits as part of the validation process (APHA, 2014).

Figure 10: A 0.2% by-pass of a process which achieves a 6-log removal gives a net 2.7-log removal. IDU = infectious dose unit



Total removal = log (1 IDU) - log(0.00200098 IDU) = 0.0 --2.7 = 2.7 log

By-pass may be minimized by ensuring good plant operational efficiency. The role of the Animal Health and Veterinary Laboratories Agency as the competent authority for validating and monitoring Animal By-Products Regulations (2011) compliant compost facilities is key in ensuring that by-pass is minimized. The examples in Table 4-6 and Figure 10 illustrate the impact of by-pass on elevating the risks, and reinforce the need for the composting sector to continue to adopt and implement robust HACCP plans. That the bulk of the composting material will consistently exceed the time/temperature parameters modelled in this risk assessment should not be taken as an opportunity to implement poor practice in operational-scale composting.

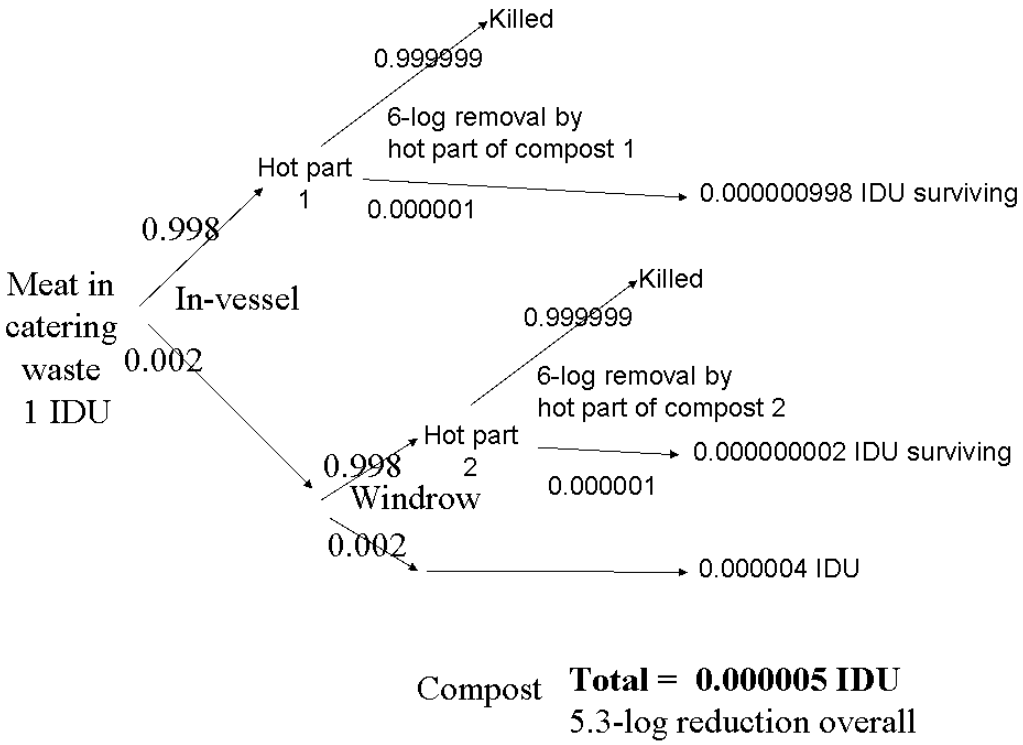
Table 4-6: Effect of by-pass (e.g. from cool spaces in a composting vessel) on the net destruction of pathogens

Treatment conditions (and operational efficiency in parentheses)	% by-passing treatment and receiving 0-log destruction (calculated as 100 – operational efficiency)	Arithmetic Mean Survival	Net log destruction
0-log destruction (100%)	0%	1	0.00
2-log destruction (100%)	0%	0.01	2.00
6-log destruction (100%)	0%	10^{-6}	6.00
6-log destruction (99%)	1%	0.01	2.00
2-log destruction (99%)	1%	0.02	1.70
1-log destruction (100%)	0%	0.10	1.00
1-log destruction (99%)	1%	0.11	0.96

4.5 The hot part of composting must achieve 6-log reduction of the pathogen

The meat fraction is composted in two stages. Each stage is modelled as having 0.2% by-pass, as set out in Figure 10. To achieve high total kills (5.3-log), the hot part must be able to achieve at least 6-log inactivation.

Figure 11: The "hot" part of composting must be able to achieve 6-log reduction of the pathogen for two stage composting with 0.2% by-pass of each stage to achieve a 5.3-log reduction of the pathogen. IDU = infectious dose unit



4.6 Windrows

Stenbro-Olsen *et al.* (1995) studied the patterns of temperature development and distribution of temperature in windrows used for composting of municipal green waste over a period of 25 days. They concluded "these plots revealed a sequential pattern of temperature development which indicated that the vast majority of the windrows' contents were maintained at temperatures in excess of 65ºC for periods of four to five days".

Joshua *et al.* (1998) studied the temperature profiles in a green waste windrow processing system. The highest and lowest temperatures recorded were 72.8°C and 17.6°C respectively. The temperature distributions are presented in Table 4-7. They concluded that predominantly thermophilic conditions were maintained in the windrows throughout processing and virtually all material was subjected to the commonly recognized 55°C for three days that ensures the destruction of potential pathogens in organic material.

Table 4-7: Percentage cross sectional area of windrows reaching certain temperatures throughout green waste composting (Joshua *et al.* 1998)

Day	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
<20°C	9	2	0	3	0	4	0	0	6	4	0	0	7	5	0
20-30°C	18	5	4	6	3	6	11	3	8	6	<1	<1	7	7	3
30-40°C	12	2	2	7	6	42	89	7	6	4	36	36	14	64	66
40-50°C	7	5	5	6	7	45	0	10	5	6	29	36	52	23	31
50-55°C	9	4	12	10	22	2	0	13	5	17	12	21	19	<1	0
>55°C	42	78	76	68	62	>1	0	63	68	63	22	6	<1	0	0
>70°C	3	4	<1	0	0	0	0	4	2	0	0	0	0	0	0

4.6.1 By-pass of windrows - modelling the effect of windrow mixing and turning

Haug (1993) in "The Practical Handbook of Compost Engineering" cites an equation for the thermal inactivation of pathogens after N turns of the pile. The number of pathogens surviving n_i is given by:

Equation 2 $$n_i = n_0\left[f_l + f_h e^{(-k_d \Delta t)}\right]^N$$

where $f_l + f_h = 1$, and f_l is the fraction of the composting material in the low temperature zone and f_h is the fraction of the composting material in the high temperature zone.

To model the effect of N on the rate of pathogen destruction, Equation 2 is simplified by assuming that a given proportion (π) of the pathogens survive in the high temperature zone after a time t. Thus:

Equation 3 $$n_i = n_0\left[f_l + f_h \pi\right]^N$$

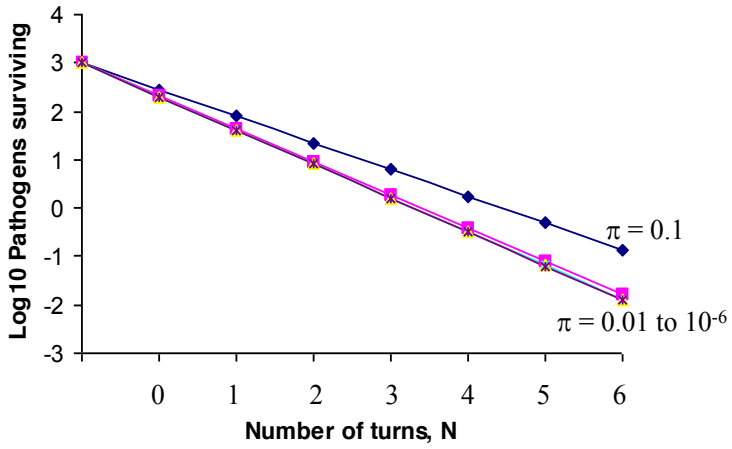
Setting values of π to 0.1, 0.01, 0.001, 10^{-4} and 10^{-6} would be equivalent to allowing for 1-log, 2-log, 3-log, 4-log and 6-log destructions in the high temperature zone over the time interval between turns. On the basis that about 80% of the material (Table 4-7) is in the high temperature zone between turns (i.e. $f_h = 0.8$), the numbers of pathogen surviving after each turn are calculated according to Equation 3 in Table 4-8. These are plotted in Figure 12. It is apparent that for destructions of 2-logs or more in the high temperature zone, then the net destruction is controlled by the number of turns.

Table 4-8: Log counts of pathogen remaining in a windrow after *N* turns. Assumes there are 1,000 (3-log) counts in the windrow at t = 0. Model allows for different degrees of destruction (π) in the high temperature zone and assumes that 80% of the material is in the high temperature zone (f_h = 0.8 in Equation 3)

Number of turns of windrow (N)	Proportion (π) of pathogens surviving the high temperature zone				
	0.1	0.01	0.001	0.0001	0.000001
(Start (t=0))	(3)	(3)	(3)	(3)	(3)
0	2.447158	2.318063	2.302764	2.301204	2.301032
1	1.894316	1.636127	1.605527	1.602407	1.602063
2	1.341474	0.95419	0.908291	0.903611	0.903095
3	0.788632	0.272253	0.211055	0.204815	0.204127
4	0.23579	-0.40968	-0.48618	-0.49398	-0.49484
5	-0.31705	-1.09162	-1.18342	-1.19278	-1.19381
6	-0.86989	-1.77356	-1.88065	-1.89157	-1.89278

Assuming the proportion (π) of pathogens surviving in the hot portion is 0.01 or less, then 3 turns of the windrow will achieve a net destruction of >2.7 logs (Table 4-8).

Figure 12: Effect of number of turns of a windrow on pathogen survival. Model assumes 80% of pathogens are in a high temperature zone where proportion (π) of pathogens survive the high temperature



For a pathogen where just 90% is destroyed in the high temperature zone, three turns are required to bring about a >2-log reduction (Table 4-8). Thus three turns reduces the counts from 3-logs to 0.79-logs.

In conclusion, the windrows should be turned at least three times to achieve a >2.7-log destruction (Gale 2002).

4.7 Particle size and the time/temperature criterion for composting and biogas

Haug (1993) estimated the heat transfer times into spherical compost particles. These are shown in Table 4-9.

Table 4-9: Estimated heat transfer times into spherical compost particles (Table 5.5 from Haug 1993)

Particle diameter (cm)	Time to reach $(T - T_0)/(T_1 - T_0) = 0.9$ (h)
2	0.1
20	10
40	40
100	250
200	1,000

In Table 4-9, T_0 is the temperature throughout the sphere as it goes into the compost (at time t = 0). T_1 is the temperature surrounding the sphere in the composting system. T is the desired temperature at the centre of the sphere for pathogen destruction. To obtain a value of T = 56°C (as required for SVDV) at the centre of the sphere, requires T_1 to be 60°C, if T_0 is 20°C. Thus according to

Equation 4 $$(T - T_0)/(T_1 - T_0) = 0.9$$

if T_1 = 60°C, the for T to reach 56°C requires a time of 40 h for a sphere of 40 cm diameter (Table 4-9). Thus for all parts to experience at least 56°C for 4 h requires 40 h + 4 h = 44 h; hence the original proposal that composting conditions achieve 60°C for 2 days in each of two composting stages.

Thus, consider a leg of pork with a bone in from an SVDV-infected pig. Assuming the diameter is 40 cm, then the external temperature would need to be 60°C for 40h to get the bone in the centre of the leg up to 56°C. Since uncooked legs of meat (with bone-in) could be disposed of to catering waste e.g. after a freezer failure, it is appropriate to define composting conditions that can deal with such challenges. Of course, this does not reflect commercial composting practice, where, to achieve consistent aeration of the composting mass, incoming food waste is shredded and mixed with bulking agents such as shredded garden (botanical) wastes. This has the effect of reducing any food particles to maximum diameters of a few centimetres, ensuring that the required core temperatures are achieved within hours, rather than days.

5.0 A credit system for modelling the barriers in composting of catering waste

5.1 Source separation and definitions

Source separation is the action of the waste producer to keep certain parts of their waste (which is required for composting) separate from the residual waste stream.

The non-meat fraction is material for composting which should be free of the majority of meat because the waste producer has been instructed to exclude meat at the source of the waste (e.g. the kitchen where food is prepared).

5.2 Composting the source-separated non-meat fraction

The non-meat fraction could contain meat "by accident" due to inefficient source separation. The risk assessment is based on a credit system such that a 4.7-log (i.e. a 50,000-fold) reduction occurs through (1) meat exclusion at source, (2) composting, and (3) stock-piling or storage of the resulting compost prior to use. Meat exclusion at source is assumed to be 90% efficient, i.e. the source-separated non-meat waste stream contains 10% of the total uncooked meat discarded to catering waste. The value of 10% is assumed to be a worst-case assumption, and represents a 1 log reduction. The barriers are set out in Table 5-1.

Table 5-1: A credit system for the barriers for composting the non-meat fraction

Process (Barrier)	Credits (log-reduction)
Meat exclusion at source	1.0
Composting process*	2.7
Stockpiling (18 days)	1.0
Total	4.7

*Windrow or "in-vessel"

Catering waste that is declared to be 'non-meat' must be composted in a process authorized by APHA (the competent body in the UK for implementation of the Animal By-Products Regulations). This risk assessment assumes that 10% of all meat modelled as being sent for composting will actually be processed in this 'non-meat' catering waste route. This requires the material to be subjected to a standard composting process (in vessel or windrow with three turns), followed by a compulsory period of stockpiling.

5.3 Composting the source-separated meat fraction

The meat fraction of catering waste must be composted by a two barrier process. First an in-vessel process in which all bar <0.2% reaches 60°C for 2 days, and secondly a turned windrow. The windrow need not be housed as it is a secondary barrier, although in practice many in the industry opt for two similar, in-vessel barriers. The barriers are set out in Table 5-2.

Table 5-2: A credit system for the barriers for composting the meat fraction

Process (Barrier)	Credits (log-reduction)
Meat exclusion at source	0.0
In-vessel composting	2.7
Windrow – 3 turns	2.7
Total	5.4*

*5.4-log if complete inactivation in hot part; actually 5.3-log reduction based on 6-log inactivation in hot part (Section 4.5)

The purpose of the second barrier is to ensure that there is only a 0.002 probability that the 0.2% in the "cold part" from the first composting stage is in the "cold" part in the second stage. Thus the probability that the same pathogen is in the "cold" part in both composting stages is 0.002 x 0.002 = 0.000004.

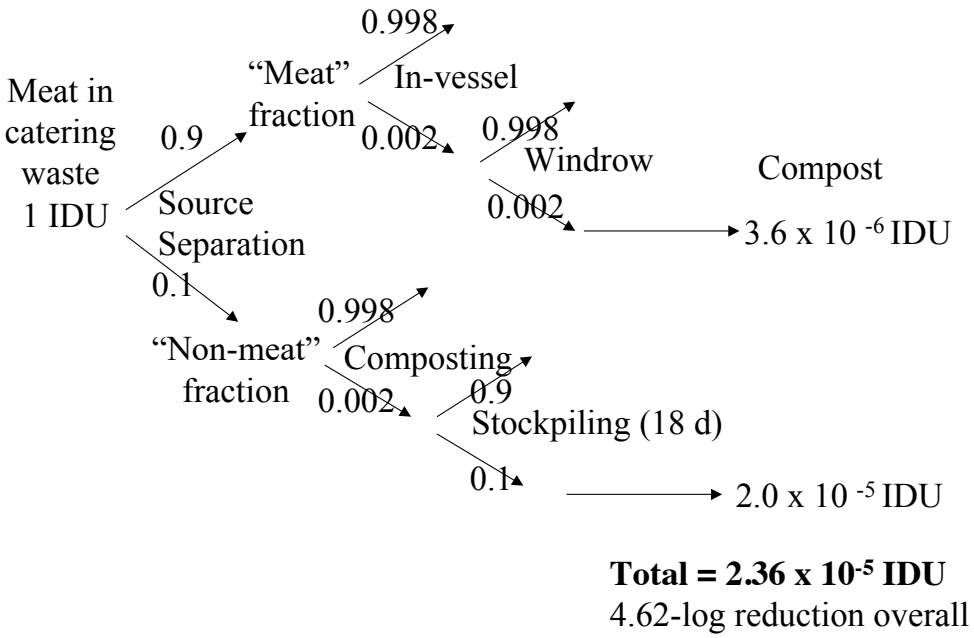
Thus, the 90% of meat that is processed via the 'meat' route is subjected to two composting stages (the first a compulsory 'in vessel' or enclosed stage, and the second a windrow turned a minimum of three times). In practice, many compost producers choose to use enclose both stages. No minimum period of stockpiling is required for such composts derived from catering waste containing meat. However, if composts are to be accredited to the UK's compost quality specification (Publicly Available Specification (PAS) 100), then a stockpiling period of several weeks is usually required to ensure that the compost meets the required degree of stability prior to despatch.

6.0 Composting – putting the barriers together

This section sets out the treatment pathways for all the uncooked meat (i.e. both meat and non-meat fractions) discarded to catering waste as event trees.

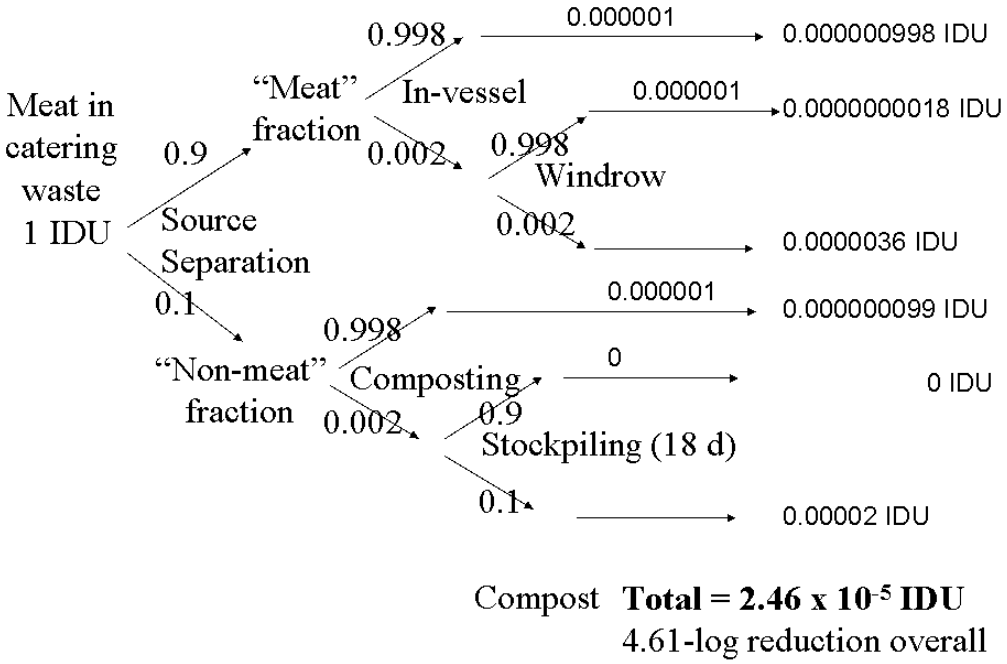
Building on the event tree approach in Figure 10 and including the credit systems set out in Table 5-1 and Table 5-2 for the non-meat and meat fractions, respectively, then overall the treatment process including composting and storage achieves a 4.62-log inactivation of pathogens in meat (Figure 13). This is calculated by following the infectivity through the event tree, which shows that 0.0000236 IDU survive from each IDU going in to the treatment process (Figure 13).

Figure 13: Composting achieves a 4.62-log reduction assuming complete inactivation in the "hot" part. IDU = infectious dose unit



The 4.62-log reduction in Figure 13 is based on complete inactivation of pathogens in the hot part of the composting process. This is a good assumption for faecal bacteria where 8-log and 9-log inactivations have been demonstrated at 55 – 60°C (see Section 4.3.5). However, inactivations of >6.0 log have not been empirically demonstrated for most of the exotic viruses (Section 4.3) and as a worst case, it is assumed that only 6-log inactivation occurs in the hot part, rather than complete inactivation (Section 4.3.6). Allowing for an inactivation of 6-log in the hot part of composting gives a more complex event tree (Figure 14) from which it may be calculated that the overall reduction is 4.61-log.

Figure 14: Composting achieves a 4.61-log reduction assuming only 6-log inactivation in the "hot" part



6.1 Removing the windrow (2nd barrier) and 18 d stockpiling stages from composting increases the risks by 83-fold

Removing the second composting stage for the meat fraction reduces the net pathogen destruction from 4.62-logs (Figure 13) to 2.73-logs (Figure 15). If this second composting stage is omitted then there is little point in applying the 18 d stockpiling stage for the non-meat compost, since omission of this stage reduces the net destruction by just 0.3-logs (2-fold) to 2.70-logs (Figure 16).

Figure 15: Composting - Failure or omission of the windrow 2nd stage for the meat fraction reduces the net destruction of composting to 2.73-logs

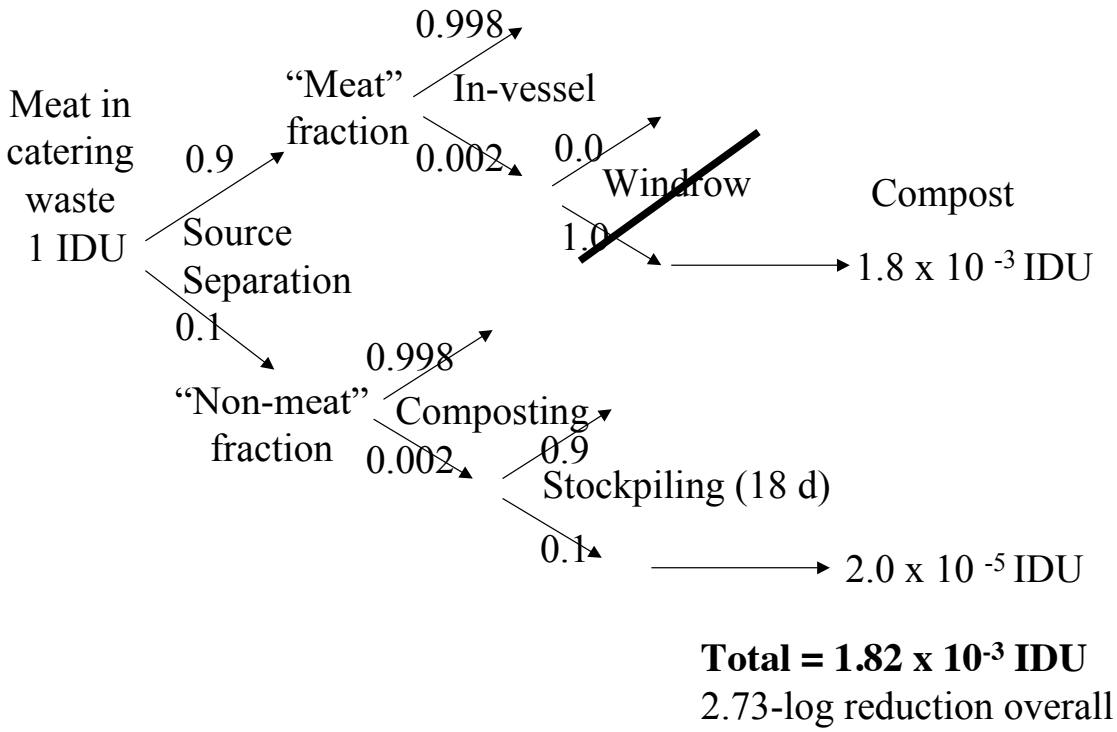
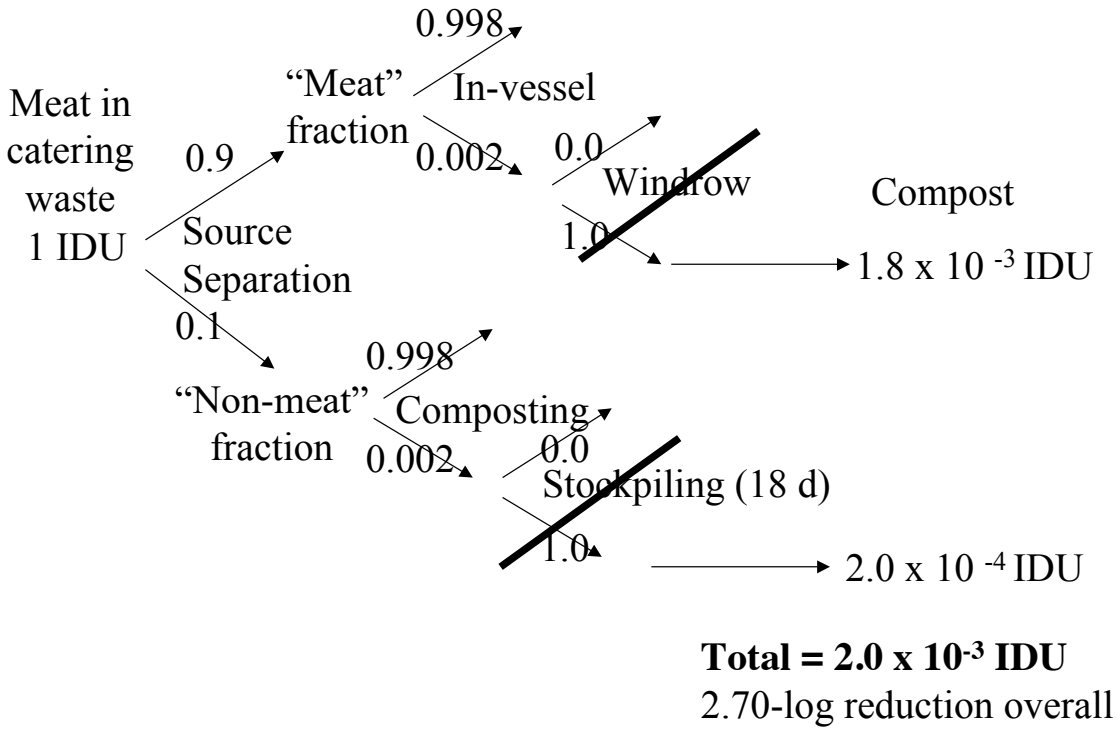


Figure 16: Composting - Stockpiling of the non-meat fraction is of little value if there is no windrow second stage for the meat fraction



6.2 Direct feeding of meat to livestock

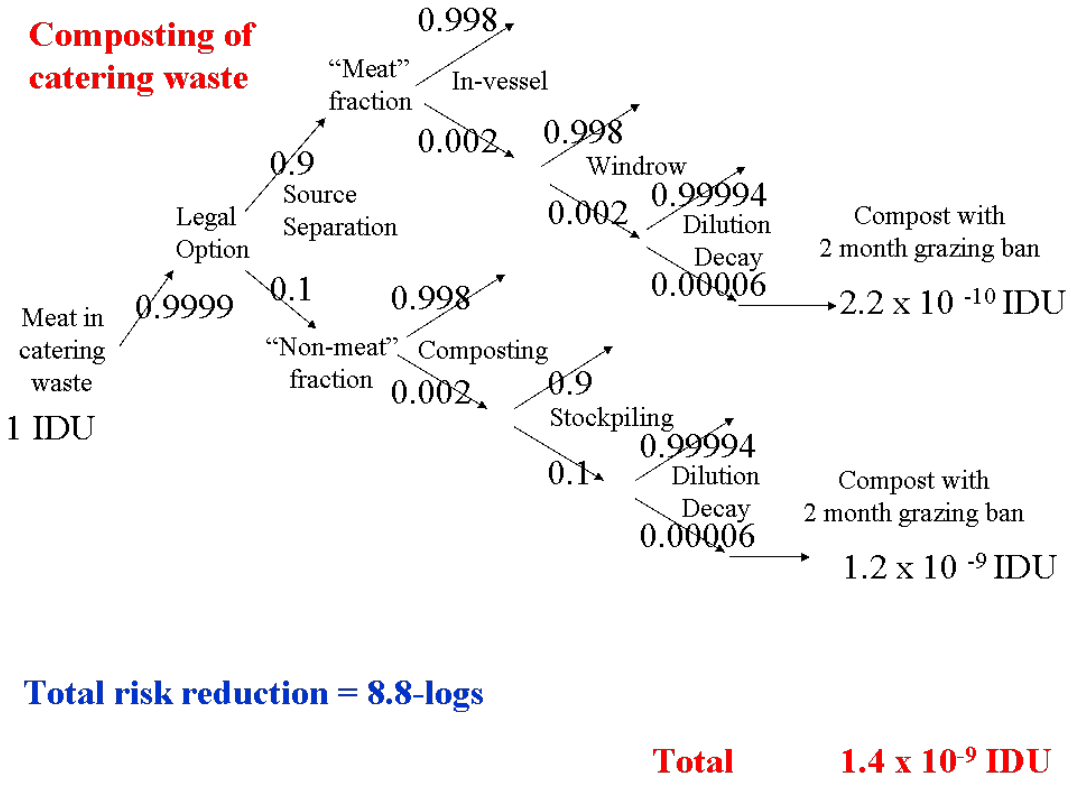
Direct feeding of meat to livestock is discussed here because of the possibility of backyard poultry owners feeding raw meat and kitchen scraps to their poultry, and the potential risks from NDV (Section 8.5) and AIV (Section 12.0) that could arise from this practice. Direct feeding of meat to livestock is illegal and may be represented as an extrinsic by-pass of the system.

Overall, two barrier composting followed by a 2 month grazing ban after tilling into a depth of 10 cm achieves a net reduction in risk of 8.8-log (Figure 17). The sequence of event trees in Figure 17 and Figure 18 displays how illegal feeding of 0.01% of discarded meat to livestock reduces the overall risk reduction by the compost and environmental barriers from 8.8-log to 4.0-log.

Indeed with feeding of just 0.01% of discarded meat directly to livestock, there is little value in composting the bulk (remaining 99.99%) of the meat. Thus, if there is 0.01% illegal feeding, removal of composting completely reduces the overall reduction by 0.2-log from 4.0-log to 3.8-log (see Figure 18 and Figure 19). A 0.2-log difference represents a $10^{0.2}$ or 1.6-fold reduction in risk.

The startling conclusion therefore is that if 0.01% of meat is fed directly to livestock, then the remaining regulated two barrier composting process in GB achieves a net of reduction in risk of just 1.6-fold. Thus, feeding just 0.01% of meat in catering waste to livestock virtually cancels out the benefits of two barrier composting of catering waste.

Figure 17: All barriers together achieve an 8.8-log reduction in risk



Total risk reduction = 8.8-logs

Total 1.4 x 10^{-9} IDU

Figure 18: Direct feeding of just 0.01% of meat to livestock means all barriers together achieve just a 4.0-log inactivation

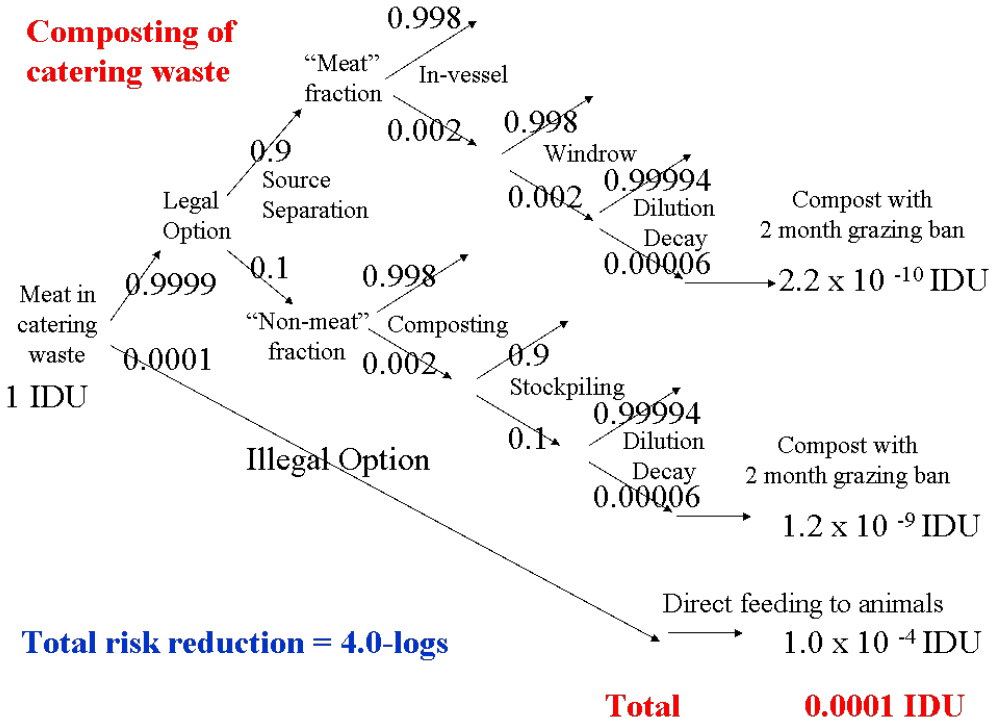
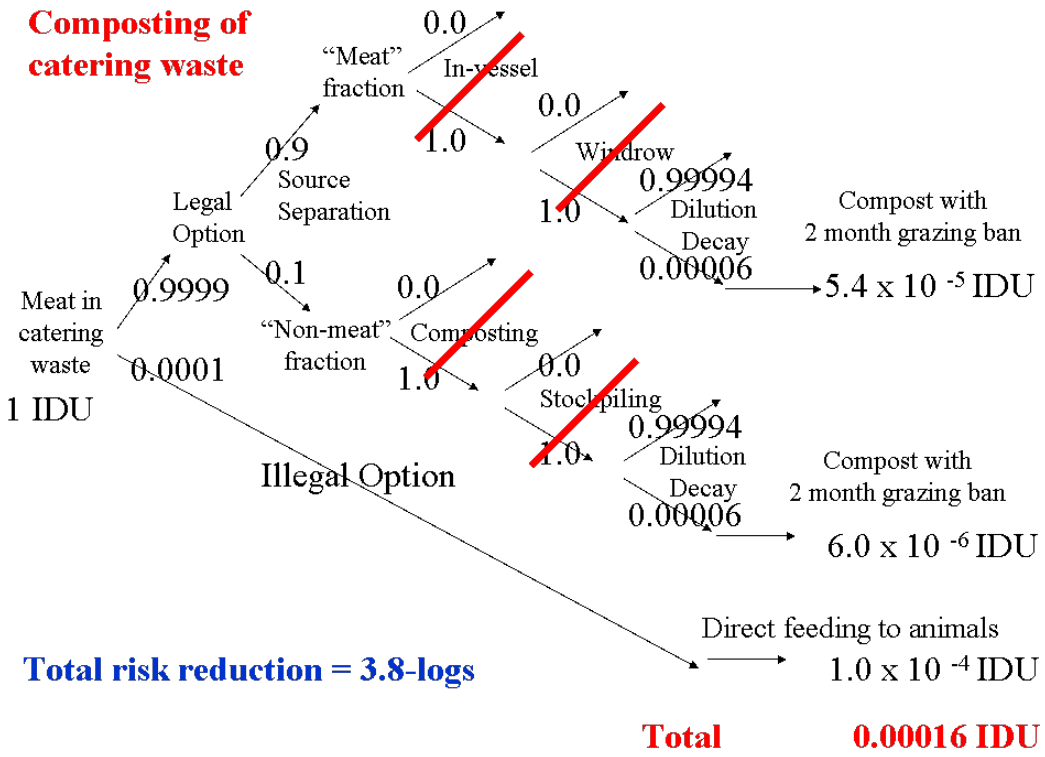


Figure 19: Direct feeding of just 0.01% of meat to livestock means all composting barriers are redundant (assuming compost has been tilled into 10 cm and there is 2 month grazing ban)



7.0 Method for calculation of risks to livestock from pathogens in catering waste-derived compost

Microbial risk assessment (MRA) is a scientific tool that can be used to evaluate the level of exposure and the subsequent risk to animal health due to a specific pathogen. In this report quantitative risk assessments are presented for BSE, scrapie, CSFV, ASFV, FMDV, SVDV, NDV, AIV and *T. gondii*, thereby providing a numerical output for the estimated magnitude of risk.

Rather than quantitative risk assessment, an overview of the risks has been brought together for some faecal-oral bacteria and for *Clostridium botulinum*, and brief reviews of the data available for possible future risk assessment have been conducted for porcine circovirus, porcine parvovirus and MRSA.

The quantitative risk assessments focus on GB. This is because available estimates for amounts of illegally imported meat infected with ASFV, CSFV, SVDV and FMDV are for GB (Hartnett *et al.* 2004) as are estimates of the amount of scrapie and BSE infectivity entering the food chain (Adkin *et al.* 2010). To correspond, therefore, the theoretical total amount of compost produced annually from catering waste for GB is used. This is 3,913,200 tonnes dry weight (Section 2.4.1) for England, Scotland and Wales, and its use in the risk assessment assumes that all household food waste is captured, composted and that compost applied to agricultural land.

7.1 Summary of parameters used and differences to the 2002 risk assessment

The parameters used in the revised risk assessment are summarised in Table 7-1. Some key amendments from the original risk assessment are now considered. In the case of CSF, for example, the estimate of the amount of CSFV-infected meat illegally imported is around 800-fold lower than the 10,000-infected carcasses considered in the original risk assessment. From Table 7-1 it can be seen that for SVDV, the new estimate for the 95th percentile of the amount of illegally imported infected meat is approximately 30 million-fold lower than that used previously in Gale (2002).

Bone marrow in infected livestock contains very high viral titres of CSFV and AFSV. Unfortunately, despite intensive searching, no information has been found on the volumes of bone marrow in livestock (or even humans for that matter). The case is made in Section 2.3.1 for reducing the value of 5.46 kg used in the original risk assessment down to 0.546 kg (1% of carcass) as a worst-case.

In the original risk assessment it was assumed that compost would be diluted to a depth of 10 cm in the soil through leaching or soil disturbance during the grazing ban. Here, it is assumed (as a worst-case) that surface-applied compost is ingested undiluted, to the complete exclusion of soil, by livestock each day for a period of one year. Indeed calculations based on densities of compost and soil suggest that the volume of compost is such that at a height of 1 cm, compost would cover 61% of the field at an application rate of 20 tds/ha. This application rate is double the value of 10 tds/ha used previously.

Table 7-1: Summary of new parameters and data sources for the risk assessment, and comparison with parameters from the original risk assessment

Variable	Values from original risk assessment	Updated values based on current evidence	Source of updated values
Estimate of percentage of raw meat discarded to waste and going to compost	1%	Poultry (2.8%) Pig meat (1.39%) Beef (0.8%) Lamb (1.09%)	WRAP (2008a)
Bone marrow weight in pigs	5.46 kg (10% of carcass weight)	0.546 kg estimated as 1% (w/w) of the dressed carcass	Sellers (1971) reports bone marrow in pig femur to be 0.011 kg. Pig bone marrow in long bones estimated at 0.315 kg (Table 2-9). Below 0.546 kg total viral loading estimated in carcass is little affected by amount of bone marrow (see Section 2.3.1)
Amount of infected meat illegally imported to GB per annum	(kg / year) 620,000 (FMD) 620,000 (CSFV) 62,000 (ASFV) 620,000 (SVDV)	(kg / year) – 95th percentile 565 (FMDV) 794 (CSFV) 0.14 (ASFV) 0.021 (SVDV)	Hartnett *et al.* (2004)
Soil consumption	(kg/animal/day) 0.41 pigs Chicken – not included 0.20 sheep 0.41 cattle	Maximum values found (kg/animal/day) 0.392 pigs 0.032 chicken 0.69 sheep – 95th percentile from WRAP (2013) 1.125 cattle – 95th percentile from WRAP (2013)	Smith (1996); Hoffmann *et al.* (2002); Thornton and Abrahams 1983); Peterson *et al.* 1974; Commission of the European Communities (1996) – see Section 3.2; van der Meulen *et al.* (2008)
Compost application rates	10 tds/ha	20 tds/ha	Fertiliser Manual (2010)
Total compost produced from catering waste per year	500,000 tonnes	6,522,000 (GB) and 6,700,000 (UK) tonnes wet weight; equivalent to 3,913,200 tds (GB); 4,020,000 tds (UK)	WRAP (2008a) and assuming compost is 60% dry matter

Variable	Values from original risk assessment	Updated values based on current evidence	Source of updated values
Total area of land to which compost is applied	50,000 ha (England and Wales)	195,660 ha (GB only) of which 58.2% (113,897 ha) is grassland for grazing	Calculated as number of ha covered at 20 tds/ha by 3,913,200 tonnes (dry weight), assuming 58.2% of land is grassland (Anon 2010)
Compost dilution in soil	150-fold dilution due to leaching to 10 cm depth	Depth tilled into soil 0 cm – Surface application giving no dilution 10 cm – minimum tillage giving 75-fold dilution 25 cm – plough depth giving dilution of 187.5-fold	Based on expert and stakeholder feedback, including Prof. Brian Chambers (Pers. Comm. 2008), discussion with WRAP, Gale and Stanfield (2001)
Livestock numbers exposed to compost-treated soil	England/Wales Cattle 42,800 Pigs 33,417 Sheep 157,100	GB, using England livestock densities for Scotland and Wales (see below) Cattle 181,096 Pigs 318,912 Sheep 580,875	Based on maximum livestock densities (see below) in England from Defra (2005, 2006a, 2006b) and application of compost to 195,660 ha in GB of which 58.2% used for grazing. Note that using the maximum density will provide an over-estimate for any risks, since for the purpose of calculating the number of livestock exposed, the arithmetic means should be used. These arithmetic mean data are not currently available.
Livestock densities for England	Cattle 0.86/ha Pigs 0.67/ha Sheep 3.14/ha	Cattle 1.59/ha Pigs 2.8/ha Sheep 5.10/ha	Defra (2005; 2006a,b) Cronin (1996)
BSE source term for GB	57.6 bovine oral ID_{50} units (28.8 from UK plus 28.8 imported) based on oral ID_{50} being 0.1 g of bovine brain.	95^{th} percentile of 260.23 bovine oral ID_{50} estimated to leave abattoir in GB to food chain in 2008	Adkin *et al.*, 2010
Number of cats in UK	7.5 million	9.2 million	Cats' Protection website

Variable	Values from original risk assessment	Updated values based on current evidence	Source of updated values
Percentage of cat litter discarded to green/catering waste	10%	1% as a worst-case	Valorgas (2012) found no cat litter in 1,000 food waste collection bags sampled in UK
Virus destruction by composting	4.7-log	4.61 log	See Figure 14
Newcastle disease virus dose-response	Chicken oral ID_{50} = 10,000 EID_{50}	Chicken oral ID_{50} = 80 EID_{50}	Preliminary analysis of unpublished data from APHA
Newcastle disease virus soil decay data	--	-0.095 log EID_{50} per day at 21 – 27°C converted to -0.0125 log EID_{50} per day at 3 to 6°C	Analysis of data for decay on grain from Echeonwu *et al.* (2008) and soil survival times of Olesiuk (1951)
Decay rates for pathogens in soil (unchanged). Note for exotic viruses decay data in slurry was used as a surrogate for soil.	Log per day CSFV (-0.05459) FMDV (-0.04847) ASFV (-0.029) *T. gondii* (-0.0119)	Log per day CSFV (-0.05459) FMDV (-0.04847) ASFV (-0.029) *T. gondii* (-0.0119)	Liquid manures, Haas *et al.* (1995) Using data of Olson *et al.* (1999) for 1 log decay of *Cryptosporidium* in 84 days soil as a surrogate
Avian influenza virus (AIV) dose response	--	H5N1, Chicken oral ID_{50} = 1,000 EID_{50}	Unpublished data from APHA
AIV decay data	--	-0.006 log per day for virus in wildfowl wintering ground in winter	Breban *et al.* (2009)
H5N1 avian influenza virus in meat	--	10^6 EID_{50} per gram	Thomas and Swayne (2007)

7.2 Risk assessment methodology and terminology

Microbiological Risk Assessments (MRAs) facilitate scientific investigations of risks including quantification of uncertainty/variability and prioritisation of control strategies, such as compost tillage and lengths of any grazing ban. Variability describes the natural variation of the process (e.g. number of bacteria per gram of cattle faeces) and uncertainty describes the lack of knowledge (e.g. as a result of small sample sizes). The quantification of uncertainty and/or variability is a challenge in MRAs. Incorporating variability and/or uncertainty in a stochastic risk assessment produces a final risk estimate with a variability or uncertainty distribution surrounding it. As a consequence of this, the majority of MRAs are stochastic. However, due to the broad scope of this risk assessment, a deterministic approach has been implemented using the arithmetic mean instead of probability distributions that describe any known uncertainty or variability.

The advantage of deterministic approaches is that they are more easily communicated, and that less information is required for calculation. For example, stochastic models require knowledge of which type of distribution is appropriate for a given variable together with the parameters. In many situations, there is insufficient quantitative data to accurately model uncertainty and variability. It should be noted, however, that the arithmetic mean accommodates the variation (see Gale 2003), although missing the "rare but all important high count samples" in skewed distributions may underestimate the value.

Deterministic approaches are valid for estimating risk and the methodology implemented here has been previously described in detail (Gale 2001; 2003; 2004; 2005a; 2005b). Rather than addressing uncertainties, worst-case estimates for modelling parameters are used, so that the overall predicted risk represents an upper estimate, and readers can be confident that the actual risks will be lower. A list of worst-case assumptions used in the model is provided in Section 16.3. Where uncertainties have been identified in a parameter, scenarios have been explored to assess the impact of alternative values for that parameter on the baseline model result. These are presented in the Sensitivity Analysis (Section 9.0).

7.2.1 Precision in the risk assessment model

One criticism of the quantitative approach to risk assessment used here has been that the inputs and hence the outputs are over precise, given the likely uncertainties, and this may give rise to over-interpretation of the outputs and a false confidence in the results. Indeed, reference has previously been made to the update of the log removal (by composting) from 4.7-log to 4.61-log in this revision of the risk assessment. The change from 4.7-log (Table 5-1) to 4.61-log (Figure 14) represents an improvement in the design of the model, so that sensitivity in both the by-pass parameter (Section 9.5) and the parameter for inactivation in the hot part (Section 9.6) can be investigated. As with all quantitative models, the most accurate value for each parameter should be used within the model.

It should be remembered that the numerical final results, although precise in themselves, are only a guide to the magnitude of the risks. Clearly there is uncertainty associated with the final result, and that uncertainty is not defined in deterministic risk assessments.

The quantitative risk estimates presented in this report provide a guide as to the magnitude of the risks at the time of this assessment given the assumptions made and available data. The key findings of this revised risk assessment are that there is no need for tightening current composting process parameters or the length of the grazing bans, and that prevention of process by-pass, whether intrinsic or extrinsic, remains a critical control point.

7.3 Approach to estimating the number of livestock exposed to compost-treated soil

The total amount of compost that could be produced from catering waste in GB is estimated to be 3,913,200 tds (Section 2.4.1), which would cover some 195,660 ha of land at an application rate of 20 tds/ha (Section 3.1.1). The total agricultural areas for England, Wales and Scotland in June 2010, together with sources, are set out in Table 7-2. It would be difficult to apply compost to rough grazing land because of physical access restrictions, wetness and steepness of slopes. Therefore ignoring rough grazing, it is calculated that grazing land accounts for 58.2% of the agricultural land to which compost could be applied (Table 7-2).

Table 7-2: Agricultural land use in GB (thousand ha). June Census 2010

	England	Wales	Scotland	Total	Percentage
Crops	3,918	85	572	4,575	41.8%
Temporary grass	587	103	423		
Permanent grass	3,288	1,021	955		
Total grassland	3,875	1,124	1,378	6,377	58.2%
Sole right rough grazing	493	230	3,192		
Common rough grazing	428	180	583		
Total	8,714	1,619	5,725	16,058	
Total excluding rough grazing	7,793	1,209	1,950	10,952	

England: (https://data.gov.uk/dataset/june_survey_of_agriculture_and_horticulture_england)
Wales: (http://wales.gov.uk/docs/statistics/2010/101117sdr1882010en.pdf)
Scotland: (http://www.scotland.gov.uk/Resource/Doc/335200/0109647.pdf)

Therefore, of the 195,660 ha to which compost could be applied in GB, some 113,897 ha are grassland (temporary or permanent). For the purposes of this risk assessment it is assumed that all those 113,897 ha of grassland are used for grazing livestock animals. The approach taken is that compost is applied homogenously over the area of 113,897 ha on which livestock graze at the highest livestock densities recorded in England by Defra (2005, 2006a, 2006b), which results in a worst-case estimate for the numbers of livestock animals exposed. Ideally, an average livestock density for England, Wales and Scotland should be used. To include Scotland and Wales, the assumption here is that the average livestock density for GB as a whole is no higher than the maximum density recorded for England.

The maximum livestock densities recorded in the England Census have been set out in Table 3-14. For example, the maximum density of pigs recorded in England in June 2005 was 280 per km^2. Note this is total land area, and not just the farmers' land where pig densities per unit area are higher locally. Since there are 100 ha to one km^2, the maximum pig density is 2.8 pigs/ha assumed over the total land area. Therefore, the total number of pigs which could be housed on land in GB to which compost has been applied is 113,897 ha x 2.8 pigs/ha = 318,912 pigs. This is shown in Figure 20. The total numbers of pigs, cattle and sheep grazing or housed on land treated with compost are set out in Table 7-4.

Using this 'total' land area approach seeks to address uncertainties over how much of the grazing land treated with compost is actually grazed at any one time. Thus, whilst it is unrealistic to assume that livestock are grazed in the same one field for an entire year and only eat compost (to the exclusion of any soil) for that year, it is not unrealistic to assume that a smaller subset of livestock are moved from one field to another over the course of a year, with compost having been applied to each of those fields. The risk assessment approach estimates a cumulative annual exposure per livestock animal which is translated into a risk of infection using the dose-response relationship (see Section 7.5). The dose-response model is an independent action model and it is irrelevant from which field each daily pathogen exposure comes from, or indeed whether on some days a particular animal

ingests a higher dose than on other days. This cumulative annual exposure is accommodated by the dose-response model used here (see Section 7.5).

7.3.1 Percentage of catering waste-derived compost (surface applied) which is ingested by livestock in this model

The 318,912 pigs each consuming 0.392 kg/pig/day of compost (Table 3-10) would ingest 125,013 kg compost per day. Each day therefore, 0.0032% of the total compost applied annually (3,913,200 tonnes) is ingested by pigs. This assumes surface applied compost is eaten to the complete exclusion of soil (Section 3.2). Over a period of 1 year, this is 45,630 tonnes of compost. According to the model therefore, for surface applied compost, pigs ingest 1.17% of the total of 3,913,200 tds of catering waste-derived compost produced annually in GB. This is shown in Figure 20. Daily and annual percentages of total GB catering waste-derived compost ingested by pigs, cattle and sheep according to this model are set out in Table 7-4. In the case of surface applied compost, pigs, cattle and sheep combined eat 6.81% of the total catering waste-derived compost produced annually in GB. This would appear to be an unrealistic estimate and reflects the worst case assumptions used here in the model.

It is important to note that the risk assessment model is not allowing for more than the total amount of compost produced annually to be ingested, i.e. that additional infectivity is not artificially created by the risk assessment calculation. Thus, the pathogen loading on a given 1 ha area is finite, reflecting the fact that only 20 tonnes (dry weight) is applied per ha. Stocking densities at which livestock would ingest all the compost applied to the surface of a given hectare are shown in Table 7-3. It is concluded that stocking densities are unlikely to be high enough for livestock to eat all the compost in a given field such that subsequent livestock would be exposed to zero risk on that hectare.

Table 7-3: Calculation of stocking density at which all 20 tonnes (dry weight) compost/ha (surface-applied) would be eaten by livestock in that hectare during the year

	Daily consumption of soil (compost) kg/head/day	Annual consumption of soil (compost) tonnes/head/year	Stocking density to ingest 20 tonnes/ha (head/ha)
Cattle	1.125	0.411	48.7
Pig	0.392	0.143	139.8
Sheep	0.69	0.252	79.4

Figure 20: Exposure of pigs to surface-applied compost in GB

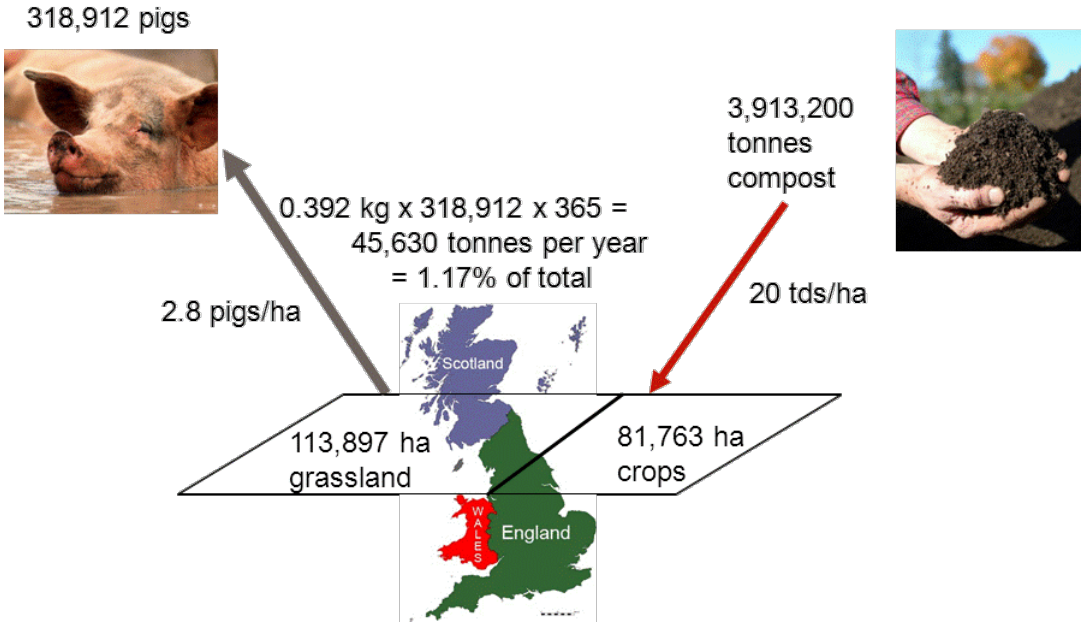


Table 7-4: Summary of proportion of total compost in GB predicted to be ingested annually by livestock after surface application (i.e. with no dilution in the soil)

Livestock	Livestock Density (head/ha)	Number of livestock on land treated with compost	Predicted percentage of total compost ingested	
			Daily	Annually
Pigs	2.8	318,912	0.0032%	1.17%
Cattle	1.59	181,096	0.0052%	1.9%
Sheep	5.10	580,875	0.0102%	3.74%

As set out in Section 3.3.2, it is concluded that it is not possible to calculate the total number of poultry which could be exposed to compost.

7.3.2 How do the numbers of livestock exposed in the revised model compare with those in the original risk assessment?

The original risk assessment assumed that England and Wales are comprised of a single field to which compost was randomly applied, and within which cattle, sheep and pigs randomly graze. The total tillage and grassland in England and Wales was an estimated 9,537,300 ha (Gale 2002). Assuming an original application rate of 10 tds/ha, the 500,000 tds of compost covered just 50,000 ha, which is 0.52% of the total tillage and grassland. The numbers of cattle, sheep and pigs on land to which compost was applied according to the original risk assessment was calculated as 0.52% of the total numbers of cattle, sheep and pigs in GB.

Table 7-5 compares the estimates of numbers of exposed livestock in this revised risk assessment with those in the original.

Table 7-5: Number of livestock in GB exposed to soil treated with compost estimated by original and revised risk assessment

Livestock species	Original risk assessment	Revised risk assessment	Increase
Cattle	42,800	181,096	4.2-fold
Pigs	33,417	318,912	9.5-fold
Sheep	157,100	580,875	3.7-fold

When compared to the original risk assessment, this revised version predicts a greater number of livestock are on land to which compost has been applied. This is because the revised risk assessment assumes more compost is produced (hence applied to a greater surface area) and also assumes higher livestock densities on that land (Table 7-1). The net result is that the revised risk assessment assumes a much greater proportion of the compost is eaten by livestock than the original risk assessment.

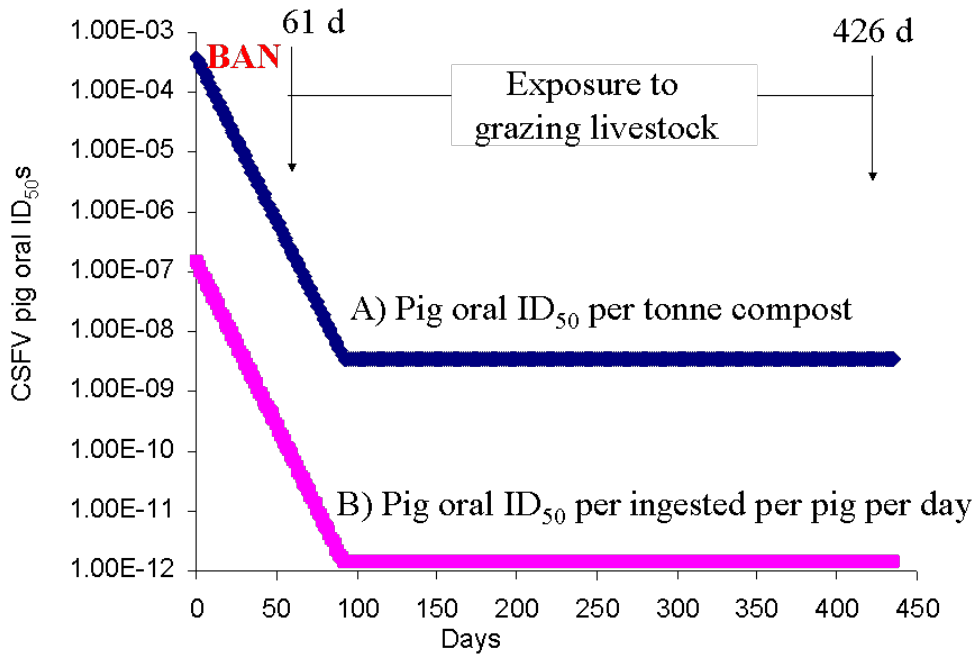
The compost application rate used here is double that used originally (Section 3.1.6). This higher application rate means twice the pathogen loading in the soil. This does not affect the individual risks from surface applied compost because it is assumed the livestock are eating neat compost anyway (so the application rate is immaterial). However, it doubles the individual risks for tilled or ploughed compost by halving the dilution factor in the soil. This is discussed further in the sensitivity analysis (Section 9.0). Doubling the application rate means a given mass of compost only goes half as far in terms of area covered, and hence only half the number of animals are exposed, albeit to double the pathogen dose. So while doubling the application rate doubles the individual risk, the total number of infected animals is unaffected.

7.4 Modelling decay of pathogens in the soil/compost

Once applied to or mixed with the soil there will be a daily decrease in the infectivity of CSFV, ASFV, FMDV, NDV, AIV and *T. gondii* due to decay. It is assumed SVDV, BSE and scrapie do not decay in the soil.

The decay data used for FMDV, CSFV and ASFV are for slurry at 4°C (Haas *et al.* 1995). No data were available for decay of these viruses in the soil. It should be noted that decay at 4°C will be less than that at higher temperatures. As in the original risk assessment, no more than 5-log decay of the pathogens is permitted in the soil. This is illustrated for CSFV in surface applied compost (i.e. with no soil dilution) in Figure 21 line A. Thus at $t = 0$, there are 0.00038 pig oral ID_{50} units of CSFV per tonne of compost on the soil surface. The decay rate is 0.05459-log per day. This rate continues for 92 days to allow for a 5-log decay, after which the virus concentration is modelled as constant in the compost/soil.

Figure 21: Predicted decay of pig oral ID_{50} units of CSFV in compost applied to the surface of the soil. A 2 month grazing ban for pigs is illustrated



7.5 Calculating the annual virus exposure and the individual risk of infection

The daily exposure to an individual pig is calculated as the product of the pathogen concentration in the soil on that day and the amount of soil ingested per day (0.392 kg soil per pig per day). The daily exposure of an individual pig to CSFV is shown in Figure 21 line B. The daily exposure is summed over every day for a period of one year to give the annual exposure in pig oral ID_{50} units. This accommodates the decay in soil over the one year period. The annual risk of infection in that individual pig is calculated by multiplying the total annual exposure by 0.69 (Gale, 2002; 2004). The units of individual risk of infection are "per pig per year".

The model assumes there is just one application of compost per year to a given field. Thus if a field were topped up daily with small amounts of fresh compost over the period of a year (to a total of 20 tds/ha), then there would be no time for decay on the soil. Assuming there is just as much chance that a farmer moves livestock to a treated field as to an untreated field at any time of the year, then moving livestock from field to field would not have an effect on the annual exposure unless farmers systemically moved the livestock to fields to which compost had just been applied. Clearly, if farmers repeatedly moved livestock to fields which had just been amended with compost, there would be less time for pathogen decay.

It is important to note that the individual risks predicted here apply only to livestock which graze for a whole year on land to which compost has been applied at 20 tds/ha. It is not an average risk to *all* livestock in GB as clearly those livestock not grazing on compost-amended land will experience zero exposure to compost (and therefore, zero risk). Presenting the individual risk reassures farmers that the risk to individual animals on a farm which has received compost is very low.

7.6 Predicting the total number of infections in GB per year

The original risk assessment was undertaken for strategic purposes at a GB level, and this approach is taken again here by estimating the number of infections in GB per year. Thus, the number of infected pigs in GB per year is calculated by multiplying the annual individual risk (per pig per year) by the total number of pigs grazing on land treated with compost

(Table 7-4). The average number of years between infections within GB is expressed as the reciprocal of the total number of infections per year. The same approach is used for calculating the number of infections in cattle (e.g. BSE and FMDV) and in sheep (e.g. scrapie) and the number of still births in sheep from *T. gondii*.

7.7 Allowing for a grazing ban

As in the original risk assessment, the effect of the grazing ban was modelled by allowing for decay of the virus in the soil over a period of 3 weeks or 2 months. Subsequently, daily exposures were summed for a period of 365 days, for example from day 61 to day 426 in the case of a two month ban as shown in Figure 21. A 3 week no grazing period was modelled by starting the soil exposure at time t = 21 days.

8.0 Results

8.1 Foot and mouth disease virus

8.1.1 Source term

During infection with FMDV, the virus has been found in tissues of cattle, sheep and pigs including lymph nodes, lungs, spleen, liver, kidneys and bone marrow (Henderson and Brooksby 1948; Cottral 1969; Sobko *et al.* 1973; Commission of the European Communities 1986). During rigor mortis, the fall in pH from build-up of lactic acid inactivates the virus in muscle tissue (Henderson and Brooksby 1948). However in clotted blood, marrow and lymph nodes virus remains active for 4 months at 4°C. Therefore, some components of imported meat could include FMDV-infected lamb, beef and pig meat.

8.1.1.1 Amount of meat illegally imported into GB and infected with FMDV

Estimates of the amounts of meat infected with FMDV and illegally imported into GB are shown in Table 8-1 (Hartnett *et al.* 2004).

Table 8-1 Amount of meat (kg) illegally imported into GB contaminated with foot-and-mouth

Pathogen	5th percentile	Mean	95th percentile
FMDV	64.6	214.2	565

8.1.1.2 FMDV loadings in an infected pig

The total FMDV loading in a viraemic "bone-in" pig carcass is estimated to be 6.2×10^{10} $TCID_{50}$s (Table 8-2).

Table 8-2 Estimated FMDV loadings in an infected pig

Tissue	Weight (kg)	$TCID_{50}$/g or /ml	Total loading in pig ($TCID_{50}$)
Flare fat	1.00	$10^{6.0}$	$1.0 \times 10^{9.0}$
Kidneys	0.26	$10^{6.6}$	$1.0 \times 10^{9.0}$
Intestinal fat	0.84	$10^{6.0}$	$8.4 \times 0^{8.0}$
Caul fat	0.11	$10^{6.0}$	$1.1 \times 10^{8.0}$
Heart	0.26	$10^{1.03}$	$2.8 \times 10^{3.0}$
Liver, gall bladder	1.50	$10^{6.6}$	$6.0 \times 10^{9.0}$
Blood	3.40	$10^{8.2}$	$5.4 \times 10^{11.0}$
Lymph nodes	0.04	$10^{8.0}$	$4.0 \times 10^{9.0}$
Bone marrow	0.5464[a]	$10^{7.6}$	$2.2 \times 10^{10.0}$
Muscle	48.63[b]	$10^{1.03}$	$5.2 \times 10^{5.0}$
Blood in muscle	5.464[c]	$10^{4.5}$	$1.7 \times 10^{8.0}$
Total (bone-in)	62.05		$5.7 \times 10^{11.0}$
Total (bone-in)[#]	**58.82**		**$6.2 \times 10^{10.0}$**

[a] assumes 1% of carcass weight
[b] assumes 89% of carcass weight
[c] assumes 10% of carcass weight
[#] assuming only 5% of blood (0.17kg) remains in carcass

8.1.2 Dose-response data for FMDV

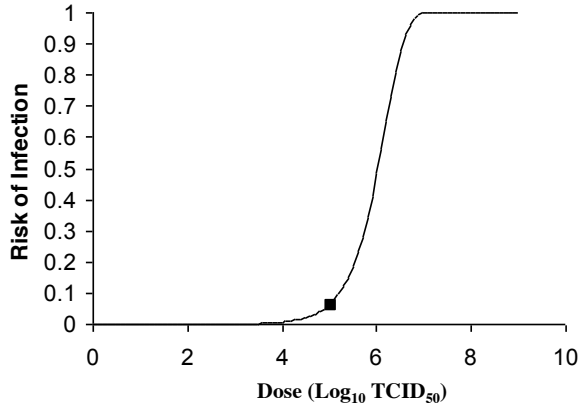
Relatively high doses of FMDV are required to initiate infection in pigs and cattle by the oral route, the "minimum infective doses" being about $10^{5.0}$ $TCID_{50}$ for pigs and $10^{6.0}$ $TCID_{50}$ for cattle (Donaldson 1997). The ID_{50} values (i.e. doses required to infect 50% of those animals which are given that dose as opposed to the "minimum infective dose") are typically in the order of 10-fold higher than the minimum infective dose (see Figure 22). Risks through inhalation are not considered here. The data available for oral infection of cattle, pigs and sheep are now described.

8.1.2.1 Cattle

Donaldson (1997) assumes that calves require a dose of $10^{6.0}$ $TCID_{50}$ by oral ingestion to initiate infection. However, this is an estimate of the minimum infective dose (i.e. the smallest quantity of virus, which in theory, is capable of initiating infection leading to clinical disease). In fact, an oral dose of $10^{6.5}$ $TCID_{50}$ did not result in any cattle being infected ((Henderson and Brooksby (1948) cited by Sellers 1971). Indeed, Henderson and Brooksby (1948) remarked on the difficulty of infecting cattle by feeding virus in glass tubes which were masticated by the animals (cited in Donaldson 1997). On this basis the oral ID_{50} for cattle will be $>10^{7.0}$ $TCID_{50}$. However, as a worst case in the risk assessment here, the oral ID_{50} for cattle is assumed to be $10^{6.0}$ $TCID_{50}$ (Table 8-3).

8.1.2.2 Pigs

Figure 22: Dose-response curve for ingestion of FMDV by pigs. The oral ID_{50} is about $10^{6.0}$ $TCID_{50}$



Farez and Morley (1997) cite experiments in which a viral titre of $10^{5.0}$ $TCID_{50}$ of FMD O-strain infected two of 30 pigs. Indeed, Donaldson (1997) has suggested that $10^{5.0}$ $TCID_{50}$ is the minimum infective dose. This point is plotted in Figure 22. Using a standard approach to plot a negative exponential dose-response curve through this one point suggests the oral ID_{50} for pigs is in the order of $10^{6.0}$ $TCID_{50}$ (Table 8-3).

8.1.2.3 Sheep

There are few, if any, data on the infectious dose for FMDV in sheep. However, Prof. Alex Donaldson (pers. comm.) suggested that sheep would be similar to cattle. The bovine oral ID_{50} is assumed to be $10^{6.0}$ $TCID_{50}$ and for the purpose of risk assessment is also assumed for sheep (Table 8-3).

Table 8-3 Summary of ID50s for FMD

Animal	Oral
Cattle	$10^{6.0}$ $TCID_{50}$
Pigs	$10^{6.0}$ $TCID_{50}$
Sheep	$10^{6.0}$ $TCID_{50}$

Schijven *et al.* (2005) have undertaken a risk assessment for FMDV transmission via water. The source term was milk illegally discharged to the sewer in the Netherlands during the 2001 outbreak. The authors use the same data source for the oral dose-response as the original risk assessment from Sellers (1971). Whereas the original edition of this compost risk assessment (Gale 2002) used a single point from the Sellers (1971) paper as cited by Farez and Morley (1997), namely that $10^{5.0}$ $TCID_{50}$s infected 2 out of 30 pigs, Schijven *et al.* (2005) have used the complete data set. The dose-response curve for ingestion used in this study is slightly more precautionary (predicting slightly higher risks from a given dose) than would be the case of the Schijven *et al.* data were used.

In summary, an oral ID_{50} of $10^{6.0}$ $TCID_{50}$ is used for cattle and sheep and pigs in this risk assessment.

8.1.3 Total FMDV loading going to compost

Using the 95th percentile of 565 kg of FMDV-infected meat illegally imported (Table 8-1) and a pig carcass weight of 62 kg gives an estimate of 9.11 FMDV-infected pig carcasses illegally imported. The total FMDV infectivity, imported illegally, is therefore 9.11 x 6.2 x 10^{10} = 5.6 x 10^{11} $TCID_{50}$s. Since the same oral infectivity is assumed for cattle, sheep and pigs (Table 8-3) the risk assessment does not distinguish between cattle, pig or sheep oral ID_{50}s. Thus, assuming 1 oral ID_{50} is $10^{6.0}$ $TCID_{50}$s (Table 8-3), 5.6 x 10^{11} $TCID_{50}$s translates into 563,231 oral ID_{50} units for cattle, sheep or pigs. Since FMDV-infected meat could be lamb, beef or pig, the largest percentage for raw identifiable mammalian meat discarded to the waste bin is used here (Table 2-3). This is 1.39% for pork. Thus 563,231 x 0.0139 = 7,829 oral ID_{50} units are discarded to the waste bin each year.

8.1.4 Predicted FMDV concentrations in compost and soil

Assuming composting removes 4.61-log of virus (Section 4.0), then a total of 0.19 oral ID_{50}s remain in the treated compost each year (Table 8-4). The predicted arithmetic mean FMDV concentrations for compost and soil are summarised in.

Table 8-4 Predicted arithmetic mean FMDV concentrations in compost

Pathogen	Oral ID_{50} in total compost per year	FMDV concentration in compost (Oral ID_{50} per tonne)	FMDV concentration in soil (Oral ID_{50} per tonne) at t = 0, assumes depth of 10 cm
FMDV	0.19	0.49 x 10^{-7}	0.66 x 10^{-9}

8.1.5 Calculation of the risks of FMD to cattle, sheep and pigs

Risks are calculated separately for cattle (Table 8-5 and Table 8-6), pigs (Table 8-7) and sheep (Table 8-8). The predicted individual risks are highest for cattle because cattle eat more soil than sheep and pigs (Table 7-1). The individual risks presented in Table 8-5 apply to each of the 181,096 cattle which could graze for a whole year on the 113,897 ha of grassland to which 58.2% of the 3,913,200 tds compost has been applied.

The individual risks for cattle are very low; the highest being 0.36 x 10^{-9} per animal per year with surface application and no grazing ban (Table 8-5). Thus, even with no grazing ban

and no dilution in the soil, the model predicts just one FMDV infection in cattle every 15,292 years in the GB (Table 8-6). With a three week grazing ban (for cattle) and minimum tillage (to 10 cm) this risk decreases to just one FMDV infection predicted every 11,917,000 years (Table 8-6).

In terms of predicted number of infections in GB per year, the highest risk from FMDV is for sheep calculated with 5.1 sheep/ha, with one case predicted every 7,773 years. This is with no grazing ban and with surface-application of the compost (Table 8-7).

Table 8-5 Individual risks* for cattle (risk per animal per year) predicted for FMDV

Length of grazing ban	Surface application (no dilution)	Minimum tillage (10 cm)	Plough depth (25 cm)
No ban	0.36×10^{-9}	0.48×10^{-11}	1.93×10^{-12}
3 week	0.35×10^{-10}	0.46×10^{-12}	1.85×10^{-13}
2 month	0.52×10^{-12}	0.69×10^{-14}	0.28×10^{-14}

*Note predicted individual risks apply to a cow spending all day, every day for 1 year on land to which compost has been applied once at 20 tds/ha

Table 8-6 Average number of years predicted between cases of FMDV in cattle in GB from the application of compost to soil

Length of grazing ban	Surface application (no dilution)	Minimum tillage (10 cm)	Plough depth (25 cm)
No ban	15,292	1,147,000	2,867,000
3 week	1,598,900	11,917,000	29,794,000
2 month	10,608,000	795,600,000	1,989,000,000

Table 8-7 Average number of years predicted between cases of FMDV in pigs in GB from the application of compost to soil – calculated assuming pig density of 2.8/ha

Length of grazing ban	Surface application (no dilution)	Minimum tillage (10 cm)	Plough depth (25 cm)
No ban	24,922	1,869,000	4,672,000
3 week	259,000	19,422,000	48,600,000
2 month	17,300,000	1,297,000,000	3,241,000,000

Table 8-8 Average number of years predicted between cases of FMDV in sheep from the application of compost to soil

Length of grazing ban	Surface application (no dilution)	Minimum tillage (10 cm)	Plough depth (25 cm)
No ban	7,773	583,000	1,457,000
3 week	80,771	6,058,000	15,144,000
2 month	5,400,000	404,000,000	1,011,000,000

8.2 Classical swine fever virus

8.2.1 Source Term

8.2.1.1 Amount of meat illegally imported into GB and infected with CSFV

Estimates of the amounts of meat infected with CSFV and illegally imported into GB (Hartnett *et al.*, 2004) are set out in Table 8-9.

Table 8-9 Amount of meat (kg) contaminated with classical swine fever virus and illegally imported into the GB each year (Hartnett et al., 2004)

Pathogen	5th percentile	Mean	95th percentile
CSFV	7.5	263	794

Of the four exotic viruses (namely FMDV, CSFV, ASFV, and SVDV) studied by APHA, the highest amount of illegally imported infected meat was for CSFV (Hartnett *et al.* 2004). These authors estimated that an average of 263 kg per year of pig meat contaminated with CSFV was imported (with 90% certainty the volume is between 7.5 kg and 794 kg per year). A weight of 263 kg of pig meat is equivalent to 4.24 pig carcasses (each of 62 kg).

8.2.1.2 CSFV loadings in an infected pig

The paper by Farez and Morley (1997) as used in the original risk assessment remains the most appropriate for viral titres in infected meat (T. Drew, APHA *pers. comm.*). The original risk assessment assumed that 1 pfu = 251 $TCID_{50}$ and this has not been changed. The Virology Department at APHA are currently undertaking experiments investigating CSFV loadings in meat, although the results have not been reported yet.

Using the assumption (Section 2.3.1) that bone marrow is 1% of the weight of the carcass gives an estimate of 3.6×10^{10} $TCID_{50}$ of CSFV per infected pig (Table 8-10).

Table 8-10 Estimated CSFV loadings in an infected pig

Tissue	Weight (kg)	$TCID_{50}$/g or /ml	Total loading in pig ($TCID_{50}$)
Flare fat	1.00	1,584	1.6×10^{6}
Intestinal fat	0.84	1,584	1.3×10^{6}
Caul fat	0.11	1,584	1.7×10^{5}
Heart	0.26	$10^{4.9}$	2.1×10^{7}
Blood drained from carcass	3.40	1.6×10^{6}	5.3×10^{9}
Lymph nodes	0.04	$10^{7.5}$	1.3×10^{9}
Bone marrow	0.5464[a]	4.0×10^{7}	2.2×10^{10}
Muscle	48.6296[b]	$10^{4.9}$	3.9×10^{9}
Blood in muscle	5.464[c]	1.6×10^{6}	8.7×10^{9}
Total (bone-in)	62.02		4.1×10^{10}
Total (bone-in)[#]	58.82		3.6×10^{10}

[a] assumes 1% of carcass weight
[b] assumes 89% of carcass weight
[c] assumes 10% of carcass weight
[#] assuming only 5% of blood (0.17kg) remains in carcass

8.2.2 Dose-response data for CSFV

In the original risk assessment, the porcine oral ID_{50} was assumed to be 100 $TCID_{50}$ units on the basis of expert advice (T. Drew, APHA *pers. comm.*). The Virology Department at APHA are currently undertaking oral dose-response experiments with CSFV in pigs, but these are not complete at the time of writing (T. Drew, APHA *pers. comm.*). However, initial analyses suggest the porcine oral ID_{50} is in the region of 10^{4} $TCID_{50}$ units. As a worst-case

assumption, a porcine oral ID_{50} of 100 $TCID_{50}$ units is used here (or 100 times lower than current experimental work is indicating may be the case). Therefore, each 62 kg bone-in carcass contains 3.6×10^8 porcine oral ID_{50} units of CSFV.

8.2.3 Total CSFV loading going to compost

The 95th percentile estimate of 794 kg of CSFV-infected pig meat (Table 8-10) is equivalent to 12.8 carcasses of 62 kg weight. The total CSFV loading (95 percentile) imported illegally to GB is therefore 4.6×10^{11} $TCID_{50}$ units or 4.6×10^9 porcine oral ID_{50} units. Of these 1.39% (6.4×10^7 porcine oral ID_{50} units) goes to composting (Table 2-2) which destroys 4.61-log, leaving a total of 1,566 porcine oral ID_{50} units in the treated compost.

8.2.4 Predicted CSFV concentrations in compost and soil

Assuming all the catering waste produced in GB is composted, 1,566 pig oral ID_{50}s of CSFV are dispersed into 3,913,200 tds of compost, giving a predicted arithmetic mean CSFV concentration for compost of 0.0004 porcine oral ID_{50} units / tonne. Using an application rate of 20 tds/ha and tilling in to a depth of 10 cm, gives an arithmetic mean CSFV concentration of 5.34×10^{-6} pig oral ID_{50} units / tonne of soil at t = 0 days. These are summarised Table 8-11.

Table 8-11 Predicted arithmetic mean CSFV concentrations in compost

Pathogen	Oral ID_{50} in total compost per year	Pathogen concentration in compost (Oral ID_{50} per tonne)	Pathogen concentration in soil (Oral ID_{50} per tonne) at t = 0, assumes depth of 10 cm
CSFV	1,566	0.0004	5.34×10^{-6}

8.2.5 Risks of classical swine fever virus to pigs

The risk to an individual pig housed for a year on land to which compost has been applied is 0.92×10^{-6} per pig per year without a grazing ban and for surface applied compost, i.e. no soil dilution (Table 8-12). Assuming a pig density of 2.8 pigs/ha, then 318,912 pigs are housed on the 113,900 ha of grassland treated with compost. Across GB as a whole, without any grazing ban, and allowing for no dilution in the soil the models predicts one case of CSFV every 3.4 years (Table 8-13). With a three week ban, the model predicts one case every 48 years for surface applied compost, and with the 2 month ban (as set out for pigs by the Animal By-Product Regulations (2011)), the predicted risk is one case every 4,109 years. It is concluded that maintaining the 2 month ban is important for pigs in respect of CSFV for surface applied compost. However, it should be noted that the pig oral ID_{50} may be in the region of 10^4 $TCID_{50}$ (APHA, unpublished data) rather than the 100 $TCID_{50}$ used here and therefore CSFV may be 100-fold less infectious and the risks 100-fold lower than predicted.

It is also worth noting that it is not considered good agricultural practice to spread organic manures (such as compost) ahead of outdoor pigs, since the pigs are themselves expected to add substantially to soil nutrient loadings, negating any agronomic benefits that might be expected to have derived from the compost.

Table 8-12 Individual risks* (risk per pig per year) predicted for CSFV to a pig grazing on fields to which compost has been applied

Length of grazing ban	Surface application (no dilution)	Minimum tillage (10 cm)	Plough depth (25 cm)
No ban	9.2×10^{-7}	1.22×10^{-8}	4.9×10^{-9}
3 week	6.6×0^{-8}	0.88×0^{-9}	3.5×10^{-10}
2 month	0.76×10^{-9}	1.02×10^{-11}	4.1×10^{-12}

*Note predicted individual risks apply to a pig spending all day, every day for 1 year on land to which compost has been applied once at 20 tds/ha

With tilling to a depth of 10 cm, periods of at least 200 years are predicted between any outbreaks Table 8-13).

Table 8-13 Average number of years predicted between cases of CSFV in pigs in GB from the application of compost to soil - calculated assuming pig density of 2.8 / ha

Length of grazing ban	Surface application (no dilution)	Minimum tillage (10 cm)	Plough depth (25 cm)
No ban	3.4	257	641
3 week	48	3,580	8,950
2 month	4,110	308,218	770,546

8.3 Swine vesicular disease virus

8.3.1 Source term

8.3.1.1 Amount of meat illegally imported into GB and infected with SVDV

Estimates of the amounts of meat infected with SVDV and illegally imported into GB (Hartnett *et al.* 2004) are set out in Table 8-14.

Table 8-14 Amount of meat (kg) contaminated with swine vesicular disease virus and illegally imported into GB each year (Hartnett et al. 2004)

Pathogen	5th percentile	Mean	95th percentile
SVDV	0.002	0.007	0.021

8.3.1.2 SVDV loading in an infected pig

Using the assumption (Section 2.3.1) that bone marrow is 1% of the weight of the carcass gives an estimate of 7.9×10^9 plaque-forming units (pfu) of SVDV per infected pig carcass (Table 8-15).

Table 8-15 Estimated SVDV loadings in an infected pig

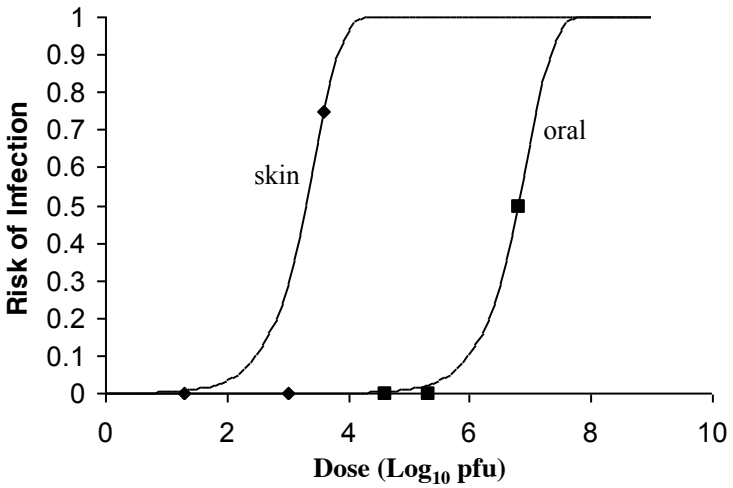
Tissue	Weight (kg)	pfu/g or /ml	Total loading in pig (pfu)
Flare fat	1.00	$10^{1.1}$	1.2×10^4
Kidneys	0.26	$10^{3.7}$	1.3×10^6
Head, tongue	5.00	10^5	5.0×10^8
Intestinal fat	0.84	$10^{1.1}$	1.1×10^4
Caul fat	0.11	$10^{1.1}$	1.4×10^3
Intestines	2.70	$10^{3.7}$	1.3×10^7
Heart	0.26	$10^{4.5}$	8.2×10^6
Liver, gall bladder	1.50	10^5	1.5×10^8
Pancreas	0.06	$10^{3.5}$	1.9×10^5
Spleen	0.11	$10^{4.5}$	3.5×10^6
Blood	3.40	10^6	3.4×10^9
Hair scrapings & hooves	0.84	$10^{4.2}$	1.3×10^7
Lymph nodes	0.04	$10^{6.2}$	6.3×10^7
Bone marrow	0.5464[a]	$10^{3.6}$	2.2×10^7
Muscle	48.62[b]	$10^{4.5}$	1.5×10^9
Blood in muscle	5.464[c]	10^6	5.5×10^9
Total (bone-in)	62.02		1.1×10^{10}
Total (bone-in)[#]	58.82		7.9×10^9

[a] assumes 1% of carcass weight
[b] assumes 89% of carcass weight
[c] assumes 10% of carcass weight
[#] assuming only 5% of blood (0.17kg) remains in carcass

8.3.2 Dose-response data for SVDV

Mann and Hutchings (1980) provide infectivity data for SVDV in pigs. No sign (or serological evidence) of disease resulted when amounts of virus of up to $10^{5.3}$ pfu were instilled into the mouth, nose and conjunctiva or painted on the tonsils. Indeed Burrows *et al.* (1974) showed that doses of $10^{1.5}$, $10^{2.5}$, $10^{3.5}$ pfu did not infect any of the 12 pigs exposed orally. Doses of $10^{6.8}$ pfu produced disease in about half of the six pigs within each group (Mann and Hutchings 1980). This suggested the oral ID_{50} is in the region of $10^{6.8}$ pfu for pigs. Fitting a negative exponential dose response curve to the data suggest that the probability (r) of infection from ingestion of just a single pfu is 1.1×10^{-7} (Figure 23).

Figure 23: Negative exponential dose response curves fitted to data for oral (r = 0.0000011) and skin (r = 0.00035) challenge of SVDV to pigs. Data from Mann and Hutchings (1980)



Therefore, each 62 kg bone-in carcass contains 1,256 porcine oral ID_{50} units of SVDV.

8.3.3 Total SVDV loading going to compost

The predicted amount of SVDV-infected meat entering GB each year is small, with a 95th percentile estimate of 0.021 kg (Table 8-14). This is equivalent to 0.033% of a 62 kg pig and, further, is equivalent to 2.7 x 10^6 pfu, or 0.42 pig oral ID_{50} (using the dose-response in Figure 23). Of this, 1.39% (Table 2-2) will enter the composting process. This amounts to 0.006 pig oral ID_{50} each year entering the compost.

8.3.4 Predicted SVDV concentrations in compost and soil

Assuming composting destroys 4.61-log of virus, then 1.4 x 10^{-7} pig oral ID_{50}s remain in the compost per year (Table 8-16). The predicted arithmetic mean pathogen concentrations for compost are summarised in Table 8-16.

Table 8-16 Predicted arithmetic mean SVDV concentrations in compost

Pathogen	Pig oral ID_{50} in total compost per year	SVDV concentration in compost (Oral ID_{50} per tonne)	SVDV concentration in soil (Oral ID_{50} per tonne) at t = 0, assumes depth of 10 cm
SVDV	1.4 x 10^{-7}	3.6 x 10^{-14}	4.8 x 10^{-16}

8.3.5 Risks of swine vesicular disease to pigs

The risk to an individual pig housed for a year on land to which compost has been applied, is insignificant at $<10^{-14}$ per pig per year even when considering surface applied compost (Table 8-17). The grazing ban is also irrelevant as it is assumed that there is no decay in the soil. Assuming a pig density of 2.8/ha such that some 318,912 pigs in GB are housed on land treated with compost, the risk assessment predicts one case of SVDV every 900 million years across GB (Table 8-18). This is without dilution in the soil.

Table 8-17 Individual risks* (risk per pig per year) predicted for SVDV to a pig grazing on fields to which compost

Length of grazing ban	Surface application (no dilution)	Minimum tillage (10 cm)	Plough depth (25 cm)
No ban	3.6×10^{-15}	4.8×10^{-17}	1.9×10^{-17}
3 week	3.6×10^{-15}	4.8×10^{-17}	1.9×10^{-17}
2 month	3.6×10^{-15}	4.8×10^{-17}	1.9×10^{-17}

*Note predicted individual risks apply to a pig spending all day, every day for 1 year on land to which compost has been applied once at 20 tds/ha

Table 8-18 Average number of years predicted between cases of SVDV in pigs in GB from the application of compost to soil - calculated assuming higher pig density of 2.8 / ha

Length of grazing ban	Surface application (no dilution)	Minimum tillage (10 cm)	Plough depth (25 cm)
No ban	876,000,000	6.6×10^{10}	1.6×10^{11}
3 week	876,000,000	6.6×10^{10}	1.6×10^{11}
2 month	876,000,000	6.6×10^{10}	1.6×10^{11}

8.4 African swine fever

8.4.1 Source term

8.4.1.1 Amount of meat illegally imported into GB and infected with ASFV

Estimates of the amounts of meat infected with ASFV and illegally imported into GB (Hartnett *et al.* 2004) are set out in Table 8-19.

Table 8-19 Amount of meat (kg) contaminated with African swine fever virus and illegally imported each year into GB (Hartnett et al. 2004)

Pathogen	5th percentile	Mean	95th percentile
ASFV	0.007	0.046	0.138

8.4.1.2 ASFV loadings in an infected pig

Using the assumption that bone marrow is 1% of the weight of the carcass (Section 2.3.1) gives an estimate of 2.4×10^{12} haemadsorbing doses (HAd_{50}) of ASFV per infected pig (Table 8-20).

Table 8-20 African Swine Fever Virus loadings in an infected pig. Tissue HAd50 titres from Farez and Morley (1997)

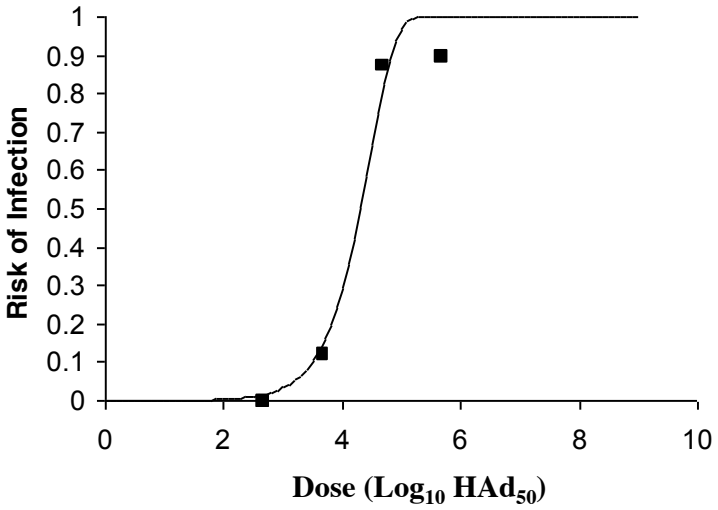
Tissue	Weight (kg)	HAd_{50} / g or / ml	Total loading in pig (HAd_{50})
Flare fat	1.00	$10^{5.4}$	2.5×10^{8}
Intestinal fat	0.84	$10^{5.4}$	2.1×10^{8}
Caul fat	0.11	$10^{5.4}$	2.7×10^{7}
Heart	0.26	$10^{6.6}$	1.0×10^{9}
Blood	3.40	$10^{7.9}$	2.7×10^{11}
Lymph nodes	0.04[c]	$10^{8.5}$	1.3×10^{10}
Bone marrow	0.5464[a]	$10^{9.5}$	1.7×10^{12}
Skeletal muscle	48.63[b]	$10^{6.6}$	1.9×10^{11}
Blood in muscle	5.464[c]	$10^{7.9}$	4.3×10^{11}
Total (bone-in)	62.02		2.6×10^{12}
Total (bone-in) [#]	58.82		2.4×10^{12}

[a] assumes 1% of carcass weight
[b] assumes 89% of carcass weight
[c] assumes 10% of carcass weight
[#] assuming only 5% of blood (0.17kg) remains in carcass

8.4.2 Dose-response data for ASFV

ASFV is less infectious to pigs than CSFV. The oral ID_{50} for pigs depends on the virulence of the strain. Values of $10^{4.3}$ and $10^{5.4}$ HAd_{50} units are quoted by Farez and Morley (1997) for porcine oral ID_{50}s. However, doses as high as $10^{6.1}$ HAd_{50} units failed to infect pigs in some experiments when administered either as liquid or solid food. The worst-case scenario is to assume the oral ID_{50} for pigs is $10^{4.3}$ HAd_{50} units. The dose response curve for infectivity for pigs through the oral route based on data from McVicar (1984) is plotted in Figure 24. It is assumed that the pig oral ID_{50} is $10^{4.3}$ HAd_{50} units of ASFV.

Figure 24: Negative exponential dose-response curve (r = 0.000035) for ingestion of ASFV by pigs. The porcine oral ID_{50} is about $10^{4.3}$ HAd_{50}. Data from McVicar (1984)



8.4.3 Total ASFV loading going to compost

The predicted amount of ASFV-infected meat entering GB each year is small with a 95th percentile of 0.138 kg (Table 8-19). This is equivalent to 0.2% of a 62 kg pig and is equivalent to 5.4 x 10^9 HAd_{50}, or 295,000 pig oral ID_{50} using the dose-response in Figure 24. It is assumed that 1.39% of this (Table 2-2) will enter the composting process. This equates to 4,110 pig oral ID_{50}.

8.4.4 Predicted ASFV concentrations in compost and soil

Assuming composting removes 4.61-log of ASFV, then a total of 0.10 pig oral ID_{50}s enter compost annually (Table 8-21). The predicted arithmetic mean pathogen concentrations for compost and soil are summarised in Table 8-21.

Table 8-21 Predicted arithmetic mean ASFV concentrations in compost

Pathogen	Oral ID_{50} in total compost per year	ASFV concentration in compost (Oral ID_{50} per tonne)	ASFV concentration in soil (Oral ID_{50} per tonne) at t = 0, assumes depth of 10 cm
ASFV	0.1	2.6 x 10^{-8}	3.4 x 10^{-10}

8.4.5 Risks of African swine fever to pigs

The risk of ASFV to an individual pig housed for a year on land to which compost has been applied is very low at 1.1 x 10^{-10} per pig per year without a grazing ban and for surface applied compost (Table 8-22). Assuming a pig density of 2.8 pigs/ha, then 318,912 pigs are housed on the 113,897 ha of grassland treated with compost. Across GB as a whole, without any grazing ban, and allowing for no dilution in the soil the models predicts one case of ASFV every 30,000 years (Table 8-23).

Table 8-22 Individual risks* (risk per pig per year) predicted for ASFV to a pig grazing on fields to which compost has been applied

Length of grazing ban	Surface application (no dilution)	Minimum tillage (10 cm)	Plough depth (25 cm)
No ban	1.1×10^{-10}	1.41×10^{-12}	5.7×10^{-13}
3 week	2.5×10^{-11}	3.39×0^{-13}	1.4×10^{-13}
2 month	1.7×10^{-12}	2.2×10^{-14}	0.90×10^{-14}

*Note predicted individual risks apply to a pig spending all day, every day for 1 year on land to which compost has been applied once at 20 tds/ha

Table 8-23 Average number of years predicted between cases of ASFV in pigs in GB from the application of compost to soil - calculated assuming pig density of 2.8 / ha

Length of grazing ban	Surface application (no dilution)	Minimum tillage (10 cm)	Plough depth (25 cm)
No ban	29,574	2,218,000	5,545,000
3 week	123,000	9,254,000	23,134,000
2 month	1,858,000	139,000,000	348,000,000

8.5 Newcastle disease

8.5.1 Source term

8.5.1.1 Amount of poultry meat illegally imported into GB and infected with NDV

Birds slaughtered for meat during disease episodes may represent an important source of NDV (Alexander 1988). The assessment of meat illegally imported into GB (Hartnett *et al.* 2004) did not include poultry meat or NDV specifically. However, a previous risk assessment included estimation of the risk of introduction of Newcastle disease into Chile through import of live ostriches (Burbano *et al.* 2005). The previous edition of this compost risk assessment (Gale 2002) assumed 100,000 poultry carcasses which were infected with NDV were composted annually for the purpose of a "What If?" analysis.

An expert virologist at the APHA has confirmed that if there is an identified outbreak(s), then no infected meat should enter the food chain, as there is a requirement to trace all meat and destroy it (Dennis Alexander, previously Head of Virology at APHA, *pers. comm*. 2009). This has been undertaken in the past at great expense. Inevitably, there is an element of delay from notification to seizure of all products already in the supply chain. Outbreaks are unlikely to go undetected in an immunologically naive flock, although the likelihood increases if the birds have been vaccinated (only a small proportion of broilers are vaccinated). However, meat from vaccinated challenged birds is unlikely to be infective even though the birds may excrete virus (D Alexander, APHA, pers. comm. 2009). There is a possibility that in some other poultry species (including turkeys) outbreaks may go undiagnosed, as the clinical signs are not as severe as the disease in chickens (D Alexander, pers. comm. 2009).

Currently it is not known how many birds/carcasses are illegally brought into the UK. The numbers could be fairly low, although the likelihood that they were infected with NDV could be fairly high - depending on the country of origin (D Alexander, APHA, pers. comm. 2009). Discussions with an expert have concluded that the estimation of 100,000 NDV-infected chickens (used previously Gale 2002) is probably even higher than an extreme worst-case (D Alexander, APHA, pers. comm. 2009). It is therefore proposed to conduct a "What If" analysis based on 10,000 NDV-infected chickens entering the GB food chain in a year. It should be noted that 10,000 chickens equate to some 15 tonnes of infected chicken meat, and it is suggested that this represents an unrealistic extreme worst-case.

8.5.1.2 NDV loadings in an infected poultry

Data published by Alexander *et al* (2006) for levels of NDV detected in organs and tissues of 6-week old chickens infected with NDV strain Herts 33/56 are presented in Table 8-24. These data were represented in the original compost risk assessment as unpublished data. The net loading for an infected chicken used in the revised risk assessment is 3.03×10^7 Egg Infectious Dose 50% units (EID_{50}) (as calculated previously by Gale 2002) based on the day 4 data.

Table 8-24 Newcastle disease virus loadings in 6-week old chickens. EID50 titres from Alexander et al. (2006)

	Weight of organ per carcass from Gale (2002)	Day post inoculation			
		1	2	3	4
Blood		1.2	1.4	2.4	-
Breast muscle	2,229 g of muscle as weight of carcass	-	1.0	2.2	4.0
Leg muscle		-	1.2	2.6	4.2
Heart, kidneys and spleen pool	30 g (in 5% of retail chickens)	1.8	4.2	5.0	6.0
Faeces	1 g	-	1.4	3.2	4.0

8.5.2 Dose-response data for NDV

In the original risk assessment, it was assumed that the chicken oral ID_{50} is $10^{4.0}$ EID_{50} units of NDV based on the oral titre required to infect 3 week old chickens (Alexander *et al.* 2006). Further experimental work at the APHA indicates that this estimate may be too optimistic. Preliminary analysis of unpublished APHA data suggests the poultry oral ID_{50} is approximately 80 (or $10^{1.9}$) EID_{50} units of NDV, which is the figure used in this revised risk assessment.

8.5.3 Total NDV loading going to compost

Assuming 10,000 NDV infected chickens enter the food chain each year, the total amount of NDV infectivity is 3.03×10^{11} EID_{50} per year. It is assumed that 2.82% of the chicken meat is discarded raw (Table 2-2) and will enter the composting process. This equates to 0.85×10^{10} EID_{50} per year. Since 80 EID_{50} units equate to one chicken oral ID_{50}, there are 1.07×10^{8} chicken oral ID_{50} entering the composting process per year.

8.5.4 Predicted NDV concentrations in compost and soil

Wambura *et al.* (2006) have published data on the thermostability of infectivity titre of NDV. After heating at 56°C for 120 minutes the infectivity titres had reduced from 9.5-log to 6.5-log. Thus the thermostability of NDV is comparable to SVDV, with 3-log destruction in 2 h, which is 6-log in 4 h assuming first order decay. Alexander and Manvell (2004) reported a decimal reduction time of 420 – 540 seconds for NDV at 60°C. Benson *et al.* (2008) reported that no viral haemagglutination activity was observed after composting for 2 days at temperatures in excess 60°C. However, samples with high virus concentration of NDV were not inactivated by treatment at 56°C for 120 min, which is consistent with the data of Wambura *et al.* (2006).

Assuming composting removes 4.61-log of NDV, then a total of 2,641 chicken oral ID_{50}s remain in the compost annually (Table 8-25). The predicted arithmetic mean pathogen concentrations for compost and soil are summarised in Table 8-25.

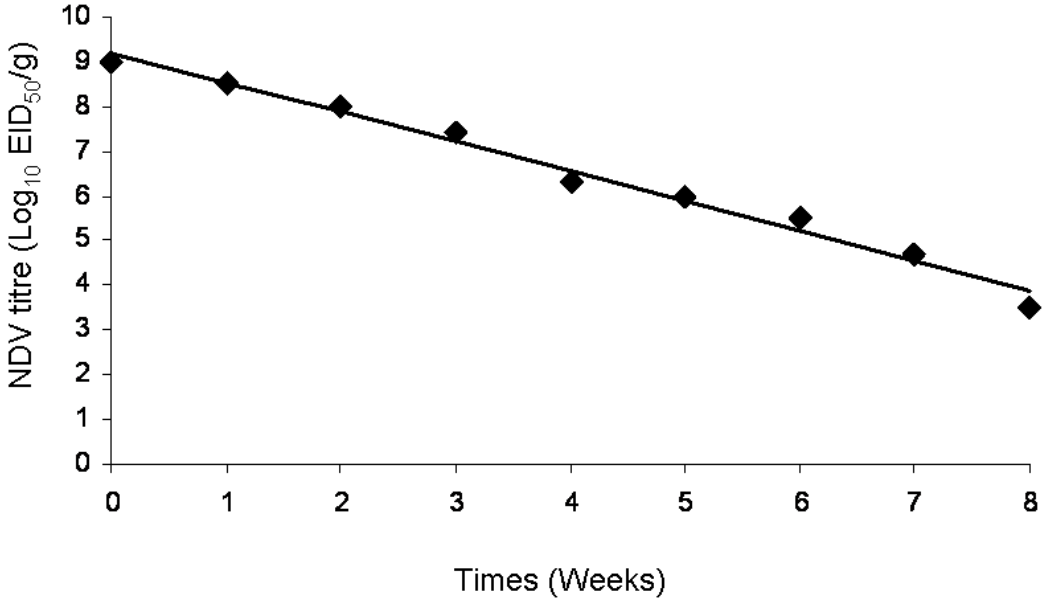
Table 8-25 Predicted arithmetic mean NDV concentrations in compost

Pathogen	Oral ID_{50} in total compost per year	NDV concentration in compost (Oral ID_{50} per tonne)	NDV concentration in soil (Oral ID_{50} per tonne) at t = 0, assumes depth of 10 cm
NDV	2,641	6.75×10^{-4}	9.0×10^{-6}

8.5.5 Data for decay of Newcastle disease virus in the environment

Echeonwu *et al.* (2008) have published data for survival of NDV on grain offals. The decay is plotted for maize offal in Figure 25. The experiments were performed at room temperature (quoted as 21 – 27°C) and a decay rate of -0.66 log per week which is -0.095 log per day was calculated by linear regression.

Figure 25: Survival of Newcastle disease virus strain V4-UPM coated on maize grain offal and exposed to room temperature (21 - 27°C). Data from Echeonwu *et al.* (2008)



Kinde *et al.* (2004) have published data for survival of exotic NDV in the commercial poultry environment following removal of infected chickens. Their data, however, are difficult to interpret. Those authors cite a paper by Olesiuk (1951) on survival of NDV in soil. They noted that the virus survived in soil for 538 days at 3°C to 6°C and for 71 days at 20°C to 30°C. The Olesiuk (1951) paper does not give viral titres over the duration of the experiment. It is therefore concluded that the survival of NDV in soil is 7.6-fold (calculated as 538 days / 71 days) longer at 3°C to 6°C compared to at 20°C to 30°C. This implies that the decay rate from the data of Echeonwu et al. (2008) of -0.095 log per day at 21°C to 27°C may be 7.6-fold slower at 3°C to 6°C and equal to -0.0125 log per day. This precautionary value is used here for the purpose of risk assessment. The time taken for a 5-log decay is 400 days.

8.5.6 Individual risk to poultry

The risk of NDV to an individual chicken living on land to which compost has been applied is low at 5.2×10^{-7} per chicken per year without a grazing ban and for surface applied compost (Table 8-26). It should be noted that this risk applies to those poultry actually grazing on land to which compost has been applied, and would not apply to those poultry in commercially housed operations. The relatively slow decay rate used for NDV in soil (at 3°C to 6°C) equates to a 1-log decay in 80 days. This means that the implementation of a three

week ban on chicken grazing has relatively little impact, roughly halving the annual risk. A two month ban reduces the risks fivefold. Tilling the compost in to a depth of 10 cm reduces the risk to $<10^{-8}$ per chicken per year even with no grazing ban.

Table 8-26 Individual risks* (risk per chicken per year) predicted for NDV to a chicken grazing on fields to which compost has been applied

Length of grazing ban	Surface application (no dilution)	Minimum tillage (10 cm)	Plough depth (25 cm)
No ban	5.2×10^{-7}	7.0×10^{-9}	2.8×10^{-9}
3 week	2.9×10^{-7}	3.8×0^{-9}	1.5×10^{-9}
2 month	9.1×10^{-8}	1.2×10^{-9}	4.8×10^{-10}

*Note predicted individual risks apply to a chicken spending all day, every day for 1 year on land to which compost has been applied once at 20 tds/ha

8.6 Bovine spongiform encephalopathy (BSE)

8.6.1 Source term

The amount of BSE-infected material entering the food chain has been decreasing steadily relative to 2001, due to the year-on-year fall in BSE incidence, although there is some variability in the data due to changes in abattoir controls which impact the food chain (Adkin *et al.*, 2010).

8.6.1.1 Estimate of the amount of BSE infectivity entering the food chain

A risk assessment has been developed to evaluate the impact of changes in BSE control measures for GB (Adkin *et al.*, 2010). The assessment includes all the routes by which edible portions of meat may plausibly be contaminated with BSE infectivity and subsequently consumed. The five bovine infectious tissues included are brain, spinal cord, dorsal root ganglia (DRG), other peripheral nervous system (PNS) tissues and tonsillar material.

The assessment has been amended specifically for this risk assessment to estimate the contribution by each contamination route of infectivity entering the food chain, including that which may not be consumed, i.e. including plate wastage.

Given the estimated number of animals by-passing SRM (Specified Risk Material) controls, and the distribution of infectivity within those animals, the model estimates the total amount of infectivity entering the food chain annually. The mean results estimated for 2008, together with the minimum, maximum, 5th and 95th percentiles which include the uncertainty and variability associated with the result are shown in Table 8-27.

Table 8-27: Estimates of BSE infectivity entering the food chain in 2008 (Bovine oral ID_{50} per year) (Adkin, pers. comm.)

Tissue type	Infectivity entering the food chain (Bovine oral ID_{50} per year)				
	mean	min	max	5th	95th
Total	99.79	0.00	727.44	4.44	260.23

The mean infectivity leaving the abattoir is estimated to be 99.79 bovine oral ID_{50} units for 2008. Of this, less than 1% is DRG, with the tonsils and peripheral nervous system tissue in the carcass making the major contribution.

The original risk assessment included imported meat. This is not considered here because it is assumed to be balanced by exports.

8.6.1.2 Total BSE loading going to compost

The total amount of beef (cooked and raw) in waste is an estimated 45,234 tonnes in the UK (Table 2-2). This is 4.3% of the total 1,052,000 tonnes purchased annually in the UK (Table 2-2). Using the 95th percentile of 260.23 bovine oral ID_{50} units as a worst-case for the year 2008 entering the food chain (Table 8-27), then 4.3%, i.e. 11.2 bovine oral ID_{50} units per year would theoretically be composted.

8.6.1.3 Concentration of BSE in compost and soil

Assuming there is no destruction of BSE by composting, the estimated concentration of BSE in compost, using the 95th percentile, is calculated as 11.2 bovine oral ID_{50} units in 3,913,200 tds of compost (Section 2.4.1) which is 2.86 x 10^{-6} bovine oral ID_{50} units/tonne (Table 8-28).

Table 8-28: Predicted concentration of BSE in compost

Pathogen	Oral ID_{50} in total compost per year	Concentration in compost (Oral ID_{50} per tonne)
BSE	11.2	2.86 x 10^{-6}

Assuming that there is no decay of the BSE agent in the soil and tillage to a depth of 10 cm, the BSE concentration in the soil is constant over time at 3.8 x 10^{-8} bovine oral ID_{50}s per tonne soil. Theoretically, in the absence of decay, infectivity could accumulate from year to year in the soil, and under the current legal framework compost could be applied to the surface of grazed land in consecutive years. The cumulative risk is not considered here because BSE prevalences for future years are not known, and tilling or ploughing of the land could occur between years, which together with other soil disturbances would dilute any residual infectivity. Furthermore, given that the risk assessment assumes that each animal eats undiluted compost for a whole year (in the case of surface-applied compost), the residual BSE infectivity in soil remaining from previous years would make a relatively low contribution to the total exposure.

8.6.2 Risk to individual cattle

A bovine consuming 1.125 kg soil per day would ingest 3.81 x 10^{-8} x 365 x 0.001125 = 1.57 x 10^{-8} bovine oral ID_{50}s per year. The risk of BSE would therefore be 0.69 x 1.57 x 10^{-8} = 1.08 x 10^{-8} per animal per year (Table 8-29). This is the individual risk of infection for cattle grazing for 365 days on soil to which compost was applied at 20 tds/ha and tilled into a depth of 10 cm. For surface applied compost, the individual risk would be 75-fold higher at 0.81 x 10^{-6} per animal per year (Table 8-29). It should be noted that this individual risk is based on the assumption that for surface application there is no dilution (Table 3-5) of the compost in the soil over the year, such that cattle are consuming 1.125 kg of compost each day per animal for a period of one year.

Table 8-29: Predicted BSE risks to cattle in GB from application of compost

Soil application method	*Individual risks (risk per animal per year)	Number of BSE infections per year in GB	Number of years between each BSE infection in GB
Surface applied	0.8 x 10^{-6}	0.09	7
10 cm depth	1.1 x 10^{-8}	0.0013	511
25 cm depth	0.43 x 10^{-8}	0.0005	1,278

*Note predicted individual risks apply to a cow spending all day, every day for 1 year on land to which compost has been applied once at 20 tds/ha

8.6.3 Estimated number of BSE infections in GB per year

With an application rate of 20 tds compost /ha, 3,913,200 tds could cover 195,660 ha. Of this 58.2%, i.e. 113,897 ha, is assumed to be grassland for grazing. The maximum cattle density is 1.59 bovines/ha (Table 3-14). Therefore, the total number of cattle exposed to the risk of 1.1×10^{-8} per animal per year (with tillage to 10 cm depth) is 181,096 (Table 7-5).

The expected number of BSE infections from compost tilled into a depth of 10 cm is therefore estimated as 0.002 per year in GB, which is 1 every 511 years (Table 8-29).

For surface applied compost, there is no dilution in the soil. The predicted risk from surface applied compost would be 75-fold higher (compared to minimum tillage to 10 cm depth) at 0.15 BSE infections per year (Table 8-29). This is 1 new infection in GB every 7 years. For surface applied compost, it is assumed that all 181,096 cattle grazing on that land to which 58.2% of the entire GB compost has been applied eat compost (with no soil for a whole year). Thus 181,096 cattle ingesting 1.125 kg compost per day ingest a total of 74,362 tonnes of compost per year. This is 1.9% of the total of 3,913,200 tds produced (Table 7-4).

With tilling to plough depth, the predicted risk is one BSE infection in GB every 1,278 years (Table 8-29).

8.6.3.1 Comparing the BSE risks from compost with those of sewage sludge

The BSE risks predicted for surface applied compost are lower than those previously estimated for sewage sludge by Gale and Stanfield (2001). Assuming that 1% of brain and spinal cord was lost to the sewer from abattoirs, Gale and Stanfield (2001) predicted a risk of BSE transmission of 7.1×10^{-5} per animal per year for cattle grazing on land to which sewage sludge had been applied. This is some 87-fold higher than that of 0.81×10^{-6} per bovine per year predicted for compost applied to the soil surface. Furthermore the risk of 7.1×10^{-5} per animal per year is for sewage sludge that has been tilled into a depth of 25 cm. Although not directly comparable because of different prevalences used in the two risk assessments, it is worth noting the level of risk which was deemed acceptable through an environmental route in 2001. It is also noteworthy that the predicted risk through sewage sludge is based on a bovine oral ID_{50} of 0.1 g of bovine brain. The recent data of Lasmézas *et al.* (2005) suggest the arithmetic mean bovine oral ID_{50} may be much lower than 0.1 g, such that the predicted BSE risks through sewage sludge would be considerably higher than of 7.1×10^{-5} per animal per year. It is documented that two of the prion diseases, namely scrapie in sheep and chronic wasting disease (CWD) in deer and elk are transmitted through environmental routes (Miller *et al.* 2004), whilst environmental transmission is not considered a significant exposure route for BSE in cattle.

8.6.4 Summary

The risks of BSE through application of catering waste-derived compost are considered to be very low. The total number of confirmed BSE cases since 1986 in GB is 179,155 (Defra 2012). Since 1992 the number of confirmed cases of BSE in cattle in GB has declined from 36,680 per year in 1992 to 0 in 2010 and 2011. One case was confirmed in 2008 and one in 2009. The number of confirmed cases in GB in 2002, for which the source term for the risk assessment was estimated, was 445 (Defra 2012). This is 230,000-fold higher than the 0.00196 infections predicted through compost (tilled into 10 cm depth).

8.7 Scrapie

Since the original risk assessment, a number of changes have occurred which may affect the outputs of this study. The original risk assessment did not differentiate between the two forms of scrapie that exist in the UK: classical scrapie and atypical scrapie. Atypical scrapie was first identified in Norway in 1998 and initial research has indicated that the tissue distribution of the prion agent is significantly more limited than that of classical scrapie (Benestead *et al.* 2003). The first case of atypical scrapie to be detected in GB was in 2002, although one case has been retrospectively identified from 1989 (Bruce *et al.* 2007) and a recent immunohistochemical study has revealed that atypical scrapie has existed in the UK since at least 1987 (Webb *et al.* 2009). In addition to advances in scientific knowledge, there have been changes in the prevalence of classical scrapie. The implementation of the National Scrapie Plan (NSP) has produced a noticeable decrease in the annual incidence of classical scrapie in the national flock (Dawson *et al.* 2008).

Here the risks from both classical and atypical scrapie potentially present in compost are considered.

8.7.1 Source Term - Classical Scrapie

Classical scrapie was first recorded around 1750 (Moore et al., 2008). Evidence suggests that the infectious agent for scrapie is comprised of an abnormally folded isoform of a normal cellular protein, PrP^{c}. The misfolded conformation, denoted PrP^{Sc}, is the only disease-specific macromolecule identified in these diseases. Infection with scrapie prions follows a familial pattern and is known to be associated with susceptible alleles of the prion protein gene (Ryder *et al.*, 2009). Sheep carrying the VRQ/VRQ or ARQ/VRQ genotype are most susceptible to infection with classical scrapie. Sheep carrying the ARR/ARR genotype are most resistant to classical scrapie (Baylis *et al.*, 2004, Hunter, 1996, EFSA opinion, 2006).

The following input parameters are considered in this risk assessment: (1) prevalence of classical scrapie in the healthy slaughter population; (2) number of infected animals entering the food chain; (3) identification of potentially infectious tissues and the associated titre of infectivity; (4) tissue distribution of scrapie infectivity with age; (5) tissue weights; and (6) utilisation of sheep tissues in the food chain.

8.7.1.1 Prevalence of classical scrapie

The prevalence of infection in the healthy slaughter stream is investigated each year by the abattoir survey, and the most recent report of the abattoir survey results has therefore been used to estimate the prevalence of classical scrapie (Ortiz-Pelaez and Arnold, 2009). The uncertainty associated with the prevalence of infection is described using a BetaPert distribution with parameter values of most likely value of 0.0009, and 95% confidence intervals of 0.00017 and 0.0029.

8.7.1.2 Number of animals infected with classical scrapie entering the food chain

The number of sheep less than one year old entering the GB food chain in 2008 was 14,352,351 (Defra, 2009a). The number of sheep more than one year old entering the GB food chain in that year was 2,344,534 (Defra, 2009a).

The number of sheep infected in each group per year is described in the assessment using a Poisson distribution P(m). The parameter m is obtained by multiplying the number of sheep slaughtered per year with the prevalence of infection. This is a worst-case assumption and assumes that no sheep or lambs are removed from the food chain due to a positive TSE test at the abattoir. Using the Beta-Pert distribution for uncertainty in prevalence described in Section 8.7.1.1 together with the total number of sheep in GB (see above), it is estimated that the mean annual number of classical scrapie-infected sheep that are less than one year

old and enter the GB food chain is 18,581. The mean annual number of classical scrapie-infected sheep that are more than one year old and enter the GB food chain is estimated to be 3,035.

8.7.1.3 Classical scrapie infectious tissues and infectivity titres

When estimating the amount of infectivity from a sheep infected with scrapie that would be included in materials destined for the food chain, it is necessary to take into account both the pathogenesis of the disease (i.e. the tissues likely to be infectious at different stages of the incubation period), the probability that such tissues will be classified as fit for human consumption post SRM controls, and the amount of infectivity present in those tissues. There are a number of tissues identified as carrying scrapie infectivity in sheep, as listed in Table 8-30.

The infectivity of classical scrapie in sheep through the oral route is estimated as described previously (Gale 2002). Thus data for the number of intracerebral ID_{50}s in various tissues from scrapie-infected Suffolk sheep (Kimberlin & Wilesmith, 1994) were used (Table 8-30). Oral ID_{50}s per gram of tissue were estimated assuming that transmission through the oral route is 100,000-fold less efficient than through intracerebral challenge (Kimberlin & Wilesmith, 1994). Thus according to Anon (1994) in mice challenged with the 139A strain of scrapie, the effective exposure by the intragastric route was about 100,000 less efficient than with the intracerebral route, i.e. 1 intragastric ID = 10^5 intracerebral ID. For example, there are $10^{5.6}$ intracerebral ID_{50}/g of medulla oblongata from brains of terminally-affected Suffolk sheep. This is equivalent to 4.0 oral ID_{50}/g (Table 8-30). In effect, the oral ID_{50} for sheep is about 0.25 g scrapie-infected ovine brain medulla oblongata.

More recently Ryder *et al.* (2009) have published data where VRQ homozygous lambs were orally challenged with sheep scrapie brain pool homogenate. The smallest dose used was 1 g which infected 25 of 25 lambs. The sheep oral ID_{50} is therefore <1g of scrapie- infected sheep brain. Unfortunately there are no experimental data for lower oral doses of brain. The mean incubation period for the lambs challenged with 1 g of brain at 14 days of age was 192 days. For older sheep challenged at 245 days of age with 1 g of brain, the mean incubation period was much longer at 345 days. The incubation period for scrapie depends on the dose, and decreases with higher doses as shown for intracerebral challenge of scrapie in mice (Somerville *et al.* 2002). Thus the mean incubation period for 14 day old lambs challenged orally with 5 g of brain homogenate was 185 days (Ryder *et al.* 2009),i.e. seven days shorter than for a challenge of 1 g. Clearly sheep exposed to any residual scrapie infectivity through compost would ingest very small subfractions of an oral ID_{50} due to dilution, dispersion and degradation of tissue during the composting process. This raises the question of how long the incubation period is for scrapie in sheep exposed to small sub-fractions of an oral ID_{50}. Data for BSE suggest that the incubation period does not continue to increase at doses below the ID_{50} and may become constant reflecting the fact there is no threshold dose (see Gale 2006). For this risk assessment it is assumed that the infectivity for scrapie is the same as that in the previous risk assessment as set out in Table 8-30.

The mean infectivity data in Table 8-30 is associated with considerable variability, represented by the standard error. In the absence of any other data allowing quantification of the uncertainty associated with these estimates, the standard error range has been used as a proxy to represent the uncertainty associated with the titre of infectivity. The minimum and maximum standard error is therefore assumed to represent the minimum and maximum uncertainty associated with the mean infectivity titre and is described in the assessment using a BetaPert distribution. Infectivity of the stomach, heart, kidney and adrenal gland is described using a single parameter value as given in Table 8-30.

Table 8-30 Infectious tissues and infectivity titres for classical scrapie

Tissue	Infectivity titre of tissue	
	Log mouse intracerebral ID_{50} g^{-1} tissue	Mean Oral ID_{50} g^{-1} tissue - assumes 1 intragastric ID_{50} = 10^5 intracerebral ID_{50} (Kimberlin & Wilesmith, 1994)
Brain & pituitary gland	5.6 ± 0.2 (51) [1]	4.0
Spinal cord	5.4 ± 0.3 (9) [1]	2.5 [3]
Lymph nodes	4.2 ± 0.1 (45) [1]	0.16 [3]
Spleen	4.5 ± 0.3 (9) [1]	0.32
Tonsil	4.2 ± 0.4 (9) [1]	0.16
Stomach (rumen & reticulum)	2 [2]	0.001 [3]
Liver	2 ± 0.1 (9) [1]	0.001 [3]
Thymus	2.2 ± 0.2 (9) [1]	0.001 [3]
Heart	1 [2]	0.0001
Kidney & adrenal gland	1 [2]	0.0001 [3]
Intestine (duodenum & jejunum)	4.7 ± 0.1 (9) [1]	0.5 [3]

[1] Kimberlin & Wilesmith, (1994): Re-analysis of data from Hadlow and others. Each titre is expressed as the arithmetic mean ± standard error of the mean of (n) samples
[2] Gale (2002)
[3] Cummins & Adkin, (2007)

It should be noted that Hadlow *et al.* (1982) did not test the stomachs of sheep for scrapie infectivity. However, Van Keulen *et al.* (2000) demonstrated the presence of positive immunostaining of PrP^{Sc} in the abomasum at 10 months of age in scrapie-infected animals. Therefore, it has been assumed here that the stomach of scrapie-infected sheep carries infectivity.

Groschup *et al.* (1996) detected scrapie agent in the peripheral nerves of a clinical scrapie-diseased sheep. The levels detected in this single sheep were similar to that observed by Hadlow *et al.* (1982) in the sciatic nerve, inferring that there are potentially low levels of scrapie infectivity in peripheral nerves. However, this conclusion is based on a single clinically affected sheep and may not be applicable, in general terms, to all scrapie infected sheep.

It should be noted that the wall of the ileum has patches of lymphoid tissue, Peyers patches, with high levels of infectivity, with about 0.5 sheep oral ID_{50} g^{-1}. The infectivity titre for the intestine should only represent the duodenum and the jejunum (and not the ileum, as that is considered separately due to it being classified as SRM in sheep of all ages). The current value of 0.5 sheep oral ID_{50} g^{-1} has been chosen since it represents the worst-case scenario (i.e. infectivity being the same as in the ileum).

Peripheral nerves have been excluded from Table 8-30 despite the fact that the PNS has been found to be involved in the pathogenesis of TSE in clinically affected sheep, albeit at very low levels (Groschup *et al.* 1999). However there is little data available to quantify the amount of scrapie in these nerves for TSE incubating animals. Andreoletti *et al.* (2004) observed that the amount of infectious prion protein in the muscle of TSE incubating sheep was very low. The presence of very low levels of infectivity is also true for blood, where recent studies have detected PrP^{Sc} at very low levels from sheep infected with classical scrapie (Terry *et al.* 2009). In the absence of any further information of the titre of infectivity in these tissues, it is concluded that the infectivity levels in PNS/blood are very low when compared to other infectious tissues listed in Table 8-30, and therefore are not included in the quantitative assessment.

8.7.1.4 Age-related distribution of classical scrapie infectivity in ovine tissues

The majority of cases of classical scrapie appear in sheep between 2 and 5 years of age (OIE Terrestrial Manual, 2009). During the progression of the disease, infectivity accumulates in different ovine tissues at different rates. It is also important to note that the vast majority of sheep are slaughtered by the age of 7 years old (Dawson, *pers. comm.*, 2009) and therefore the disease may not be fully developed in all parts of the sheep by the time of slaughter. An estimation of the percentage increase in infectivity at different ages is presented in DNV (2002). In this risk assessment, two age groups are being considered, lambs <1yr and sheep >1 yr. Therefore the percentages are adjusted accordingly and are presented in Table 8-31. For lambs under one year of age, the percentages are the same as those presented in DNV (2002) for lambs over six months of age except for the lymph nodes and the intestine (duodenum and jejunum) which have been lowered from 50% to 40%. For sheep over the age of one year, the percentage of infectivity in all tissues is estimated to lie between 70% and 100%. This range is described in the model by a uniform distribution.

Table 8-31 Distribution of classical scrapie infectivity in ovine tissues with age (adapted from data from DNV, 2002)

Tissue	Percentage of infectivity by age (%)	
	Lamb < one year	Sheep ≥ one year
Brain & pituitary gland	0.10%	70.00 - 100.00%
Spinal cord	0.10%	70.00 - 100.00%
Lymph nodes	40.00%	70.00 - 100.00%
Spleen	10.00%	70.00 - 100.00%
Tonsil	10.00%	70.00 - 100.00%
Stomach (rumen & reticulum)	10.00%	70.00 - 100.00%
Liver	10.00%	70.00 - 100.00%
Thymus	10.00%	70.00 - 100.00%
Heart	10.00%	70.00 - 100.00%
Kidney & adrenal gland	10.00%	70.00 - 100.00%
Intestine (duodenum & jejunum)	40.00%	70.00 - 100.00%

8.7.1.5 Weights of ovine tissues

The weights of certain lamb tissues are presented in Table 8-32, along with the utilisation of these tissues in food. It has been assumed that the weights of tissues in mutton are 1.6-fold greater than in lamb (Gale, 2002). From this, the average weight of tissue per animal used in food can be calculated. It is important to note that some of the weights of lamb tissues that were used in the original risk assessment have been amended with more recent data. Weights for these tissues from the original risk assessment (Gale 2002) were lymph nodes (40g), tonsils (100g), stomach (1000g), liver (650g) and the intestine (1,200g).

Table 8-32 Weights of ovine tissues

Tissue	Weight of tissues (g)	
	Lamb < one year	Sheep ≥ one year
Brain & pituitary gland	100 [1,2,3]	160
Spinal cord	40 [1,2,3]	64
Lymph nodes	38 [3]	60.8
Spleen	100 [1,2,3]	160
Tonsil	2 [3]	3.2
Stomach (rumen & reticulum)	600 [1,3]	960
Liver	610 [1,3]	976
Thymus	50 [1,2,3]	80
Heart	200 [1,2]	320
Kidney & adrenal gland	100 [1,2]	160
Intestine (duodenum & jejunum)	930 [3]	1,488

[1] Hart et al. (1997).
[2] Gale (2002).
[3] Fryer et al. (2007). Supplementary material.

8.7.1.6 Utilisation of ovine tissues in food

The model here takes account of SRM controls for sheep (as did the original compost risk assessment). The utilisation of ovine tissues in food is presented in Table 8-33. No parts of the spleen or tonsils enter the food chain as these tissues are classified as SRM for sheep of all ages. The brain and spinal cord are classified as SRM for sheep over 12 months so it is assumed that these tissues for mutton do not enter the food chain. Some values that were used in the original risk assessment have been updated with more recent data. For both lamb and mutton the proportions of tissue going into the food chain in the original risk assessment were assumed to be the following: lymph nodes (100%), stomach (10%), liver (100%), thymus (100%) and the intestine (90%). The updated values are given in Table 8-33. In practice, all of the spinal cord should be removed for sheep over 1 year of age and therefore should not go to the food chain. For sheep less than 1 year of age the model assumes that 20% of spinal cord material goes to the food chain. The figure of 20% was from a DNV study in 2002 and is also used in the Gale (2002) risk assessment.

Table 8-33 Utilisation of ovine tissues in food

Tissue	Utilisation in food	
	Lamb < one year	Sheep ≥ one year
Brain & pituitary gland	5% [2]	0% [2,3]
Spinal cord	20% [2]	0% [2,3]
Lymph nodes	80% [3]	80% [3]
Spleen	0% [2,3]	0% [2,3]
Tonsil	0% [2,3]	0% [2,3]
Stomach (rumen & reticulum)	17% [1,3]	17% [1,3]
Liver	85% [3]	85% [3]
Thymus	5% [3]	5% [3]
Heart	50% [1,2]	50% [1,2]
Kidney & adrenal gland	100% [1,2]	100% [1,2]
Intestine (duodenum & jejunum)	85% [3]	85% [3]

[1] Hart et al. (1997).
[2] Gale (2002).
[3] Fryer et al. (2007). Supplementary material.

8.7.1.7 Titre of infectivity entering the food chain from classical scrapie per year

Each of the input parameters is multiplied by tissue type and by the two age groups (under one, and greater than one year old) to ascertain the total amount of infectivity entering the food chain per year. For the intestine the assumption is made that there is a 100-fold reduction in infectivity during the cleaning process (DNV 2002).

The stochastic model was run 25,000 times (or iterations) using the software package @Risk (© Palisade) Version 5.0, an add-on package within Microsoft Excel (© Microsoft). The results presented follow the standard form of the arithmetic mean and the 5th and 95th percentile values.

8.7.2 Source term – Atypical scrapie

Atypical scrapie or 'Nor-98' was first identified in Norway in 1998 (Benestad *et al.,* 2003). The first case of atypical scrapie to be detected in GB was in 2002 although it has existed in the UK since at least 1987 (Webb *et al.* 2009). Sheep most at risk of atypical scrapie are of genotype AHQ/AHQ, $AF_{141}RQ/AF_{141}RQ$ and $AF_{141}RQ/AHQ$ (Dawson *et al.,* 2008).

Previous studies have suggested that atypical scrapie is restricted to the central nervous system (brain and spinal cord), although there is some speculation that infection can also occur in the lympho-reticular system (LRS) (SEAC Sheep Subgroup position statement 2006). APHA have a number of studies looking at LRS involvement in atypical scrapie and to date have been unable to identify this. In both field cases and experimental challenge, the LRS in clinical disease is negative by rapid test (ELISA) and by IHC - so no detectable prion protein (PrP). Bioassays are ongoing. Although they are not complete, they have been running for a considerable time period, and it can already be concluded with some confidence that if infectivity exists in the peripheral tissues, it is probably 5 or 6 logs less than in the CNS.

8.7.2.1 Prevalence of atypical scrapie

The most recent report of the abattoir survey has been used to estimate the prevalence of atypical scrapie (Ortiz-Pelaez, pers. comm., 2009). However, there are a number of key assumptions applied in producing this estimate: (1) the incubation period of atypical scrapie is the same as that estimated for classical scrapie; (2) the survivability of sheep infected with atypical scrapie is the same as for classical scrapie; and (3) the sensitivity of the test for atypical scrapie is the same as for classical scrapie (Ortiz-Pelaez, pers. comm., 2009).

The parameter values are described in the assessment using a BetaPert distribution of most likely value of 0.0024 with 95% confidence intervals of 0.00085 to 0.0051.

8.7.2.2 Number of animals infected with atypical scrapie entering the food chain

The number of sheep less than one year old entering the GB food chain in 2008 was 14,352,351 (Defra, 2009a). The number of sheep more than one year old entering the GB food chain in that year was 2,344,534 (Defra, 2009a).

The number of infected sheep is described in the model using a Poisson distribution. The worst-case assumption is made that no sheep or lambs are removed from the food chain due to a positive TSE test at the abattoir.

Using the Beta-Pert distribution for uncertainty in prevalence described in Section 8.7.2.1 together with the total number of sheep in GB (see above), it is estimated that the mean annual number of atypical scrapie-infected sheep that are less than one year old and entering the GB food chain is 39,543. The mean annual number of atypical scrapie-infected sheep that are more than one year old and entering the GB food chain is estimated to be 6,460.

8.7.2.3 Atypical scrapie infectious tissues and infectivity titres

The number of potentially infectious tissues for atypical scrapie has been found to be more restricted than for classical scrapie. Previous studies suggest that infection is limited to the central nervous system (Benestad *et al.* 2008). However, there is speculation that infection can also occur in the lymph nodes. The model considers infection of both the CNS and lymph nodes. The assumption is made that no other peripheral tissues contain infectivity. For those tissues which are infected, the titre of infectivity is not known. Therefore, for CNS tissues, it is assumed that the titre is the same as that measured for the CNS for classical scrapie as presented in Table 8-30. If infection is present in the peripheral tissues then the titre of infectivity is estimated to be 5 to 6 logs less than in CNS tissues (Simmons, per. comm., 2009). Therefore the lymph nodes have been assigned an infectivity titre of 1 log mouse intracerebral ID_{50} g^{-1}. The infectious tissues and infectivity titres are presented in Table 8-34.

Table 8-34 Atypical scrapie infectivity titres

Tissue	Infectivity titre of tissue	
	Log mouse intracerebral ID_{50} g^{-1} tissue	Mean Oral ID_{50} g^{-1} tissue - assumes 1 intragastric ID_{50} = 10^5 intracerebral ID_{50} (Kimberlin & Wilesmith, 1994)
Brain & pituitary gland	5.6 ± 0.2 (51)	4.0
Spinal cord	5.4 ± 0.3 (9)	2.5
Lymph nodes	1	0.0001

Similarly to the assessment for classical scrapie, the standard error range from Kimberlin & Wilesmith, (1994) has been used as a proxy to represent the uncertainty associated with the titre of infectivity for CNS tissues. The minimum and maximum standard error is assumed to represent the minimum and maximum uncertainty associated with the mean infectivity titre for atypical scrapie and is described in the assessment using a BetaPert distribution.

8.7.2.4 Distribution of atypical scrapie infectivity in ovine tissues with age

There is increasing evidence that atypical scrapie is not transmitted in the field to the same degree as classical scrapie. A French study has found no evidence of clustering of atypical scrapie cases on infected farms, which suggests that the disease may spontaneously occur or is not particularly contagious as compared to classical scrapie (Fediaevsky *et al.* 2009). However, there is no data on the transmission of atypical scrapie through composted materials which may contain infectious tissues. Therefore, the results from this risk assessment for atypical scrapie should be viewed with caution as they represent a transmission route which has yet to be experimentally confirmed. The incubation period of atypical scrapie is at least 2 – 3 times longer than for classical scrapie based on experimental research (M. Simmons, APHA, pers. comm., 2009). This would fit with the presenting age of clinical suspects, and the distributions of infectivity with age for CNS tissues and lymph nodes as presented in Table 8-34 have been adjusted accordingly.

For lambs under one year old, the percentages for CNS tissues remain at 0.1%. For the lymph nodes, the percentage of infectivity is lowered to 0.1% as there is no evidence to suggest that the progression of the disease is faster in these tissues than in the CNS. For sheep over the age of one year, the percentage of infectivity in all of the tissues at the time of slaughter is estimated to lie between 40% and 80%. This range is described in the model using a uniform distribution.

8.7.2.5 Titre of infectivity entering the food chain from atypical scrapie per year

In a similar manner to the model framework for classical scrapie, each of the input parameters is multiplied by tissue type and by the two age groups (under one, and greater than one) to ascertain the total amount of infectivity entering the food chain per year.

8.7.3 Results of estimation of the amount of scrapie infectivity entering the food chain and compost

The mean amount of classical scrapie entering the food chain per year is estimated at 10.9 x 10^4 sheep oral ID_{50}, with 90% certainty that this value lies between 2.0 x 10^4 and 2.4 x 10^5. A sensitivity analysis, based on the correlation coefficient, was carried out for this output, and the uncertainty associated with the prevalence of disease was found to significantly affect the final result.

For atypical scrapie, the mean amount entering the food chain per year is estimated at 1700 sheep oral ID_{50}, with 90% certainty that this value lies between 560 and 3200. A sensitivity analysis, based on the correlation coefficient, was performed for this result. As was observed for the classical scrapie component of the model, the uncertainty associated with the prevalence of disease was found to significantly affect the final result.

Since meat may be treated differently to spinal cord by the consumer, total infectivity of scrapie is calculated separately for the meat fraction (i.e. all tissues except spinal cord) and the spinal cord fraction.

The mean results per year, together with the 5th and 95th percentiles which include the uncertainty and variability associated with the result are shown in Table 8-35 for classical scrapie and for atypical scrapie. Total amounts of infectivity for classical and atypical scrapie are also shown. The estimated mean of the classical and atypical scrapie combined entering the food chain is 110,467 sheep oral ID_{50} in 2008. This compares with an estimated mean infectivity leaving the abattoir for BSE of just 99.79 bovine oral ID_{50} units for 2008 (Section 8.6.1.1).

Table 8-35 Estimates for infectivity titres of classical scrapie, atypical scrapie and both types combined scrapie entering the food chain in 2008 (sheep oral ID_{50} per year)

Tissue type	Infectivity entering the food chain (sheep oral ID_{50} per year)		
	mean	5th	95th
Classical scrapie			
All tissues except spinal cord	108,400	20,300	236,000
Spinal cord	394	68	915
Total All tissues	108,800	20,400	237,000
Atypical scrapie			
All tissues except spinal cord	835	275	1,625
Spinal cord	838	260	1,720
Total All tissues	1,673	560	3,240
Classical and atypical combined			
All tissues except spinal cord	109,235	21,200	236,000
Spinal cord	1,232	495	2,280
Total All tissues	110,467		

8.7.3.1 Scrapie loadings going to compost

It should be noted that scrapie infectivity is inactivated at lower temperatures than the more thermally-resistant BSE agent (Somerville *et al.* 2002). It is therefore plausible that there will be some destruction of scrapie infectivity both by cooking and by composting (see Section 8.7.4). However, it is assumed here that there is no destruction of scrapie agent by cooking in the kitchen. This was considered appropriate by Robert Somerville (Roslin Institute, Edinburgh) who noted that meat is often unevenly cooked so that the inside is much cooler than the outside. Therefore the risk assessment here uses the total amount of lamb going into the compost, and does not take into account that only 18.5% is raw (Table 2-3). The total amount of lamb (cooked and raw) in waste is an estimated 27,606 tonnes in the UK per year (Table 2-2). This is 5.89% of the total 469,000 tonnes purchased annually in the UK (Table 2-2). In addition, it is assumed that all spinal cord in food is discarded to waste rather than being eaten.

Classical scrapie (95th percentile)

Using the 95th percentile of 236,000 classical scrapie sheep oral ID_{50} units per year entering the food chain in all tissues except spinal cord as a worst-case (Table 8-35), then 5.89%, i.e. 13,891 sheep oral ID_{50} units per year would be composted. In addition, 100% of the 915 classical scrapie sheep oral ID_{50} (95th percentile) (Table 8-35) in spinal cord are also composted. The 95th percentile for the total infectivity going to composting for classical scrapie is therefore 14,806 sheep oral ID_{50} units per year.

Atypical scrapie (95th percentile)

Using the 95th percentile of 1,625 atypical scrapie sheep oral ID_{50} units per year entering the food chain in all tissues except spinal cord as a worst-case (Table 8-35), then 5.89%, i.e. 95.6 sheep oral ID_{50} units per year would be composted. In addition, 100% of the 1,720 atypical scrapie sheep oral ID_{50} (95th percentile) (Table 8-35) in spinal cord are also composted. The 95th percentile for the total infectivity going to composting for atypical scrapie is therefore 1,816 sheep oral ID_{50} units per year.

8.7.4 Evidence for degradation of scrapie prion protein during composting

In the original risk assessment, it was assumed that composting would have no effect on the scrapie agent (Gale 2002). Since then Huang *et al.* (2007) have investigated whether the abnormal prion protein (PrP^{Sc}) in tissues from sheep with scrapie would be destroyed by composting. Their experiments used much longer timescales than in the current ABP

Regulations with temperatures above 60°C for about 2 weeks, and infective material being buried in the compost for up to 148 days. In one of the two experiments, PrP^{Sc} was not detected after composting in the tissue remnants or the surrounding sawdust. In the second experiment, 1 of 5 specimens tested negative after composting, whereas PrP^{Sc} was detected in the other four bags, although in greatly reduced amounts compared with those before composting. The microbial diversity was greater in experiment 1 than in experiment 2. Huang *et al.* (2007) interpreted their results in terms of composting as a means for degrading PrP^{Sc} in carcass and other wastes. There is further evidence (Hinckley *et al.* 2008) that microbial activity may degrade PrP^{Sc} from experiments undertaken in mesophilic anaerobic digestion (MAD). Data from Hinckley *et al.* (2008) showed a decrease in the intensity of the extractable PrP^{Sc} signal with time in the MAD process. This reduction in intensity over time did not occur in autoclaved sludge. Autoclaving destroys both proteolytic enzymes and microbes, suggesting microbes together with their secreted proteases are degrading PrP^{Sc} in MAD over the 20 day period. Similarly γ-ray irradiation, which kills bacteria, also prevented the reduction in intensity to some degree, again suggesting the importance of bacteria.

On the basis of the evidence suggesting that microbial activity in MAD can result in degradation of PrP^{Sc} and that composting also results in loss of PrP^{Sc} (Huang *et al.* 2007) it is considered appropriate to allow for some reduction of scrapie infectivity in the composting process. Both the studies of Huang *et al.* (2007) in compost and Hinckley *et al.* (2008) in MAD used protein blotting techniques to test for loss of PrP^{Sc} (the abnormal prion protein). The results are semi-quantitative in that the degradation of PrP^{Sc} was estimated from the intensity of bands. In the immuno-blotting results of Huang et al. (2007), very intense bands rated as ++++ or +++ were observed at t = 0. After composting, either no band was visible (-) or only 1 or 2 bands were visible (+).

It should be noted that analysing for PrP^{Sc} is not the same as testing for loss of infectivity. Indeed, one potential criticism of the results of Hinkley *et al.* (2008) and Huang et al. (2007) is that antibodies were used to detect part of a protein, albeit PrP^{Sc}. However, this does not mean the protein is still infectious. More recently, quantitative methods based on detection of infectivity (rather than parts of a protein) were used to study survival of PrP^{Sc} from a mouse-adapted scrapie in mesophilic anaerobic digestion (Miles *et al.* 2011). Those authors observed a 2.43-log reduction in PrP^{Sc} under mesophilic conditions (37°C) after 15 days and a 3.41-log reduction after 10 days under thermophilic conditions (60°C). Of great importance is that the experimental approach of Miles et al. (2011) used the standard scrapie cell assay (SSCA) which detects infectivity and determines the quantity of infectious prions. In contrast, Hinkley et al. (2011) used antibodies to detect protein (namely PrP^{TSE}) which is not the same as detecting infectivity. Indeed it could be argued the assay of Miles et al. (2011) is more appropriate for the purpose of risk assessment than that of Hinkley et al. (2008) because infectivity is the parameter of interest, rather than the detection of part of a protein.

In buffer solutions, Miles *et al.* (2011) reported a 1.13-log reduction after 15 days at 37°C and a 1.80-log reduction after 10 days at 60°C. Miles *et al.* (2011) also presented data showing greater reduction of scrapie prions in biosolids than in control buffers at the same temperatures, suggesting that factors other than temperature were playing a role in the loss of infectivity of the prions in the biosolids. For catering waste derived compost, the bulk of the meat undergoes 4 days at 60°C (see Section 4.0) and assuming a linear log decay with time a 1.80-log reduction in buffer at 60°C in 10 days (see Miles *et al.* 2011) would represent a 0.72-log reduction in 4 days. Taking a precautionary approach it is here it is assumed that composting removes 80% (0.69-log removal) of the scrapie agent. This is considered a worst-case assumption because most compost operations would continue for longer than 4 days and no account is taken of inactivation by proteolytic and microbiological

processes in the composting process. In the sensitivity analysis (Section 9.10), the risks are calculated assuming that composting removes 2.43-log of scrapie infectivity, which may be a more realistic estimate than the 0.69-log removal used in the baseline assessment.

8.7.5 Predicted concentration of scrapie in compost and soil

Assuming there is a 0.69-log removal of scrapie by composting, the estimated concentration of classical scrapie in compost, using the 95th percentile, is calculated as 3,023 sheep oral ID_{50} units in 3,913,200 tds of compost (Section 2.4.1) which is 7.7 x 10^{-4} sheep oral ID_{50} units/tds (Table 8-28). Similarly the concentration of atypical scrapie (95th percentile) is 9.5 x 10^{-5} sheep oral ID_{50} units/tds (Table 8-36).

Table 8-36 Predicted concentration of scrapie in compost

Pathogen	Statistic	Sheep oral ID_{50} in total compost per year	Concentration in compost (sheep oral ID_{50} per tds)
Classical scrapie	95th Percentile	3,023	0.00077
Atypical scrapie	95th Percentile	371	0.000095

Assuming that there is no decay of the scrapie agents in the soil over the 3 week no grazing period for sheep, and tillage to a depth of 10 cm, the scrapie concentrations in the soil are constant over time at 1.0 x 10^{-5} and 1.3 x 10^{-6} sheep oral ID_{50}s per tonne soil (95th percentiles) for classical scrapie and atypical scrapie respectively.

Theoretically, in the absence of decay, infectivity could accumulate from year to year in the soil and under the current legal framework compost could be applied to the surface of grazed land in consecutive years. The cumulative risk is not considered here because scrapie prevalence for future years is not known and could change with changing PrP genotype. Tilling or ploughing of the land could occur between years, which together with other soil disturbances would dilute any residual infectivity. Furthermore, given that the risk assessment assumes that each animal eats undiluted compost for a whole year (in the case of surface applied compost), the residual scrapie infectivity in soil remaining from previous years would be expected to make a relatively low contribution to the total exposure.

8.7.6 Risk to individual sheep

The sheep oral ID_{50}s are those for scrapie in susceptible sheep and no assumption is made about the PrP genotype, i.e. the individual risks calculated here are for susceptible sheep, whereas the individual risks would actually be negligible for resistant sheep. For classical scrapie, a sheep consuming 0.69 kg soil per day would therefore ingest 1.0 x 10^{-5} x 365 x 0.00069 = 2.6 x 10^{-6} sheep oral ID_{50}s per year (95th percentile). The risk of classical scrapie would therefore be 0.69 x 2.6 x 10^{-6} = 1.8 x 10^{-6} per animal per year (Table 8-37). This is the individual risk of infection for sheep grazing for 365 days on soil to which compost was applied at 20 tds/ha and tilled into a depth of 10 cm. For surface applied compost, the individual risk would be 75-fold higher at 1.34 x 10^{-4} per animal per year (Table 8-37). It should be noted that this individual risk is based on the assumption that for surface application there is no dilution (Table 8-37) of the compost in the soil over the year, such that sheep are consuming 0.69 kg of compost each day per animal for a period of one year. The individual risks to sheep grazing on compost-amended soil from atypical scrapie are about a factor of ten lower than those for classical scrapie (Table 8-38).

Table 8-37 Predicted risks of classical scrapie to sheep in GB from application of compost. Calculations based on 95th percentile classical scrapie titres estimated in food (Table 8-35)

Soil application method	*Individual risks (risk per animal per year)	Number of scrapie infections per year in GB	Number of years between infections
Surface applied	1.34×10^{-4}	14.7	0.07
10 cm depth	1.8×10^{-6}	0.2	5.1
25 cm depth	7.2×10^{-7}	0.078	12.8

*Note predicted individual risks apply to a sheep spending all day, every day for 1 year on land to which compost has been applied once at 20 tds/ha.

Table 8-38 Predicted risks of atypical scrapie to sheep in GB from application of compost. Calculations based on 95th percentile scrapie titres estimated in food (Table 8-35)

Soil application method	*Individual risks (risk per animal per year)	Number of scrapie infections per year in GB	Number of years between infections
Surface applied	1.65×10^{-5}	9.6	0.10
10 cm depth	2.2×10^{-7}	0.13	7.8
25 cm depth	8.8×10^{-8}	0.051	19.6

*Note predicted individual risks apply to a sheep spending all day, every day for 1 year on land to which compost has been applied once at 20 tds/ha.

8.7.7 Estimated number of scrapie infections in GB per year

With an application rate of 20 tds compost /ha, 3,913,200 tds could cover 195,660 ha, of which 58.2% i.e. 113,897 ha are grassland. The maximum sheep density is 5.1 animals/ha (Table 3-14). Therefore, the total number of sheep grazing (for the period of one whole year) on land to which to compost has been applied is 580,875 (Table 7-5).

However, of 579 sheep randomly selected from the GB scrapie surveillance surveys (January to December 2009) (Weston 2009) only 109 had genotypes susceptible to classical scrapie (i.e. NSP Group IV, V and ARQ/ARQ Type III). Thus only 18.8% (i.e. 109/579) of the GB sheep population is susceptible to classical scrapie. It is therefore assumed here that 580,875 x 0.188 = 109,204 sheep grazing for one year on land to which compost has been applied are susceptible to classical scrapie, each with an individual risk of infection of 1.8×10^{-6} per sheep per year (Table 8-37) assuming tilling to 10 cm depth. The predicted number of classical scrapie infections per year is therefore $1.8 \times 10^{-6} \times 109{,}204 = 0.19$ (95^{th} percentile) in the GB. This is one infection every 5.1 years.

For surface applied compost, there is no dilution in the soil. The predicted risk from surface applied compost would be 75-fold higher (compared to minimum tillage to 10 cm depth) at 14.7 classical scrapie infections per year (Table 8-37). For surface applied compost, it is assumed that all 580,875 sheep grazing on that land to which the compost has been applied eat compost (with no soil) for a whole year. Thus 580,875 sheep ingesting 0.69 kg compost per day ingest a total of 146,293 tonnes of compost per year. This is 3.74% of the total of 3,913,200 tds produced (Table 7-4).

With tilling to plough depth (0.25 m), the predicted risk is 0.078 classical scrapie infections in GB every per year. This is one every infection every 13 years (Table 8-37).

The risks for atypical scrapie calculated using the 95^{th} percentiles for loadings in food are presented in Table 8-38. It is assumed that 100% of the flock, i.e. all 580,875 sheep grazing on compost-treated land are susceptible to atypical scrapie in GB. Despite this, the predicted

numbers of infections are lower by about a third than predicted for classical scrapie. With tilling in to 10 cm depth, one infection of atypical scrapie is predicted every 8 years.

8.7.8 Summary

The risk assessment predicts 14.7 new classical scrapie infections per year in GB (Table 8-37) and 9.6 new atypical scrapie infections (Table 8-38). This is based on the assumption that the compost is not tilled in such that each sheep is ingesting 0.69 kg soil per day for a year. These are 95th percentiles. With tilling into a depth of 10 cm, the risk assessment predicts just 0.195 and 0.13 new infections for classical and atypical scrapie respectively per year. This is 0.325 new infections combined.

Scrapie is an endemic disease in the UK. Monte Carlo simulations are undertaken here using the number of sheep entering the GB food chain per year and the estimated prevalences for classical and atypical scrapie (see Sections 8.7.1.2 and 8.7.2.2) estimate 67,619 scrapie-infected sheep entering the GB food chain annually. These represent the current "natural" background in GB. The additional 0.325 infections per year predicted through application of compost would be just 0.0005% of the underlying number of total infections in GB.

It should be noted that the estimate of 67,619 infections per year refers to the total number of infected sheep in the population going to slaughter per year rather than the number of new infections per year. It is calculated as the product of the number of lambs and ewes slaughtered per year (16,696,885 see Section 8.7.1.2) and the estimated prevalence of both classical scrapie and atypical scrapie in those age groups. On the basis that there may be some 30,000,000 sheep in England and Wales (see Gale 2002), the number of background scrapie infections may be nearer 140,000.

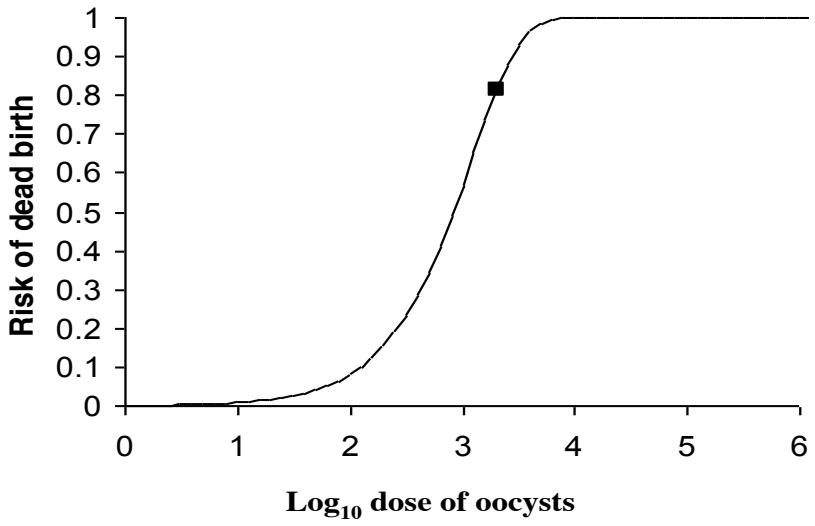
8.8 QRA for protozoan parasites such as Toxoplasma

8.8.1 Toxoplasma gondii

8.8.1.1 More recent data on dose-response for T. gondii in livestock

Toxoplasma is endemic in the UK (G. David, APHA *pers. comm.*). *T. gondii* is the second most common cause of abortion in sheep in GB (Mearns 2007). The original compost risk assessment used a dose-response curve for the risk of stillbirth in pregnant sheep. It was based on data cited by Buxton (1998) stating that when susceptible pregnant sheep were challenged orally with a dose of 2,000 *T. gondii* oocysts, fewer than 18% of lambs were born alive. Using that single point, a negative exponential dose-response curve with an LD_{50} of 800 oocysts (r = 0.00085) was used. This is shown in Figure 26. Dose-response curves for *Cryptosporidium* oocysts are typically represented by negative-exponential dose-response curves (Teunis *et al.* 2002). Although it is acceptable to fit a negative exponential model to a single data point, the lack of data for lower doses adds to uncertainly in the model in Figure 26. In particular, it cannot be ruled out that there is not a more susceptible subpopulation. Therefore WRAP asked APHA to look for additional dose-response data for *T. gondii* with emphasis on data more recent than those cited in Buxton (1998).

Figure 26: Negative exponential dose-response curve (r = 0.00085) for risk of dead birth in sheep from oral exposure to *T. gondii*. Data from Buxton (1998) who cited an experiment in which less than 18% of births were live after an oral dose of 2,000 oocysts. This is plotted as the point. Taken from Gale (2002)



An ISI Web of knowledge search was conducted for the following terms:

- "*Toxoplasma gondii*, dose response";
- "*Toxoplasma gondii*, infection, sheep"; and
- "*Toxoplasma gondii*, infection, sheep, dose".

The search revealed prevalence data for different countries, mainly serological and seroprevalence results, together with some papers on dose-response in livestock. For example, Esteban-Redondo *et al.* (1999) compared infection in the tissues of sheep and cattle following oral challenge with doses of 10^3 and 10^5 oocysts of *T. gondii*. Their results showed that sheep were more susceptible than cattle with more oocysts being detected in tissues at the higher dose. Esteban-Redondo and Innes (1998) used PCR to examine the presence of *T. gondii* in sheep tissues after experimental infection with oral doses of 10^3, 10^4 and 10^5 oocysts.

Bartova *et al.* (2004) obtained oral dose-response data for *T. gondii* in domestic ducks by investigating doses between 10 and $10^{5.7}$ oocysts administered orally through a micropipette. The authors observed that the ID_{50} for *T. gondii* in ducks is very low and a dose of just 10 oocysts caused infection in 4 out of 4 ducks, with oocysts detected (by mouse bioassay) in muscle from all 4 ducks. Although domestic ducks are more susceptible to *T. gondii* infection than other birds, they show no clinical signs of infection.

Kajerova *et al.* (2003) obtained oral dose-response data for *T. gondii* K21 strain in budgerigars looking at oral doses ranging from 10^2 to 10^6. Similarly to ducks, the infected birds showed no signs of disease. The authors concluded that the ID_{50} in budgerigars is <100 oocysts, as all 37 birds fed 10^3 or more oocysts became infected, whilst 6 of 9 birds fed 100 oocysts became infected.

Use of the oral infection data for sheep and cattle by Esteban-Redondo and Innes (1998) and Esteban-Redondo (1999) was considered for this risk assessment. The problem is in deciding which tissue type to use for the definition of infection. In sheep, *T. gondii* DNA was detected in the peripheral blood mononucleocytes (PBM cells) in 1 of 4 sheep at an oral dose of 10^3, in 0 of 4 sheep at a 10^4 dose and in all sheep at a 10^5 dose (Esteban-Redondo and Innes 1998). The most susceptible tissue in sheep appeared to be cardiac muscle with a 10^3 dose resulting in DNA detection in 2 of 4 sheep, but only 1 of 4 sheep with a 10^4 dose, and 3 of 4 sheep with 10^5 dose. *T. gondii* DNA was only detected in skeletal muscle in 1 of 4 sheep at the highest dose of 10^5 oocysts. The results of Esteban-Redondo and Innes (1998) suggest that for sheep, at least, the ID_{50} (for detection of DNA in sheep tissues) is not less than 10^3 oocysts. However, assuming an ID_{50} of 10^3 oocysts would be less precautionary than using the abortion dose-response curve for which the LD_{50} is 800 oocysts (Figure 26).

8.8.1.2 Assessment of which dose-response data are appropriate for risk assessment for T. gondii

Via the oral route, *T. gondii* oocysts are highly infectious to ducks with an ID_{50} of < 10 oocysts as determined by mice bioassay of tissues in the challenged ducks (Bartova *et al.* 2004). The mice bioassay may be more sensitive than the DNA detection methods used by Esteban-Redondo and Innes (1998). Therefore, it may not be the case that the oral ID_{50} for *T. gondii* oocysts is $>10^3$ in sheep (from the data of Esteban-Redondo and Innes, 1998), because mouse bioassay may have yielded more positives if used instead of DNA detection. In this respect the LD_{50} (for still birth in pregnant ewes) of 800 oocysts used in the original compost risk assessment (Figure 26) is considered more precautionary for use here, in that the measurement is dependent on an observed health response and is not affected by the sensitivity of a detection method.

There is also the question of which response should be modelled. Choices of response include the risk of detection of oocysts in a given tissue type (i.e. infection), or the risk of abortion in ewes (as in the original risk assessment). Choosing infection as a response is complicated by the finding that detection of oocysts in different tissues (e.g. blood cells, brain, skeletal muscle, cardiac muscle) in sheep is not consistent for a given dose, suggesting that different tissues vary in risk of infection. For each tissue type, the risk appears to increase with higher challenge doses (Esteban-Redondo and Innes, 1998). Furthermore, there are conflicting results; thus while Esteban-Redondo and Innes (1998) report that brain and heart are the favoured tissue for detection, they cite another study which reported that oocysts were more frequently detected in muscle.

It is concluded that, at present, there are no dose-response data for *T. gondii* that offer a clear improvement on the dose-response approach used in the original compost risk assessment.

8.8.1.3 Realistic estimate of T. gondii oocysts in meat

Most publications do not provide data on the counts of *T. gondii* oocysts per unit weight of meat, and instead report the detection, i.e. presence or absence of oocysts. Thus, Esteban-Redondo and Innes (1998) report the presence of oocysts in different sheep tissues as detected by DNA methods. Similarly Bartova *et al.* (2004) used mouse bioassay to assess whether the duck tissue was positive. A recent study by Aspinall *et al.* (2002) has reported the prevalence of *T. gondii* in commercial meat samples obtained from UK retail outlets. In that study, a DNA methodology was again used to confirm the presence or absence. In total, 27 meat samples out of 71 obtained from UK retail outlets were positive for *T. gondii*. It is not feasible to estimate *T. gondii* oocyst counts in meat for the purpose of a quantitative risk assessment, and it is suggested that the approach used in the original risk assessment of estimating the *T. gondii* loading in compost from cat litter in Municipal Solid Waste (MSW) is still appropriate.

8.8.1.4 Assessing the risks of Toxoplasma gondii from compost

The national annual incidence of abortion in sheep flocks is 2% to 3% (Mearns 2007). In 1981, a study in Scotland detected *T. gondii* antibodies in 15.2% of sheep, 9.2% of pigs and 28% of stray cats (McColm *et al.* 1981). More recently, during the 2005 lambing season in Scotland, *T. gondii* passed *Chlamydophila abortus* to become the most commonly diagnosed cause of infectious abortion in sheep (SAC Veterinary Services, 2005). Serum antibody to *T. gondii* was detected in 47% of cats on sheep farms in the Bristol area of GB (Gethings *et al.* 1987). A study of 200 house mice trapped in 27 rodent-infested properties in Manchester UK showed 59% were positive for *T. gondii* (Murphy *et al.* 2008).

Mechanisms of introduction onto farm premises include introduction of infective oocysts in cat faeces via straw, hay, or concentrate feed. Also, invertebrates such as flies, cockroaches and earthworms can spread oocysts mechanically. Mechanisms of spread from infected premises include the movement of cats which are shedding infective oocysts. The greatest oocyst output is from young, elderly or immunosuppressed cats. In rural areas male cats may have territories of 60-80 hectares (150-200 acres). Females usually occupy 1/10 of this area. Other mechanisms of spread include movement of products containing oocysts in cat faeces, e.g., straw, hay, bedding muck, or contaminated concentrate feed, to another farm. Mearns (2007) has suggested steps to reduce the risks of transmission on farms including neutering of farm cats to reduce their roaming, prevention of access of feed to cats, and not feeding pregnant ewes the top bales of hay from store (in case of contamination with cat faeces).

On the basis that *T. gondii* is endemic, widespread and common in the UK (G. David, *pers. comm.*), and that infected cats roam around sheep farms, it is proposed that the additional risks through application of catering waste compost are low compared to other routes. This is not to say that compost contaminated with cat litter from MSW could not initiate infection in sheep at new sites.

The original compost risk assessment modelled the risk of pregnant ewes giving rise to a stillbirth after grazing on compost-amended soil for a year. This may be a more appropriate outcome to model than that of the risk of sheep becoming infected, as *T. gondii* is endemic in GB. The risk assessment is refined below using the new parameters presented in Table 7-1.

8.8.1.5 Recalculation of the quantitative risks from T. gondii in compost

The risks from *T. gondii* are calculated for the UK as opposed to GB. This is because data for cat numbers are for the UK.

Source Term

In the original risk assessment, Gale (2002) estimated the risks from the presence of cat litter containing *T. gondii*-infected cat faeces. The risk assessment assumed 25% of UK homes had cats (7.5 million cats in total), with 20% of those homes using cat litter. It also assumed that 10% of Municipal Solid Waste (MSW) (and hence cat litter) would go to composting with catering waste. The total number of oocysts entering composting from homes in the UK was estimated at 5.4×10^{12} per year in the original risk assessment.

A better estimate of the number of cats is obtained from the Cats' Protection website (Cats' Protection 2008) which states that the UK cat population is 9.2 million. This is slightly more than the 7.5 million estimated by Gale (2002).

Gale (2002) estimated that 10% of UK cat litter contaminated the catering waste for composting. This was the same as assuming that 10% of households using cat litter dispose of their cat litter in the catering waste bin all year instead of the residual waste bin. A 2010/11 study of food waste in 1,000 bags from collection schemes in the UK identified 1 nappy bag with dog faeces (Valorgas 2012). No cat litter was found. Thus, rather than assuming 10% of cat litter is composted a more realistic estimate may be closer to (or less than) 0.1%. As a worst case it is assumed that 1% of cat litter in the UK is incorrectly disposed of in food waste.

As in the original risk assessment, it was assumed that 20% of these cats used cat litter all year (although there are no data for this) and that 1% of the cat population is shedding oocysts at any one time (Buxton 1998). It was assumed that cats performed a single defecation per day, and that a single defecation contains as many as 10^7 oocysts (Buxton 1998). Over a period of 365 days therefore, the total loading to compost is 10^7 oocysts/day x 365 days x 9.2 million cats x 0.01 (shedding) x 0.2 (cat litter) x 0.01 (MSW contamination) = 6.7×10^{11} oocysts.

Predicted T. gondii concentrations in compost.

A 4.61-log reduction and dilution into 4,020,000 tonnes dry weight of compost for the UK gives a compost concentration of 4.1 oocysts per tonne.

Risk of abortion in pregnant ewes

The 4,020,000 tonnes of compost will cover 201,000 ha of land (in the UK), of which 117,005ha (58.2%) is assumed to be grassland for grazing. Assuming a sheep density of 5.1 /ha, some 596,728 sheep would be grazing on compost amended soil. Assuming sheep eat 0.69 kg soil/compost/day (Section 3.2.1) and that the *T. gondii* oocysts decay at a rate of 1-log in 84 days (0.0119 log per day), then the individual risks are set out in Table 8-39. These are calculated assuming the risk from a single oocyst is 0.00085 (Figure 26).

Table 8-39 Individual risks* for abortion in pregnant ewes (risk of abortion per pregnant sheep per year) predicted for *Toxoplasma gondii*

Length of grazing ban	Surface application (no dilution)	Minimum tillage (10 cm)	Plough depth (25 cm)
No ban	8.9×10^{-5}	1.2×10^{-6}	4.7×10^{-7}
3 week	5.0×10^{-5}	6.7×10^{-7}	2.67×10^{-7}
2 month	1.67×10^{-5}	2.23×10^{-7}	8.9×10^{-8}

*Note predicted individual risks apply to a pregnant sheep spending all day, every day for 1 year on land to which compost has been applied once at 20 tds/ha

Table 8-40 Average number of abortions in pregnant ewes predicted from T. gondii in UK from the application of compost to soil - calculated assuming pregnant ewe density of 5.1 / ha

Length of grazing ban	Surface application (no dilution)	Minimum tillage (10 cm)	Plough depth (25 cm)
No ban	53	0.714	0.28
3 week	29.8	0.4	0.16
2 month	10.0	0.13	0.053

Assuming a three week grazing ban and that the compost is tilled in to a depth of 10 cm, the individual risk to pregnant sheep is 6.7×10^{-7} per sheep per year. If all 596,728 sheep (in the UK) on land to which compost had been applied were pregnant all year then the expected number of abortions due to *T. gondii* would be about 0.4 per year (Table 8-40). However, as all sheep are not pregnant all of the time, the actual number would be less than this.

In 2004, 336 incidents of abortion were recorded in GB in sheep and goats due to *T. gondii* (G. David, APHA *pers. comm.*). The majority of cases are in sheep, as there are far fewer goats in the UK than sheep (Kate Whitaker, APHA *pers. comm.*). In 2007 there were 203 cases of toxoplasmosis in sheep in England and Wales with 13 in goats, plus 172 cases in Scotland in 2007 (Kate Whitaker, APHA *pers. comm.*) which is 375 cases in GB in total.

Therefore, the presence of *T. gondii* from contamination of catering waste with 1% of UK cat litter (compost tilled into 10 cm depth with 3 week grazing ban) would contribute 0.4 abortions in sheep per year in the UK, which is approximately 0.1% of the underlying number of abortions from *T. gondii* infection in sheep in GB alone. For surface applied compost with no grazing ban, the individual risk of 8.9×10^{-5} per sheep per year translates into 53 still births per year in the UK. This is worst-case in assuming all sheep on land with compost are not only pregnant all year, but also ingest compost to the exclusion of soil for a year.

It should be noted that although 375 cases of *T. gondii* abortion were confirmed in 2007 in sheep in GB, many incidences would go unreported. In the majority of sheep flocks there will almost always be a low level of abortion, with 3% to 4% losses being accepted without investigation (Kate Whitaker, APHA *pers. comm.*).

9.0 Sensitivity analysis for the quantitative risk assessments

In this section, a sensitivity analysis is undertaken to illustrate "What if?" scenarios and to assess the impact of uncertainty in the parameters in the model. These parameters include changes in the compost application rate, amount of soil ingested, degree of inactivation of pathogens in the "hot" part of composting, by-pass of the processes, quantities of illegally imported meat and decay rate on land.

Grazing ban lengths and depth of incorporation are not considered here because they are controllable parameters and are not associated with uncertainty. Moreover quantitiative estimates of risk are presented for different depths and grazing bans in the specific section for each pathogen.

9.1 Baseline QRA results

The baseline risks predicted for each pathogen are summarised in Table 9-1. These risks are presented for comparison with those predicted through changing the values of the other parameters in the sensitivity analyses conducted in the rest of this section. Grazing ban length and depth of incorporation are given baseline values of 3 weeks and 10 cm depth, respectively.

Table 9-1: Summary of baseline risks (3 week grazing ban and 10 cm depth of incorporation)

	Individual risk (per animal per year)	Number of years between infections in GB
FMDV	0.5×10^{-12} (for cattle[b])	6,058,000 (for sheep[b])
CSFV	0.88×10^{-9}	3,579
ASFV	3.4×10^{-13}	9,254,000
SVDV	4.9×10^{-17}	6.4×10^{10}
NDV*	0.5×10^{-6}	ND
[a] *Toxoplasma gondii*	0.67×10^{-6}	2.5
BSE	1.1×10^{-8}	511
Classical scrapie	1.8×10^{-6}	5.1
Atypical scrapie	2.2×10^{-7}	7.8

*No grazing ban and surface applied
ND, not determined (see Section 8.5.6)
[a]UK
[b]Highest risk out of the three livestock species considered

9.2 Sensitivity analysis – doubling the application rate

The results of a sensitivity analysis in which the application rate is doubled from 20 tonnes per ha to 40 tonnes per ha of dry matter is set out in Table 9-2. Doubling the application rate is interesting from a risk assessment model perspective in that it doubles the individual risk, providing there has been incorporation of the compost into the soil. If there has been no incorporation into the soil, then the model already assumes each animal is eating neat compost every day for one year. Therefore, the application rate is immaterial and has no effect on individual risk. Moreover, the predicted number of infections in GB from surface-applied compost halves because only half the animals are now exposed to compost-amended soil because at 40 tonnes/ha, 3,913,200 tonnes only covers 56,948 ha (instead of 113,897 ha). In the baseline model, however, compost has been diluted 75-fold by soil after tilling to a depth of 0.1 m. Doubling the application rate simply increases the net pathogen loading in that soil by a factor of two so that grazing livestock receive double the exposure and hence double the risk (per individual animal).

However, the total number of infections predicted is unchanged by varying the application rate (assuming it's tilled in). This is due to the fact that doubling the application rate, means the compost only covers half the original area and therefore only half the number of livestock are exposed. In fact only 56,948 ha of grazing grassland now receive compost. Assuming the livestock density is unchanged, then only half the number of livestock animals are exposed to compost-amended soil, albeit at double the individual risk. In effect, the application rate cancels out in the calculation of the number of livestock infected.

Table 9-2: Risks predicted assuming application rate has doubled from 40 tonnes/ha to 20 tonnes/ha (3 week grazing ban and 10 cm depth of incorporation)

	Individual risk (per animal per year)	Number of years between infections in GB	Summary of change relative to baseline
FMDV	0.9×10^{-12} (for cattle)	6,058,000 (for sheep)	Individual risk doubles, but total number of infections in GB unaffected because only half the number of livestock exposed
CSFV	1.7×10^{-9}	3,579	
ASFV	6.8×10^{-13}	9,300,000	
SVDV	9.8×10^{-17}	6.4×10^{10}	
[a] *Toxoplasma gondii*	1.3×10^{-6}	2.5	
BSE	2.2×10^{-8}	511	
Classical scrapie	3.6×10^{-6}	5.1	
Atypical scrapie	4.4×10^{-7}	7.8	
NDV*	0.5×10^{-6}	ND	Individual risk not affected because chickens are eating neat compost for a whole year (no dilution in soil) and so application rate is immaterial

*No grazing ban and surface applied
ND, not determined (see Section 8.5.6)
[a]UK

9.3 Sensitivity analysis – 100% of compost is applied to grassland used for livestock grazing

The results of a sensitivity analysis in which 100% of the compost is applied to grassland used for livestock grazing are shown in Table 9-3. Since application rates, soil dilution and decay factors are unaffected the individual risks are the same as baseline. However, 1.7-fold more livestock are exposed because instead of applying compost to 113,897 ha, compost is now applied to 195,660 ha. Thus the number of infections predicted increase by 1.7-fold.

Table 9-3: Risks predicted assuming 100% of land (i.e. 195,660 ha) treated with compost is used for grazing rather than just 58.2% (3 week grazing ban and 10 cm depth of incorporation)

	Individual risk (per animal per year)	Number of years between infections in GB	Summary of change relative to baseline
FMDV	4.6×10^{-13} (for cattle)	3,526,000 (for sheep)	Individual risk is unaffected but number of infections in GB increases proportionately by 1.7-fold
CSFV	8.6×10^{-10}	2,083	
ASFV	3.3×10^{-13}	5,387,000	
SVDV	4.9×10^{-17}	3.7×10^{10}	
BSE	1.1×10^{-8}	298	
Classical scrapie	1.8×10^{-6}	2.98	
Atypical scrapie	2.2×10^{-7}	4.6	
NDV*	0.5×10^{-6}	ND	Individual risk not affected; grazing land not relevant to poultry
[a] *Toxoplasma gondii*	6.7×10^{-7}	1.5	Individual risk is unaffected but number of infections in UK increases proportionately by 1.7-fold

*No grazing ban and surface applied
ND, not determined (see Section 8.5.6)
[a]UK (201,000 ha in UK)

9.4 Sensitivity analysis – reducing the amount of soil ingested by sheep

In a recent risk assessment for scrapie in fertilisers, Adkin and Kosmider (2011) used lower soil ingestion rates for sheep. The baseline risk assessment for compost presented here assumes sheep eat 0.69 kg soil per day. The arithmetic mean consumption of soil by sheep used in the fertiliser risk assessment (Adkin and Kosmider 2011) was just 0.0937 kg soil per day. In Table 9-4 risks are recalculated for the four pathogens which infect sheep assuming that sheep ingest 0.0937 kg soil per animal per day. 0.69 kg is 7.36–fold greater than 0.0937 kg and consequently, all risks decrease by 7.36–fold (Table 9-4) in this scenario.

Table 9-4: Risks predicted assuming sheep ingest 0.0937 kg per animal per day instead of 0.69 per animal per day (3 week grazing ban and 10 cm depth of incorporation)

	Individual risk (per animal per year)	Number of years between infections in GB	Summary of change relative to baseline
FMDV	3.9×10^{-14}	44,610,000	Risks decrease by 7.36-fold
[a] *Toxoplasma gondii*	9.1×10^{-8}	19	
Classical scrapie	2.4×10^{-7}	37.7	
Atypical scrapie	2.9×10^{-8}	58	

[a]UK

Using this more realistic value for soil consumption by sheep, the predicted number of classical and atypical scrapie infections through compost tilled into 10 cm depth in the soil was 0.027 and 0.017 per year, respectively. For surface applied compost this gave 2.0 and 1.3 classical and atypical scrapie infections per year.

9.5 Sensitivity analysis – by-pass of the composting processes

In the base-line case it is assumed that each compost process removes 2.7-logs of pathogens (Figure 10). This is based on the assumption that there is just 0.2% by-pass of each composting step. This is separate to the assumption that 10% of the catering waste is processed through the less intensive 'meat free' composting route (see Figure 28). For the purpose of the sensitivity analysis it is assumed that each composting process only removes 1.7-log of pathgens, i.e. there is 2% by-pass instead of 0.2%. This is shown in Figure 27 with 1% intrinsic by-pass and 1% extrinsic by-pass (which combined give 2% by-pass).

The overall inactivation of pathogens with 2% by-pass of each composting step is 3.25 log (Figure 28). This assumes that the hot-part of composting achieves a 6-log inactivation of the pathogens. The baseline removal is 4.61-log. Thus, the loss in removal by composting is 1.36-log and the risks would be expected to increase by $10^{1.36}$ or 23-fold relative to baseline, which is what is observed in the results (Table 9-5).

Figure 27: 1% instrinsic by-pass and 1% extrinsic by-pass give a 1.7-log removal

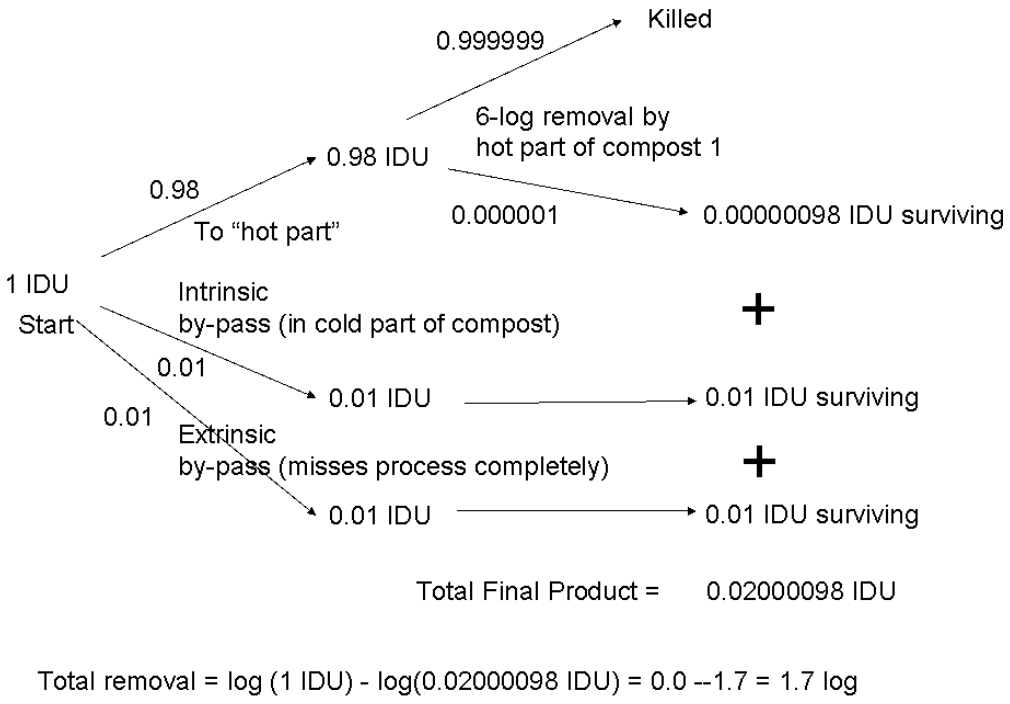
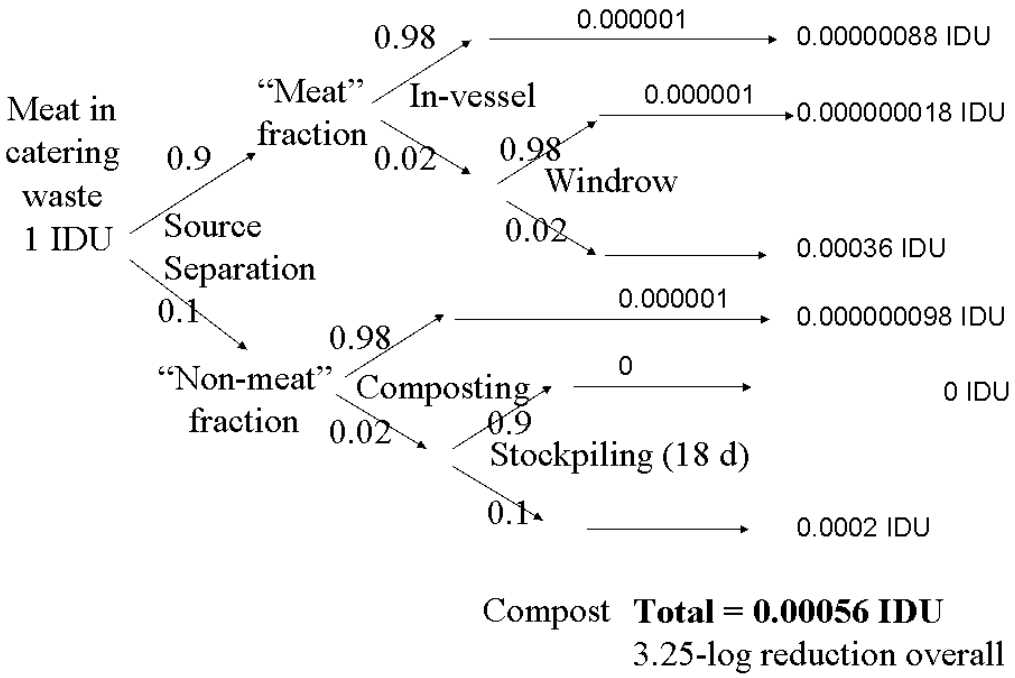


Figure 28: The overall reduction is 3.25-log with 2% by-pass of each compost step. This assumes composting in the hot part removes 6-log of pathogens



In the case of scrapie, the baseline risk assessment assumes that composting removes just 80% of the scrapie infectivity. Therefore according to the baseline model 20% of the scrapie survives composting. Allowing for a 2% by-pass would increase this to 22%. The net removal is therefore 4.54-fold which is a 0.657-log removal. This is used in the sensitivity analysis in Table 9-5.

Table 9-5: Risks predicted assuming 2% by-pass of each compost step instead of 0.2% by-pass, i.e. composting removes 3.25-log overall of pathogens except for scrapie for which removal is 0.657-log (3 week grazing ban and 10 cm depth of incorporation)

	Individual risk (per animal per year)	Number of years between infections in GB	Summary of change relative to baseline
FMDV	1.1×10^{-11} (for cattle)	264,000 (for sheep)	Risks increase by 23-fold
CSFV	2.0×10^{-8}	156	
ASFV	7.8×10^{-12}	404,000	
SVDV	1.1×10^{-15}	2.8×10^{9}	
NDV*	1.2×10^{-5}	ND	
[a] *Toxoplasma gondii*	1.5×10^{-5}	0.11	
BSE	Assumed that composting has no effect on BSE agent in the model		
Classical scrapie	1.9×10^{-6}	4.74	Very small increase in risk
Atypical scrapie	2.4×10^{-7}	7.3	

*No grazing ban and surface applied
ND, not determined (see Section 8.5.6)
[a]UK

The risks of BSE are not affected, while those for scrapie increase marginally. Of the four exotic livestock viruses SVDV, ASFV, CSFV and FMDV, the highest risk was predicted for CSFV with one infection every 156 years (Table 9-5).

The sensitivity analysis predicts one case of CSFV in GB every 179 years with a 2 month grazing for surface applied compost produced by a process that only removes 3.25-log of virus due to 2% by-pass. The predicted risk increases to one CSFV case every 2 years for surface-applied compost with a 3 week grazing ban. For pigs, a 2 month grazing ban is enforced (Animal By-Products Regulations 2011).

For *T. gondii*, nine abortions in pregnant ewes were predicted per year with 2% by-pass of composting, a 3 week grazing ban and tilling in to 10 cm depth. This is equivalent of one abortion every 0.11 years (Table 9-5).

9.6 Sensitivity analysis – inactivation efficiency in the "hot" part of composting

As discussed in Section 4.3, 6-log inactivations of the exotic viruses FMDV and CSFV at 56°C for 4 hours have not been demonstrated experimentally. The highest reported removal at 55°C is about 4-log for CSFV. This, however, reflects the starting titres and length of the experiments (5 mins). With higher starting titres or longer experiment times, removals of >4.2-log may have been demonstrated. It is assumed in the baseline that 56°C for 4 hours gives 6-log inactivation. Indeed most of the material is at 60°C for at least 2 x 48 hours in the composting step. The 56°C at 4 hours reflects the centre of a particle of diameter 40 cm (Section 4.0). In the case of FMDV, >5-log inactivations have been reported in aqueous buffer within 37 minutes. However, there is some evidence that FMDV is inactivated less rapidly by heat in meat compared to aqueous media and slurries. For this reason, a sensitivity analysis is performed for FMDV with an inactivation rate of 1.92-logs in the "hot" part (Section 9.6.2). This is based on the inactivation at 55°C of 1.92-log albiet after only 10 min (Table 4-1). The sensitivity analyses presented below are undertaken with 4-log inactivation of CSFV in the "hot" part and 1.92-log inactivation for FMDV.

9.6.1 CSFV – 4-log inactivation

With 6-log reduction in the hot-part, the net removal by composting is 4.61-log (Figure 14) with 0.2% by-pass of each composting process. Reducing the inactivation in the hot part to just 4-log gives a net log reduction of 3.91-log (Figure 29). This is a 0.7-log or 5-fold increase in CSFV survival of the composting process and is reflected in a 5-fold increase in the predicted risk relative to base-line (Table 9-6).

Figure 29: Modelling only 4-log reduction of CSFV in the "hot" part of composting. The overall reduction is 3.91-log with 0.2% by-pass of each compost step.

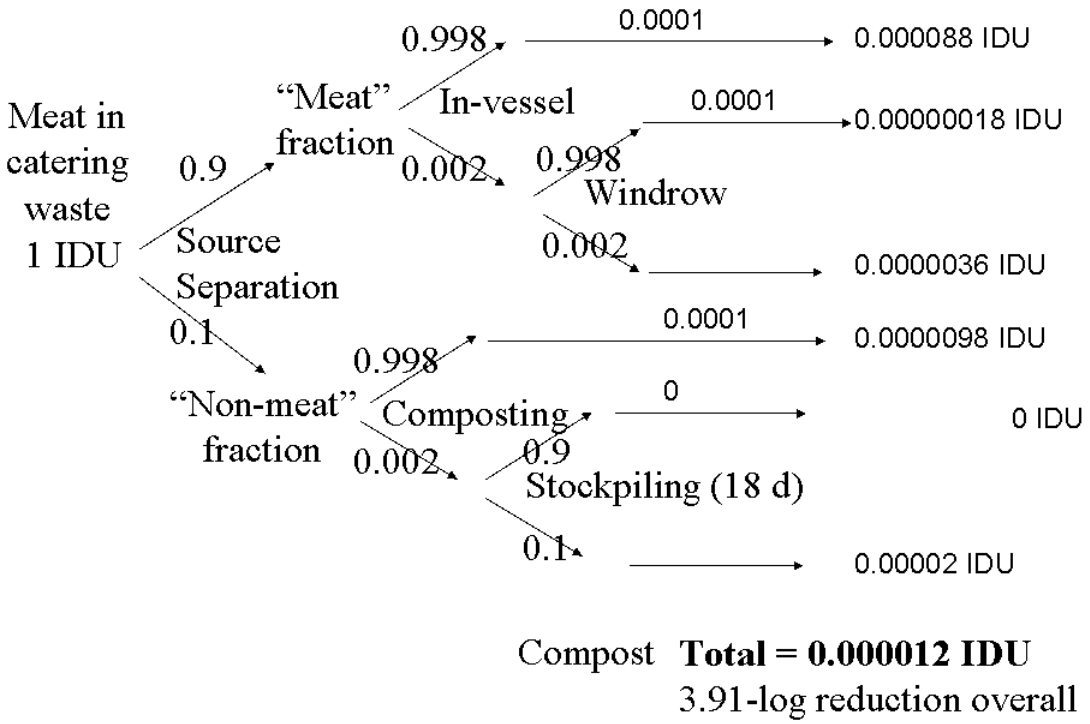
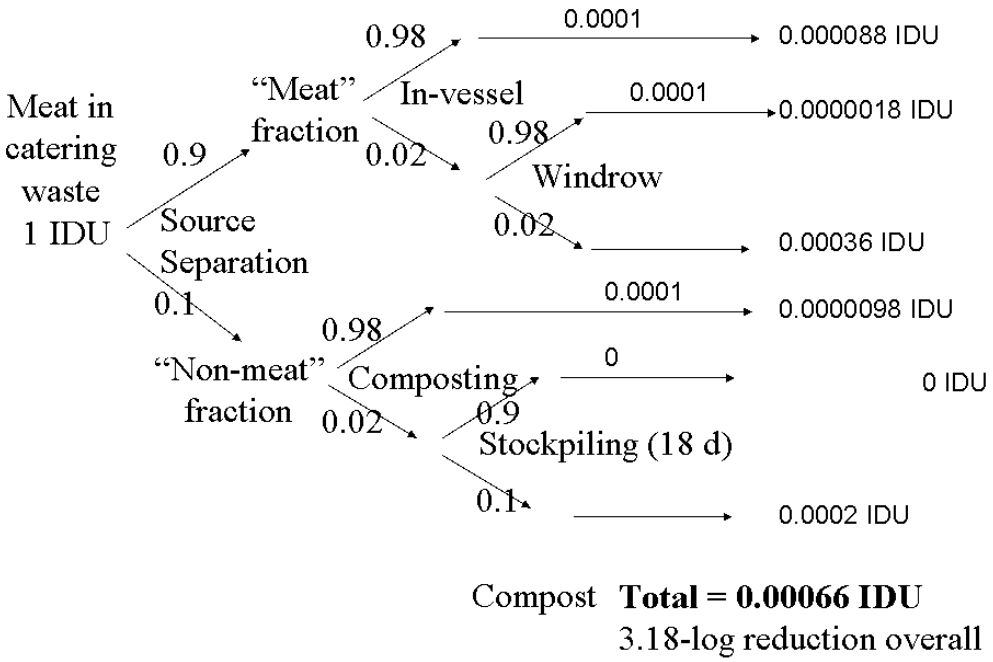


Table 9-6: Risks predicted assuming 4-log inactivation of CSFV in the "hot" part of composting with 0.2% by-pass of each compost step, i.e. composting removes 3.91-log overall (3 week grazing ban and 10 cm depth of incorporation)

	Individual risk (per animal per year)	Number of years between infections in GB
CSFV	4.4×10^{-9}	714

This raises the question of what happens if the by-pass is 2% instead of 0.2%. As shown in Figure 30 with 4-log removal in the hot part and 2% by-pass the net removal is 3.18-log. This is very similar in magnitude to the 3.25-log removal used in Section 9.5 and the sensitivity analysis is therefore not illustrated.

Figure 30: Modelling only 4-log reduction of CSFV in the "hot" part of composting. The overall reduction is 3.18-log with 2% by-pass of each compost step.



9.6.2 FMDV – 1.92-log inactivation

Reducing the inactivation in the hot part of the compost to 1.92-log of FMDV with 0.2% by-pass gives a net removal of 1.92-log (Figure 31). The 0.2% by-pass makes little contribution. This removal is 2.69-log lower than the baseline of 4.61-log and the risks of FMDV increase by about 500-fold (Table 9-7). However, the risks of FMDV are still remote even with just 1.92-log inactivation by the composting process. With 2% by-pass of each compost step the net log removal is 1.90-log (not shown) which is very similar to the 1.92-log.

Figure 31: Modelling only 1.92-log reduction of FMDV in the "hot" part of composting. The overall reduction is 3.91-log with 0.2% by-pass of each compost step.

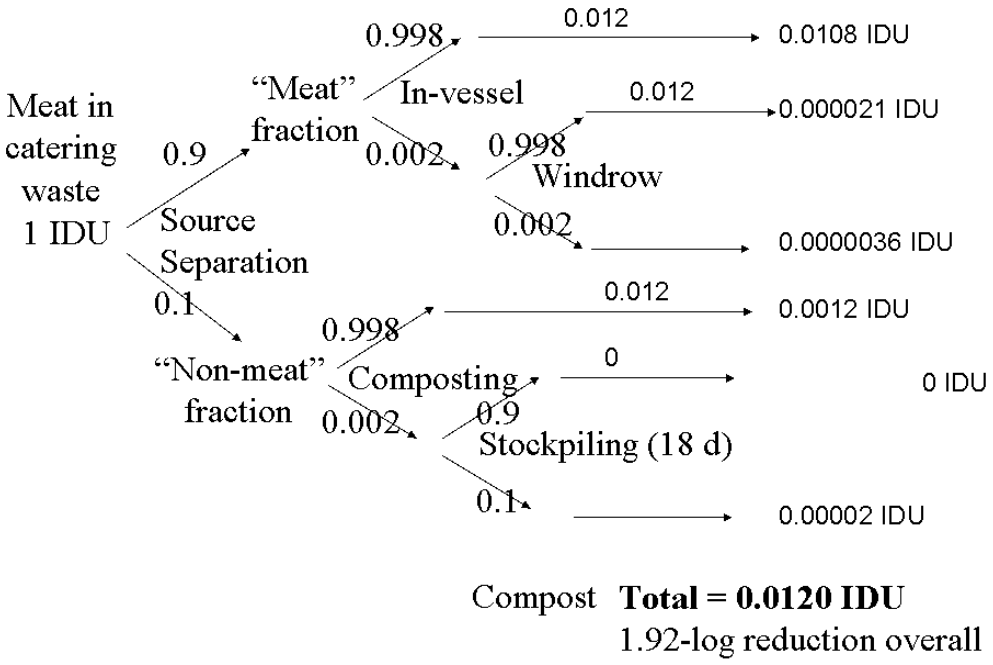


Table 9-7: Risks predicted assuming 1.92-log inactivation of FMDV in the "hot" part of composting with 0.2% by-pass of each compost step, i.e. composting removes 1.90-log overall (3 week grazing ban and 10 cm depth of incorporation)

	Individual risk (per animal per year)	Number of years between infections in GB
FMDV	(Cattle) 2.3×10^{-10}	(Sheep) 12,368

9.7 Sensitivity analysis – quantities of illegally imported meat entering GB

The amounts of illegally imported meat infected with exotic viruses were taken from Hartnett *et al.* (2004). Those estimates were made based on worldwide prevalence of exotic viruses in 2003. Since then ASFV has emerged in areas of Russia and Georgia, and the amounts of infected meat potentially entering the UK could have increased. For the purpose of the sensitivity analysis it is assumed that amounts of illegally imported, infected meat are 10-fold higher than those used in the baseline as estimated by Hartnett *et al.* (2004). Using such an assumption results in the estimated risks increasing by 10-fold (Table 9-8).

Table 9-8: Risks predicted assuming amount of illegally-imported infected meat increases by 10-fold (3 week grazing ban and 10 cm depth of incorporation)

	Individual risk (per animal per year)	Number of years between infections in GB	Summary of change relative to baseline
FMDV	4.6×10^{-12} (for cattle)	606,000 (for sheep)	As expected, the risks increase by 10-fold
CSFV	0.88×10^{-8}	357	
ASFV	3.4×10^{-12}	925,000	
SVDV	4.9×10^{-16}	6.4×10^{9}	

9.8 Sensitivity analysis – amount of meat discarded to composting

Exotic viruses in raw pig meat

The baseline risk assessments assume 1.39% of pig meat is discarded raw to compost, based on published data on the characteristics of food waste (Table 2-3). For the sensitivity analysis for FMDV, SVDV, CSFV and ASFV, it is assumed that 5% of pig meat is discarded raw.

Scrapie in raw and processed lamb

It is assumed that cooking has no effect on scrapie and therefore that all estimated 27,606 tonnes of lamb (including mincemeat and meat balls) reported as discarded in the WRAP (2008a) study (see Table 2-2) should be included in the estimation of the proportion of total lamb discarded (raw or processed) with regard to scrapie. Overall, 27,606 tonnes accounts for 5.88% of the 469,000 tonnes of lamb purchased annually in GB (Table 2-3). This value of 5.88% is therefore used in the baseline risk assessment for scrapie infectivity in all tissues except the spinal cord. However, sandwich spread, unidentified meat/offal, unidentified bones and cured meats add an extra 34,000 tonnes to the total of all discarded meats (Table 2-1). For the sensitivity analysis it is assumed that this extra 34,000 tonnes is all lamb and that in total 34,000 + 27,606 tonnes of lamb (raw or processed) is therefore discarded for composting. This combined figure of 61,606 tonnes accounts for 13.1% of the total lamb supplied. For the sensitivity analysis therefore 13.1% of that scrapie infectivity entering the food chain in all tissues except spinal cord is now assumed to go to composting. It should be noted that the baseline risk assessments for both classical and atypical scrapie already assume 100% of infectivity in any spinal cord present in meat in the food chain is composted. Similarly the sensitivity analysis assumes 100% of spinal cord is composted. Since the ratios of infectivity in spinal cord to other tissues differ in classical and atypical

scrapie (Table 8-31), the risks change by different proportions in Table 9-9 as the percentage of lamb discarded is changed.

BSE in raw and processed beef
As for scrapie it is assumed that cooking and processing have no effect, and therefore all 45,234 tonnes of beef discarded is included in the estimation of the proportion of BSE infectivity in the food chain that is discarded. In the baseline case this is 45,234 tonnes of 1,052,000 tonnes which is 4.3%. For the sensitivity analysis, the additional 34,000 tonnes from sandwich spread, unidentified meat/offal, unidentified bones and cured meats (Table 2-1) is included. The total raw and processed beef is therefore 45,234 + 34,000 = 79,234 tonnes, which is 7.53% of the total beef purchased (1,052,000 tonnes). For the sensitivity analysis therefore 7.53% of the BSE infectivity entering the food chain is now assumed to go to composting.

NDV in raw poultry meat
The baseline risk assessments assume 2.82% of poultry meat is discarded raw to compost (Table 2-3). For the sensitivity analysis for NDV, it is assumed that 5% of poultry meat is discarded raw.

T. gondii in cat litter
The baseline assessment assumed 1% of cat litter went to compost by mistake. This was a worst-case assumption and it is not considered appropriate to double this value to provide an unrealistic case.

Even with the changes described above, the results of these scenarios on increasing the amount of meat going to composting cause little concern (Table 9-9).

Table 9-9: Risks predicted assuming 5% of pig meat (FMDV, CSFV, SVDV, ASFV) and poultry meat (NDV) is discarded raw for composting and 13.1% of lamb (scrapie) and 7.53% of beef (BSE) are discarded (raw or processed) (3 week grazing ban and 10 cm depth of incorporation)

	Individual risk (per animal per year)	Number of years between infections in GB	Summary of change relative to baseline
FMDV	1.7×10^{-12} (for cattle)	1,684,000 (for sheep)	Risks increase by 3.6-fold
CSFV	3.1×10^{-9}	995	
ASFV	1.2×10^{-12}	2,573,000	
SVDV	1.8×10^{-16}	1.8×10^{10}	
NDV*	0.9×10^{-6}	ND	Risks increase by 1.8-fold
BSE	1.9×10^{-8}	292	Increase in risk is less than a factor of 2.
Classical scrapie	3.85×10^{-6}	2.38	2.15-fold increase in risk, but note increase is not linear because 100% of spinal cord is assumed in both baseline and sensitivity analyses
Atypical scrapie	2.3×10^{-7}	7.4	

*No grazing ban and surface applied
ND, not determined (see Section 8.5.6)

9.9 Sensitivity analysis – decay rates in the soil

In the baseline model, decay rates for ASFV, CSFV and FMDV were based on data for decay in slurry at 4°C (Haas *et al.* 1995). It is acknowledged that slurry may present a more aggressive environment than soil. The decay rate for *Toxoplasma gondii* oocysts in soil was based on that for *Cryptosporidium* (Olson *et al.*1999). Consequently a sensitivity analysis has been conducted to assess the effect on the overall risks should pathogens decay more slowly in the soil. In the absence of data on pathogen decay in compost-amended soils hypothetical decay rates have been used that are a half or a quarter of the decay rates used in the baseline models. This allows a relative comparison of reduced decay rates between the different QRAs. This sensitivity analysis is not undertaken for BSE, scrapie or SVDV as the baseline models assume these pathogens do not decay in the soil.

The risks from decay rates at 0.5 and 0.25 the baseline rates are shown in Table 9-10 and Table 9-11 respectively. Note that, as before, each model was adjusted to allow for up to 5-log decay in the soil after which no further decay occurred. Thus, the longer times for a 5-log inactivation were 183 (366) days, 206 (413) days, 338 (677) days with 0.5x (0.25x) baseline decays for CSFV, FMDV and ASFV, respectively.

Halving the soil decay rates increases the risks of CSFV to one infection every 787 years (Table 9-10). The predicted risk increases to one case every 207 years on quartering the soil decay rate (Table 9-11). The predicted risks for NDV and *T. gondii* are less sensitive to the soil decay rate (Table 9-10 and Table 9-11). The risks from FMDV and ASFV are still negligible.

Table 9-10: Risks predicted assuming soil decay rates are 0.5 those of baseline rates (3 week grazing ban and 10 cm depth of incorporation)

	Individual risk (per animal per year)	Number of years between infections in GB	Summary of change relative to baseline
FMDV	0.29×10^{-11} (for cattle)	967,180 (for sheep)	6-fold increase in risk
CSFV	6.3×10^{-9}	495	7-fold increase in risk
ASFV	1.36×10^{-12}	2,304,000	4-fold increase in risk
NDV*	1.0×10^{-6}	ND	Little effect because slow decay rate for NDV in soil
[a] *Toxoplasma gondii*	1.75×10^{-6}	1.0	Little effect because slow decay rate

*No grazing ban and surface applied
ND, not determined (see Section 8.5.6)
[a]UK

Table 9-11: Risks predicted assuming soil decay rates are 0.25 those of baseline rates (3 week grazing ban and 10 cm depth of incorporation)

	Individual risk (per animal per year)	Number of years between infections in GB	Summary of change relative to baseline
FMDV	1.0×10^{-11} (for cattle)	273,000 (for sheep)	22-fold increase in risk
CSFV	2.4×10^{-8}	130	27-fold increase in risk
ASFV	3.9×10^{-12}	815,000	11-fold increase in risk
NDV*	1.88×10^{-6}	ND	Little effect because slow decay rate for NDV in soil
[a] *Toxoplasma gondii*	3.73×10^{-6}	0.45	Little effect because slow decay rate

*No grazing ban and surface applied
ND, not determined (see Section 8.5.6)
[a]UK

9.10 Sensitivity analysis – effect of composting on scrapie inactivation

According to Miles *et al.* (2011) mesophilic anaerobic digestion at 37°C removed 2.43-logs of scrapie agent after 15 days as determined by a method that detected scrapie infectivity. Moreover under thermophilic conditions (60°C) a 3.41-log reduction was observed at 10 days. The baseline risk assessment assumes that compost inactivates 80% of the scrapie agent. This gives a 5-fold or 0.69-log reduction in scrapie activity. As discussed in Section 8.7.4 this is a worst case assumption. In practice, higher levels of inactivation may occur not only due to temperature but also due to proteolytic degradation during the composting process. The uncertainty analysis here therefore considers the impact of a 2.43-log (269-fold) reduction as observed by Miles *et al.* (2011) for PrP^{Sc} under mesophilic anaerobic digestion at 37°C for 15 days. It is likely that production of compost of sufficient stability to comply with the PAS100 specification would require composting durations of more than 15 days in total, including a controlled period of 60°C for 4 days. Thus, in practice, the overall inactivation of scrapie may be greater than 2.43-log. However, although thermophilic anaerobic digestion gives 3.41-log inactivation (Miles et al. 2011), the net removal by composting would be limited to 2.7-log by the 0.2% by-pass (Figure 10). The predicted risks and numbers of years between infections in sheep are presented in Table 9-12, and, as expected, the risks show a 55-fold decrease relative to baseline. Overall, the sensitivity analysis predicts just 0.004 classical scrapie and 0.002 atypical scrapie (0.006 classical and atypical total) infections per year if 2.43-log is used for removal by composting. Thus, composting could be a major barrier for scrapie.

Table 9-12: Risks predicted assuming a 2.43-log (269-fold) inactivation of scrapie by composting instead of the baseline 0.69 log (five-fold) inactivation (3 week grazing ban and 10 cm depth of incorporation)

	Individual risk (per animal per year)	Number of years between infections in GB	Summary of change relative to baseline
Classical scrapie	3.3×10^{-8}	281	55-fold decrease in risks
Atypical scrapie	4.0×10^{-9}	431	

9.11 Sensitivity of modelling to the stocking density

The livestock densities used in the baseline model are 2.8 pigs/ha, 1.59 cattle/ha and 5.1 sheep/ha (Table 3-14). Here it is assumed that those stocking densities double. The results of the sensitivity analysis are shown in Table 9-13. The individual risks are unaffected. In contrast the number of infections per year in GB double, and the time between infections halves compared to the baseline.

Table 9-13: Risks predicted assuming livestock densities are doubled (3 week grazing ban and 10 cm depth of incorporation)

	Individual risk (per animal per year)	Number of years between infections in GB	Summary of change relative to baseline
FMDV	4.6×10^{-13} (for cattle)	3,029,000 (for sheep)	Individual risk is unaffected but number of infections in UK increases proportionately
CSFV	8.8×10^{-10}	1790	
ASFV	3.4×10^{-13}	4,627,000	
SVDV	4.9×10^{-17}	3.2×10^{10}	
BSE	1.1×10^{-8}	256	
Classical scrapie	1.8×10^{-6}	2.6	
Atypical scrapie	2.2×10^{-7}	3.9	
NDV*	0.5×10^{-6}	ND	Individual risk not affected; grazing land not relevant to poultry
[a] *Toxoplasma gondii*	6.7×10^{-7}	1.3	Individual risk is unaffected but number of infections in UK increases proportionately

*No grazing ban and surface applied
ND, not determined (see Section 8.5.6)
[a]UK

10.0 Endemic faecal bacterial pathogens – Salmonellas, *E. coli* O157 and Campylobacters – possible risks to livestock

10.1 Effects of composting on genetic properties of bacteria

10.1.1 Antibiotic resistance plasmids

Guan *et al.* (2007) determined if mobile plasmids carrying antibiotic resistant genes could survive and be transferred in chicken manure during composting. At 23ºC plasmids were transferred from donor *E. coli* to recipient *E. coli* in chicken manure and in compost mesocosms that comprised mixes of chicken manure and peat. At 50ºC, neither the plasmids not their *E. coli* hosts could be detected. The authors concluded that composting of chicken manure at high temperatures could prevent spread of antibiotic-resistant genes via plasmids in the environment.

10.1.2 Loss of virulence of E. coli O157 during storage

Duriez *et al.* (2008) evaluated changes in the population structure and stability of virulence genes in *E. coli* during manure storage on a commercial farm that housed healthy pigs. After seven weeks a genotype that did not carry any virulence genes dominated the surviving population. Other experiments indicated a significant reduction in the carriage of virulence genes by *E. coli* during manure storage. This is important as it suggests that storage of *E. coli* O157 in manures and perhaps in composts too may result in a loss of virulence such that the pathogens pose less of a public health risk.

10.2 Regrowth of E. coli, salmonellas and other Enterobacteriaceae in compost and cowpats, and in compost-amended soil environments

Christensen *et al.* (2002) observed regrowth of *E. coli* and total coliforms during the lower-temperature stabilization phase following the high-temperature stage of composting, while Wichuk and McCartney (2007) have more recently reviewed the issue of regrowth of bacterial pathogens in compost. They note that regrowth is a potential problem even in composts that have been properly treated to reduce pathogenic bacteria to very low levels.

10.2.1 Regrowth during the composting process itself

Shuval *et al.* (1991) observed *Salmonella* sp. regrowth in the cool exterior of windrows. Turner (2002) observed that *E. coli* concentrations actually increased from their initial levels when composting was carried out at temperatures of <45ºC. Wichuk and McCartney (2007) conclude that low-temperature zones within compost are a concern not only because pathogens may not be sufficiently reduced, but also because conditions may be favourable for bacterial pathogen regrowth. It is worth noting that, under national legislation, there is a requirement for compost operators to demonstrate that the whole composting mass achieves 60°C for 48 hours in each enclosed composting stage.

10.2.2 Regrowth of Salmonella in the stored composts

Salmonella have been shown to survive some composting processes in low numbers (Ross and Yanko, 1981) and then grow in soil amendments and stored biosolids under certain conditions. It has been observed that *Salmonella* is often present in composted biosolids below detectable limits, and later on regrows when the product is marketed (Skanavis and Yanko, 1994). Russ and Yanko (1981) observed regrowth of salmonellas from undetectable levels even after the compost had been stored in a desiccated state for approximately one year.

Russ and Yanko (1981) reported that repopulation of *Salmonella* was affected by moisture, temperature and nutrient content of compost. Indigenous microbial competition is also known to influence *Salmonella* regrowth (see Sidhu *et al.*, 1999). The regrowth of pathogens, being affected by a number of different inherited and environmental factors, is difficult to predict. Sidhu *et al.* (1999) suggest an approach for testing the regrowth potential by seeding compost with *Salmonella*. In composted biosolids which had been

sterilized by autoclaving twice at 121ºC (20 min), Sidhu *et al.* (1999) demonstrated that seeded *Salmonella* and *E. coli* grew rapidly, reaching population densities of more than 10^8 g^{-1} after 30 hour incubation (Figure 32). In a further paper, Sidhu *et al.* (2001) note that growth of seeded *S.* Typhimurium was suppressed in non-sterilised compost with a maximum *S.* Typhimurium density of < 10^3 g^{-1}. There was a significant decline in the growth rate of seeded *Salmonella* in sterilized compost when the compost was stored, suggesting that bio-available nutrients declined with storage. However, in non-sterilized compost this was not the case (Sidhu *et al.,* 2001). This suggests that the indigenous microflora play a significant role in suppression of *Salmonella* regrowth in composted biosolids. There was a strong negative correlation (−0.85) between the *Salmonella* inactivation rate and the maturity of compost in non-sterilized compost (Sidhu *et al.,* 2001). The *Salmonella* inactivation rate was seven times higher in biosolids composting for two weeks as compared to compost stored for two years. This suggests that the antagonistic effect of indigenous microorganisms towards *Salmonella* declined with compost storage. Sidhu *et al.* (2001) concluded that all composted biosolids had a *Salmonella* regrowth potential. However, the indigenous microflora significantly reduced this potential. According to Sidhu *et al.* (2001), long-term storage of compost is not recommended as this may increase the pathogen regrowth potential.

Although Sidhu *et al.* (1999) demonstrated an approximate 8-log increase in *Salmonella* counts in sterile compost, this would not be appropriate for modelling of operational composts, which are not sterile. As noted by Sidhu *et al* (2001), the indigenous microflora constitute the most important factor in suppressing *Salmonella* regrowth. While salmonellas reached $10^8/10^9$ g^{-1} in sterile compost, they did not exceed 10^3 g^{-1} in non-sterilised compost. It is also worth noting that their absence has to be demonstrated in composts derived from catering wastes under both national and EU legislation. Samples submitted for such testing must be taken from storage.

Figure 32 Growth curves for seeded bacteria in sterile composted biosolids (taken from Sidhu et al. 1999)

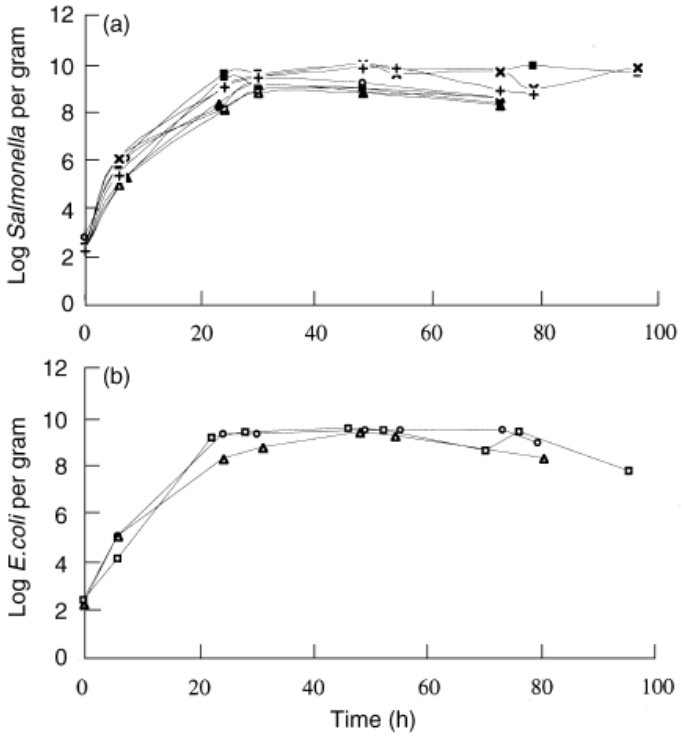


Fig. 2 Growth curves for seeded bacteria in sterile composted biosolids (a) ○, *Salm.* Anatum (8414); Δ, *Salm.* Chester (8605); □, *Salm.* Mbandaka (9098); ●, *Salm.* Infantis (8527); ◆, *Salm.* Havana (7685); ■, *Salm.* Typhimurium (9451R); ×, *Salm.* Typhimurium (9451); +, *Salm.* Typhimurium (Mu-S5–83); (b) ○, *E. coli* (NCTC 8196); Δ, *E. coli* (Mu-E9–83); □, *E. coli* (ATCC 25922).R = Rifampicin resistant strain

10.2.3 Post-treatment contamination, handling and conditions may affect regrowth

Zaleski *et al.* (2005) noted that following rainfall events, significant increases in numbers were observed for both faecal coliforms and salmonellas in digested biosolids which had been subject to desiccation in drying beds. They concluded that moisture promoted the growth of faecal coliforms and salmonellas from external sources.

Duffy *et al.* (2002) expressed concern with potential regrowth of pathogens when composts are handled inappropriately. Processing of composts to produce watery extracts called compost teas is gaining in popularity among organic growers and home gardeners. Preparation methods often call for the addition of carbon sources such as molasses to stimulate a rapid increase in microbial populations. Duffy *et al.* (2002) monitored the risk potential for regrowth of *E. coli* O157:H7 and *Salmonella* Thompson in teas made from various types of compost. Amendments greatly increased regrowth of both bacteria in teas derived from dairy compost, yard waste compost and chicken manure compost (approximately 2.5 log increase with 0.5% molasses in most cases). However, regrowth potential depended on the pathogen and also on the type of compost used to prepare teas. For example, with 0.5% molasses, *Salmonella* increased from 1 to over 1000 cfu/ml in teas made from dairy or yard compost but exceeded 350,000 in chicken manure compost tea, whereas *E. coli* regrowth was the same in all types of compost teas (Duffy *et al.* 2002).

Chesshire and Ferry (2006) reported on the possibility that stored pasteurised digestate could become subject to recontamination through microbial regrowth. A series of digested and pasteurised slurries was stored for two week and four week periods using both open and closed storage systems, and then retested. The results are set out in Table 10-1.

Table 10-1: Pasteurised digestate storage: Faecal indicator organism recontamination trial

Sample	Total Coliforms per g	*E. coli* per g	Enterococci (CFU in 100ml)
Trial 1 Baseline Results	*<10*	*<10*	*<100*
14 days open storage	6	<1	71
28 days open storage	<1	<1	<1
14 days closed storage	<1	<1	26
28 day closed storage	<1	<1	<1
Trial 2 Baseline Results	*<1*	*<1*	*150*
14 days closed storage	<1	<1	<1
14 days closed storage	2	<1	240
Trial 3 Baseline Results	*10*	*10*	*100*
14 days open storage	<1	<1	70
28 days open storage	<1	<1	290
14 days closed storage	<1	<1	60
28 day closed storage	<1	<1	1000
Trial 4 Baseline Results	*<1*	*<1*	*400*
14 days closed storage	4	<1	190
14 days closed storage	<1	<1	560

The results show that there is little recontamination during post-treatment storage, with many samples recorded as below the level of detection. Any regrowth that occurred tended to be limited to *Enterococci*. This is greater in closed containers and may be due to the fact that the closed storage conditions were favourable for *Enterococci* growth. In both the farms during Trial 2 closed storage was disrupted due to the tank lid blowing off. This may have led to some recontamination and the slightly increased level of Enterococci. The closed sample has a higher level of faecal indicator organisms. The rest of the samples seemed to show a decrease in faecal indicator loadings with increased duration of storage.

10.2.4 Regrowth of faecal coliforms and E. coli in cowpats and bovine faeces

Van Kessel *et al.* (2007) studied survival of faecal coliforms and *E. coli* in cattle faecal samples deposited as shaded and non-shaded cowpats in field, and also under laboratory conditions. They reported that both faecal coliforms and *E. coli* concentrations increased by as much as 1.5-log in cowpats in pasture and in laboratory conditions during the first week. After that, concentrations declined. The die-off was much slower in shaded cowpats, and was faster in the field than in the laboratory. Similarly, Sinton *et al.* (2007) reported up to 1.5-log increases in *E. coli*, faecal streptococci and enterococci in the first one to three weeks in bovine faeces on pasture. Thus *E. coli* counts increased from 3.0×10^6 to 4.8×10^7 / g. Thereafter, the counts decreased. *Salmonella enterica* counts doubled, and there was no increase recorded in *Campylobacter jejuni* counts, which decayed.

10.2.5 Conclusions

There is evidence that *E. coli* and salmonellas, and other *Enterobacteriaceae* may grow in compost, particularly sterilised composts. This may be less relevant for the composts considered in this risk assessment, which after maturation should not be sterile. Growth is suppressed by indigenous bacteria. There is evidence that *E. coli* and total coliform bacteria grow in cattle faeces applied to soils. Therefore growth could occur in manures after application to soils, although a search of the literature has not so far found any papers which address this.

10.3 Overview of the risks of E. coli O157, salmonellas and campylobacters to livestock from application of compost to soil

Dose-response data for faecal bacterial pathogens in livestock are not available (with the exception of a study on *Salmonella* in pigs (Loynachan and Harris 2005)). Full quantitative risk assessments cannot therefore be undertaken. The approach taken here is to compare the total loadings and concentrations of *E. coli* O157 predicted in stored manure and conventionally-treated sewage sludge with those predicted in composted catering waste in England and Wales (Table 10-3). The total loadings are of more relevance because the group risk to a country as a whole is proportional to the total pathogen loading contributed to the soil environment through each route, rather than the actual concentration. The derivation of these data is presented below.

10.3.1 Estimation of total E. coli O157 loadings in compost from meat in the UK

The source of *E. coli* O157 in compost is assumed to be through faecal contamination on meat, namely beef and lamb. A survey on the prevalence VTEC O157 in minced beef available at retail in the Republic of Ireland detected 43 positive samples out of a total of 1533 i.e. 2.8% of samples were positive (Cagney *et al.,* 2004). In 21 of the positive samples the number of VTEC O157 ranged from 0.52 - 4.03 log CFU g^{-1}, whereas the remaining 22 positive samples were enumerated at <0.52 log CFU g^{-1}. In the absence of other data it is assumed that these values are representative of the contamination of meat across the UK. These data were used to fit a normal distribution of the log counts with mean -5.5 and standard deviation 2.8. This distribution gives similar results to the data given above, the mean count from the resulting log normal distribution was 44 VTEC O157 g^{-1} of minced beef, which is the value that is used here to estimate the VTEC O157 loading in meat going to compost**.**

It was estimated that a total of 8,382 tonnes of beef and 5,115 tonnes of mutton and lamb were discarded raw in the UK each year (Table 2-3). Assuming a worse-case scenario of all this meat being composted and that the average *E. coli* O157 count is 44 CFU g^{-1}, it is estimated that the total VTEC O157 loading in meat to compost is 5.94 x 10^{11} CFU per year. Using a worst-case assumption for the purposes of modelling, it is assumed that *E. coli* O157 can achieve a 4-log regrowth in the meat once discarded and before processing. This is based on results by Berry and Koohmaraie (2001). The growth kinetics depend both on the temperature and the levels of beef microflora. At 4°C no growth occurred even after 14 days. At 12°C under aerobic conditions 4-log growth occurred between 7 and 10 days. Thus if meat is treated within seven days of removal from a refrigerator, then only 2-3 log regrowth would occur. Furthermore, with medium to high microflora present, which would be case, only 3-log growth is achieved at 10 days. Thus 4-log regrowth is worst-case and unlikely if medium to high levels of beef microflora are present and the meat is in the bin awaiting composting for less than 7 days. Assuming 4-log regrowth, therefore, the final VTEC O157 loading to compost in meat after regrowth is 5.94 x 10^{15} CFU per year. A 4.61-log inactivation by composting leaves 1.46 x 10^{11} *E. coli* O157 in compost in GB (Table 10-3).

10.3.2 Estimation of total E. coli O157 loadings in conventionally treated sewage sludge in England and Wales

Gale (2005) estimated that the inputs of *E. coli* O157 at abattoirs from slaughter of sheep and cattle in England and Wales is 7.35 x 10^{14} cfu per year. Of this 5% went to the sewer where 82.9% partitioned into raw sewage sludge. Thus 3.05 x 10^{13} cfu of *E. coli* O157 go to sludge each year. The sewage sludge is then treated by mesophilic anaerobic digestion (MAD). According to Horan *et al.* (2004), primary sludge digestion removes 1.66-log of *E. coli*. This is 45.7-fold inactivation, leaving a fraction of 0.022 surviving. Adding in a further 0.01 (i.e. 1%) from by-pass gives 0.032 as the total fraction of *E. coli* surviving. The net removal is therefore 1.49 log. This is in good agreement with the 1.5-log removal for *E. coli* by primary digestion obtained in an 18 month survey of all digestion sites in northwest

England (cited in Horan *et al.* 2004). Furthermore, Smith *et al.* (2005) conclude that a 2-log reduction of *E. coli* is likely to challenge most MAD digesters unless strict maintenance and management practices are adopted to minimize dead zones and by-pass. The 1.49-log reduction used here would therefore seem to be realistic. The total *E. coli* O157 loading in MAD-treated sewage sludge in England and Wales is therefore 9.8 x 10^{11} cfu per year (Table 10-3).

10.3.3 Estimation of total E. coli O157 loadings in treated manure in England and Wales

Some 77% of manure is of bovine origin and likely to contain *E. coli* O157. In England and Wales, some 52,300,000 tonnes of manure are applied to land (ADAS *pers. comm.*). The level of zoonotic agents in British livestock manures have been derived in a national study conducted between April 2000 and December 2002 (Hutchison *et al.*, 2004). Livestock wastes were collected both as fresh (from the pen or yard) and stored (waste that had been taken to secondary storage). The results of this study indicated that VTEC O157 is present in 13.2%, 11.9% and 20.8% of fresh cattle, pig and sheep manures respectively. The arithmetic mean VTEC O157counts in CFU g^{-1} are given in Table 10-2.

Table 10-2: Level of *E. coli* O157 (CFU g^{-1}) in positive livestock manure (Hutchison *et al.*, 2004)

	Cattle (n=810)	Pig (n=126)	Sheep (n=24)
Arithmetic mean	2.9 x 10^6	6.9 x 10^4	1.1 x 10^4
Maximum	2.6 x 10^8	7.5 x 10^5	4.9 x 10^4
Number positive	107	15	5

Assuming 13.2% of livestock manure contains the arithmetic mean of 2.9 x 10^6 cfu, the total *E. coli* O157 loading in England and Wales is 2.0 x 10^{19} cfu per year. Storage on the farm will give a 1-log reduction every 20 days (ADAS data). Thus, assuming a 90 day storage period (prior to land-spreading) there will be a decay of 4.5-logs. The total *E. coli* O157 loading in England and Wales on land through stored manure is 6.33 x 10^{14} cfu per year (Table 10-3).

10.3.4 Comparison of the loadings in sewage sludge, manure and composted catering waste

Allowing for 13,498 of raw lamb and beef (Table 2-3), to be composted, the predicted *E. coli* O157 loading is 4,460-fold lower that for stored manure. On this basis, it is concluded that the risks from *E. coli* O157:H7, salmonellas and campylobacters through application of compost made from catering waste containing meat are much lower than for those from stored manures.

The fact that the loading in compost is only 5-fold lower than for MAD-treated sewage sludge is of little concern. Firstly the 4-log regrowth of each and every *E. coli* O157 bacterium in all of the meat is unrealistic. Secondly, sewage sludge is primarily human waste, and humans are not a major reservoir of *E. coli* O157.

The compost loadings in Table 10-3 did not allow for any regrowth of *E. coli* O157 in the compost (final product) although a 4-log growth in the meat prior to composting is included, which is worst-case. The indigenous microflora present in compost greatly suppresses regrowth of salmonellas compared to non-sterile compost (Sidhu *et al.* 2001). As summarised in Section 10.2.2, *Salmonella* did not exceed 10^3 in non-sterilised compost (Sidhu *et al.* 2001). Even if bacterial concentrations increased by 1,000-fold in compost, the predicted total *E. coli* O157 loadings would not exceed those predicted in stored manures. It should be noted that the risk is proportional to the total pathogen loading rather than the relative differences in loading. Thus although a 1,000-fold increase in the mean of 42,000 cfu/tonne for compost (Table 10-3) may give greater (per tonne) loadings than the 12,000,000 cfu/tonne for manures, the total numbers are still lower, and hence the overall

risks are lower – because the quantity of colonised material is lower. Furthermore regrowth of *E. coli* O157 can occur in cowpats as well as composts. It is concluded that, even allowing for regrowth in compost, the risks from faecal bacteria such as salmonellas and *E. coli* O157 in composted catering waste do not exceed those of stored manures. Currently manure is used on 78% of farms (Section 2.4.2). Unlike *E. coli* O157, Campylobacters do not grow in the environment as they require unusual conditions for growth (Corry and Atabay 2001).

Table 10-3: Comparison of predicted *E. coli* O157 loadings (cfu per year) in total "stored" manure, conventionally-treated sewage sludge in England/Wales and composted catering waste (UK). Predicted *E. coli* O157 concentrations also estimated

	Stored manure	Sewage sludge (Gale 2005)	Composted catering waste[1]
Arithmetic mean concentration E. coli O157 (cfu per year/total tonne per year)	1.2 x 107 per tonne (wet)	1.0 x 106 per tonne (dry)	4.2 x 104 per tonne (dry)
Total tonnes produced per year	52,300,000 (wet)	967,000[3] (dry)	3,466,394[2] (dry)
Total predicted loading *E. coli* O157 (cfu per year)	6.33×10^{14}	9.8×10^{11}	1.46×10^{11}

[1] *E. coli* O157 loadings based on total UK beef and lamb being composted (Table 2-3) and 4-log regrowth.
[2] Estimated tonnes dry weight of catering waste compost produced by England and Wales according to data from Table 4 (WRAP 2008)
[3] Tonnes dry solids of sewage sludge produced in England and Wales (Gale 2005)

11.0 Clostridium botulinum (Botulism)

Clostridium botulinum is a Gram positive, spore-forming bacterium that produces neurotoxins. It is an obligate anaerobe. Seven types of neurotoxin (A – G) have been identified. *C. botulinum* occurs in soil and sediments with a heterogeneous worldwide distribution. The spores are very resistant to heat and a range of chemicals and can survive for many years in soil (Smith, 1988; ACMSF 2006).

11.1 Impact of composting on bacterial spores

Briancesco *et al.* (2008) note that *Clostridium perfringens* spores, because of their high resistance to treatments, could be considered for assessing the composting process with regard to more resistant pathogen reduction.

As noted by Wichuk and McCartney (2007) the spores of *Bacillus* and *Clostridium* can survive for extended periods under a variety of environmental conditions. *B. cereus*, for example, was detected after composting for 7 days at temperatures below 70ºC, but could not be detected if temperatures above 70ºC persisted for a period of 2 to 3 days. Clostridial spores can survive at temperatures of 100ºC for 2 h. Wichuk and McCartney (2007) conclude that spores of *Clostridium botulinum* and *C. perfringens* can survive composting conditions that inactivate other bacteria, and that it is unlikely that bacterial endospores would be removed from compost via thermal destruction with the regulated time-temperature criteria.

This suggests the assumption in the original risk assessment that composting would not remove *C. botulinum* spores was appropriate.

11.2 Confirmation of the presence of C. botulinum spores in compost

In a study in Germany, Böhnel and Lube (2000) found that compost produced from household waste contained *C. botulinum* in up to 50% of the samples. Unfortunately, quantitative data on the number of spores and their viability were not determined.

11.3 Behaviour of Clostridium botulinum in soil

It should be noted that *C. botulinum* is commonly found in the soil, and its spores have been found on raw potatoes, for example (Vugia *et al.* 2009). Gessler and Böhnel (2006) make two points regarding *C. botulinum* in compost-amended soil. First, compost is high in organic matter, which might influence the indigenous microbial community favouring the survival or destruction of *C. botulinum* scores. Second the spores might be spread on soil where a *C. botulinum* population was not present, subjecting them to population dynamics that are likely to be different to those of the compost, which could affect survival and growth.

Their sensitivity to molecular oxygen generally restricts *Clostridium* spp. to anaerobic environments such as water, submerged soil, rumina and intestines. Clostridia have also been isolated from non-submerged soil and litter, indicating their occupation of anoxic microsites in soil particles and litter.

Grass silage undergoes fermentation, which is an anaerobic process. Notermans *et al.* (1981) investigated botulism outbreaks in cattle on farms in the Netherlands. They noted that cycles of disease occurred through feeding of grass silages. The silages were made from grass harvested from pastures contaminated with faeces from botulinum-infected cattle. Notermans *et al.* (1981) demonstrated that in such grass silages, the number of *C. botulinum* organisms increased. In two edge samples of silage, *C. botulinum* was present in samples of just 0.0001 g weight, suggesting concentrations of 10,000 g^{-1}. This is considerably greater than the levels measured in faeces or soil and suggests growth in the silage. Notermans *et al.* (1981) suggested that growth of *C. botulinum* in the grass silage is

determined by pH and water activity. Water activity is the most important factor and increases at the edges of the silo, allowing outgrowth of *C. botulinum*.

Notermans *et al.* (1981) also give data on spore levels in faeces of cattle. When silage not contaminated with *C. botulinum* was fed to cattle, or when cattle were not fed silage in summertime, *C. botulinum* organisms were present in the cattle faeces only in low numbers (<1 /g). The average number of *C. botulinum* organisms present in the faeces during the housing period of the winter of 1978/79 was about 100/g and 600/g in 1979/80. Numbers of *C. botulinum* organisms present in soil samples obtained from the pastures were as high as 130/g of soil (Notermans *et al.* 1981).

11.3.1 Regrowth of Clostridium botulinum in soil

Gessler and Böhnel (2006) provide experimental evidence of persistence and translocation of *C. botulinum* spores in compost-amended soil samples. In one experiment compost spiked with 10^3 spores/g compost was applied to the soil (Gessler and Böhnel 2006). A total of 30 kg of the compost (i.e. 3 x 10^7 spores) was applied to $10m^2$ of soil and raked into a depth of <5 cm. It is calculated here assuming a soil density of 1.5 g/cm^3 and that the compost is only diluted to 1 cm depth, that the average spore concentration would be 200 spores/g soil. It is also calculated here that the average spore concentration would be just 40 spores/g soil if tilled into 5 cm depth. Indeed spore counts between 0 and 200 spores/g were observed for the first 200 days (Figure 33). However, at days 260 and 315 much higher counts were observed. By 700 days, counts were down to 20 spores/g. Gessler and Böhnel (2006) attributed the variation to spatial heterogeneity. Spatial heterogeneity is due to clumping or clustering of spores such that some areas have very high counts while large areas have none. This is supported by the fact that zero counts are observed at days 50, 180, and 370 (Figure 33). Gessler and Böhnel (2006) do not rule out regrowth of the spores from their data but do not attempt a statistical proof. Indeed the average count over the 939 days is 1,550 spores/g, which is 8 - 40-fold higher than expected. However, evidence against regrowth of spore counts is that marked increases are not observed at lower soil levels (10 – 30 cm depth) where conditions would be expected to be less aerobic (Figure 34) and therefore more suitable for *C. botulinum* growth.

Figure 33: *C. botulinum* counts in the top layer (0- 5 cm) from soil plots with compost spiked with 10^3 *C. botulinum* spores g^{-1}. Figure 3 from Gessler and Böhnel (2006).

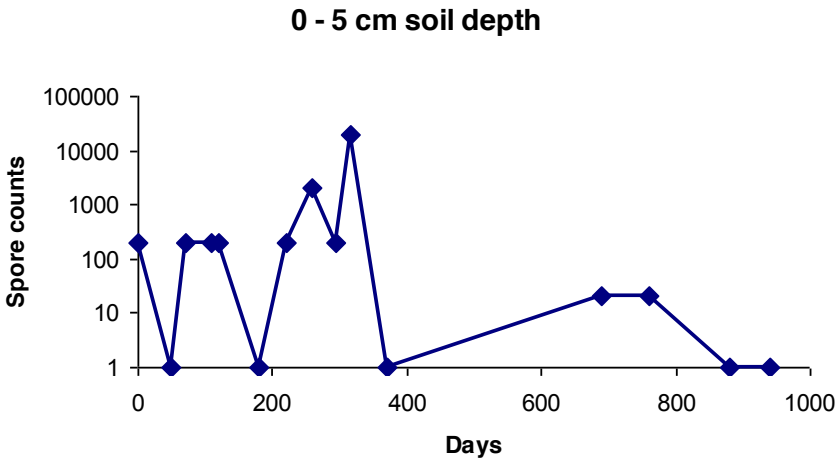
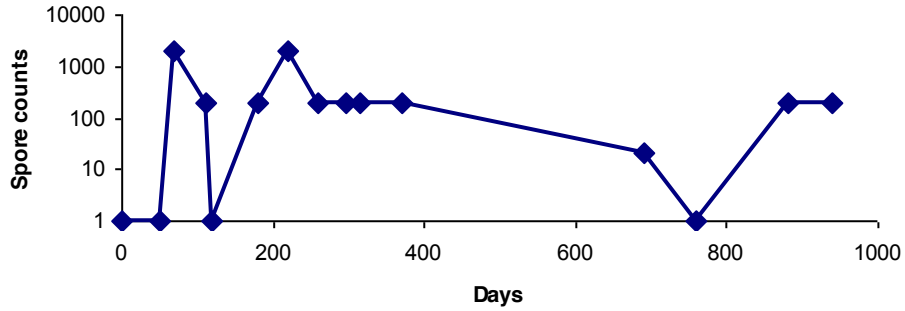


Figure 34: *C. botulinum* counts in the top layer (10 - 30 cm) from soil plots with compost spiked with 10^5 *C. botulinum* spores g^{-1}. Figure 4 from Gessler and Böhnel (2006).



For soils spiked with compost containing 10^5 spores/ g compost, the results from the top 0 cm to 5 cm are more difficult to interpret in terms of regrowth because several points exceeded the maximum detectable by the most probable number methodology used (see Fig 4 of Gessler and Böhnel, 2006). It is calculated here that the average soil density (assuming the compost was tilled to just 1 cm depth) would be 20,000 spores/g soil. Spore concentrations of up to 20,000 /g soil were detected in the upper level (0 cm to 5 cm depth), while spore counts appear to show some decline (Figure 33 and Figure 34) over the 939 day period particularly after 200-400 day in the soil. Indeed Gessler and Böhnel (2006) note that their results on the persistence are in accordance with those of other workers who "observed that *Clostridium sporogenes* declined only slightly (<0.7 log units) in the soil. This may be in part due to leaching to lower soil layers. Gessler and Böhnel (2006) show that *C. botulinum* is vertically translocated in soil, however, the results of Gessler and Böhnel (2006) are difficult to interpret in terms of growth or decline and their main conclusion is that *C. botulinum* spores can persist in soil.

11.3.2 Leaching of Clostridium botulinum to lower layers

Gessler and Böhnel (2006) investigated if *C. botulinum* introduced with compost into *C. botulinum*-free soil can persist and be translocated within the soil. At spike concentrations of 10^5 spores/g of compost, *C. botulinum* persisted for the whole 939 day period, with vertical translocation to 10 cm to 30 cm depth starting 70 days after the compost was spread.

11.4 Overview of risks to livestock of Clostridium botulinum through compost

The work here has focused on reviewing data published since 2002 on regrowth of *Clostridium botulinum* in soil and its impact on the risk to grazing livestock. *C. botulinum* spores are widespread in the environment in the UK, and are found in soils. Exposure of livestock to spores is assumed to be a common occurrence as soil is ubiquitously, if heterogeneously, contaminated with *C. botulinum* spores. Therefore, occurrence of spores of *C. botulinum* is not unique to composts and poses relatively little risk in the absence of anaerobic conditions. Importantly, healthy adult mammals do not usually develop disease following ingestion of *C. botulinum* spores because the spores appear to compete poorly with the normal gut flora and a toxaemic infection does not usually develop. Botulism in cattle is an intoxication and not an infection, and requires anaerobic conditions in the soil for multiplication of the bacteria such that sufficient toxin is produced to cause intoxication. The presence of large numbers of spores in ingested putrid material may also contribute, along with ingested toxins, to toxicoinfection – proliferation of the organism with toxin production within the intestine (ACMSF, 2006; Notermans, *et al.*, 1981; Notermans, *et al.*, 1979).

At the higher temperatures of ~70°C regularly achieved in composting, considerable inactivation of the botulinum toxin is likely. Thus Losikoff (1978) demonstrated a >5-log inactivation of type A toxin in ~4 h at 70°C at pH 6.8 in beef broth. Hubalek and Halouzka (1988) reported that the time required for a 2-log reduction of toxicity of type C toxin was less than 20 minutes at 50°C. The heat denaturation of botulinum neurotoxins was studied by Zhou *et al.* 2008 where the impact of different temperatures at 30 minutes exposure times on the toxicity and protein structure were monitored. The study concluded that heat treatments of at least 50°C resulted in significant (at least 3-log) decrease in toxin toxicity. Bradshaw *et al.* (1979) reported ~4-log inactivations of type A and type B toxins in 50 minutes at 67.8°C in beef and mushroom patties. In contrast at 74°C, Woodburn *et al.* (1979) reported times of an hour or more for 3-log inactivation, albeit in acid foods. The toxins are more stable in acidic environments (pH 5) than in neutral conditions (pH 6.8) (Losikoff 1978). Rasooly and Do (2010) report that type B toxin is not completely inactivated by conventional milk pasteurisation (63°C, 30 min) although type A toxin is inactivated.

Calculating the quantitative risks for *C. botulinum* toxicoinfection in cattle is not possible because of lack of available data to calculate toxin concentrations in compost, and also lack of information on the distribution of the toxin in the compost and soil. Such information would be needed to determine whether grazing cattle could ingest a threshold dose given dilution of the toxin in the compost.

The risk of botulinum intoxication to cattle through compost is considered low because composting, unlike silage production, is an aerobic process such that regrowth of any *C. botulinum* bacteria present in the meat will not occur during composting. Also, at temperatures of 56°C to 60°C, any bacteria germinating from spores would be inactivated. Furthermore, any botulinum toxin may be diluted during the composting and soil application processes, such that cattle exposures are below the threshold dose (although this has not been assessed here and would depend on the nature of any particles to which the toxin was attached). It is concluded that the risks to grazing livestock from *C. botulinum* in compost which has been tilled into a depth of 10 cm are low under normal aerobic soil conditions. However, it cannot be ruled out that spores present in compost could not multiply up in silages produced from grass. Indeed, data from botulism outbreaks in the Netherlands suggest growth of *C. botulinum* under the anaerobic conditions found in grass silage (prepared with wilted grass from pastures and covered with plastic sheets).

12.0 Brief examination of the risks to livestock associated with avian influenza virus (AIV)

Influenza-A viruses which infect poultry can be divided into two distinct groups on the basis of their ability to cause disease. The very virulent viruses cause a disease formerly known as fowl plague and now termed highly pathogenic avian influenza virus (HPAI) in which mortality may be as high as 100%. These viruses have been restricted to subtypes H5 and H7, although not all viruses of these subtypes cause HPAI. All other viruses cause a much milder disease (Alexander *et al.* 2005). Outbreaks of disease in chickens in Europe by H5 and H7 subtypes may follow migratory bird activity in an area (Shortridge 2005), although other routes of transmission including legal importation of poultry meat may present higher risks of release. Systematic surveillance studies into the presence of influenza viruses in avian species revealed enormous pools of influenza-A viruses in wild birds, especially migratory waterfowl (Alexander *et al.* 2005). Unlike mammals, where the number of subtypes appears to be limited, all 15 H and 9 N subtypes currently recognised have been recorded in birds. The H5N1 subtype of HPAI is of particular interest because it was previously a purely avian disease until its isolation from infected people in 1997, some of whom died.

12.1 Routes of transmission of AIV

There is evidence that consumption of HPAI virus-infected meat by birds (including poultry) is a route of infection. Indeed, oral uptake is an efficient way of transmitting HPAI virus among poultry and mammals (cited in Harder *et al.* 2009). Herring gulls have been shown to be infected through consumption of AIV-infected chicken meat (Brown *et al.* 2008), confirming that discarded raw poultry meat itself presents a potential route of infection to scavenging wild birds. Similarly, outbreaks of H5N1 have occurred in backyard poultry flocks in Germany through feeding of raw scraps of infected duck meat (Harder *et al.* 2009).

12.1.1 The role of environmental transmission in recurrent avian influenza epidemics

Environmental transmission of AIV may have been overlooked compared to other exposure routes. Breban *et al.* (2009) propose that, in addition to direct faecal/oral transmission, birds may become infected by ingesting avian influenza virions that have long persisted in the environment. According to Breban *et al.* (2009) very low levels of environmental transmission (i.e. a few cases per year) are sufficient for AI to persist in populations where it would otherwise disappear.

12.2 Source Term

12.2.1 Titres of H5N1 HPAI virus in infected ducks and chickens

The feathers, oro-pharyngeal secretions and faeces from AIV-infected birds contain high titres of virus (Yamamoto *et al.* 2008). There are numerous papers detailing titres of HPAI in poultry tissues including thigh meat and breast meat (Thomas and Swayne 2007; Gao *et al.* 1999). Thus, Thomas and Swayne (2007) report H5N1 HPAI virus titres of up to $10^{8.0}$ EID_{50} (embryo infectious dose 50%) per gram of uncooked thigh sample, and $10^{7.5}$ EID_{50} per gram of uncooked breast sample (Table 12-1).

Table 12-1: Virus titres in meat from chickens infected with H5N1 HPAI virus (data from Thomas and Swayne 2007)

Strain	Meat source	Titre (log EID_{50}/g)	Clinical status of infected bird
A/chicken/Korea/ES/03	Chicken thigh	8.0	Dead
A/chicken/Korea/ES/03	Chicken thigh	6.8	Dead
A/chicken/Korea/ES/03	Chicken breast	7.5	Dead
A/chicken/Korea/ES/03	Chicken breast	5.6	Dead
A/chicken/Korea/ES/03	Chicken breast	7.3	Dead
A/chicken/Anyang/AVL-1/01	Chicken breast	5.5	Dead or sick
A/chicken/Indonesia/05/05	Chicken breast	7.9	Dead

Gao *et al.* (1999) infected chickens (2-weeks-old) intranasally and orally with 10^5 plaque-forming units (pfu) of human strains of HPAI H5N1 virus and determined virus titres in various tissues at 1 day after infection. These are presented in Table 12-2.

Table 12-2: Systemic replication of human H5N1 viruses in chickens. Virus titres (log pfu/g) presented by Gao *et al.* (1999)

Virus	HK483	HK486
Lungs	6.5	5.3
Nasal turbinates	6.8	4.1
Spleen	5.2	5.1
Liver	5.1	5.3
Kidneys	4.9	4.2
Brain	3.6	3.7
Pancreas	4.8	3.8
Colon	5.1	4.8

The H5N1 virus titres in meat from chicken from Thomas and Swayne (2007) are set out in Table 12-3. Note the duck meat titres (Table 12-3) are lower than for chicken meat (Table 12-1). Pantin-Jackwood et al. (2007) measured HPAI H5N1 titres in 2 and 5 week old Peking ducks infected under laboratory conditions. Portions of brain, lung, skeletal muscle, heart and kidney were analysed together with oropharangeal and cloacal swabs collected from all ducks. The viral titres are summarised in Table 12-3.

Table 12-3: Virus titres in meat from domestic ducks infected with H5N1 HPAI virus (data from Thomas and Swayne 2007)

Strain	Meat source	Titre (log EID_{50}/g)	Clinical status of infected bird
A/duck/Anyang/AVL-1/01	Duck thigh	3.4	Clinically normal
A/Env/Hong-Kong/437-6/99	Duck thigh	2.0	Clinically normal
A/Vietnam/1203/04	Duck thigh	5.7	Sick
A/Prachinburi/6231/04	Duck thigh	4.0	Sick
A/crow/Thailand/(1C)/04	Duck thigh	5.6	Sick
A/egret/Hong Kong/757.2/02	Duck thigh	6.0	Sick
A/egret/Hong Kong/757.2/02	Duck thigh	2.8	Clinically normal

Table 12-4: H5N1 titres in tissues from infected ducks (data from Pantin-Jackwood *et al.* 2007)

Tissue	Titre EID_{50}/ml		
	2 week old	5 week old	
Brain	$10^{6.2}$	$10^{3.8}$	Maximum mean value recorded 2 dpi*
Heart	$10^{6.2}$	$10^{4.2}$	Maximum mean value recorded 2 dpi
Lung	$10^{8.0}$	$10^{7.2}$	Maximum mean value recorded 2 dpi
Muscle	$10^{6.0}$	$10^{2.8}$	Maximum mean value recorded 2 dpi

*dpi; days post inoculation

Chickens show severe symptoms of H5N1 and, in GB, outbreaks are rapidly detected so the risk of HPAI being in the food chain through chicken meat is low. In contrast, ducks show asymptomatic (silent) infection and the disease may be undetected with the possibility of infected duck meat entering the food chain as in Germany (Harder *et al.* 2009).

12.2.2 Titres of H5N1 HPAI virus in infected pigs

Pigs have low susceptibility to infection with H5N1 HPAI. Inoculation of pigs with H5N1 viruses resulted in asymptomatic to mild symptomatic infection restricted to the respiratory tract and tonsils. To determine sites of H5N1 replication in infected piglets, Lipatov *et al.* (2008) analysed 18 organs from infected piglets: nasal turbinate, tonsils, trachea, lungs, olfactory bulbs, brain, heart, whole blood, spleen, liver, stomach, pancreas, small intestine, large intestine, kidney, adrenal gland, diaphragm and skeletal muscle. H5N1 was detected only in tissues from the respiratory organs, namely nasal turbinate, tonsils, trachea and lungs. All the H5N1 viruses studied were found in the lungs of the piglets. The highest titre was $10^{6.5}$ EID_{50}/g of lung tissue. The key point for risk assessment is that muscle from AIV-infected pigs does not contain H5N1, suggesting that pork products from AIV-infected pigs would not pose a significant risk compared to poultry products.

12.3 Pathway term

12.3.1 Thermal inactivation of AIV

Thomas and Swayne (2007) presented the D-values for the thermal inactivation of H5N1 HPAI virus in thigh and breast meat from infected chickens at temperatures of 57°C to 61°C. At the lowest temperature of 57°C, a 4-log reduction was observed in 15 min. At the highest temperature studied (61°C) over 3-log inactivation was observed in 90 sec. It was concluded that cooking chicken meat according to the USDA Food Safety and Inspection Service time-temperature guidelines will inactivate HPAI Korea/03 in a heavily contaminated meat sample with a large margin of safety. The impact of composting on HPAI in lung, trachea and air sacs of experimentally infected birds was studied by Senne *et al.* (1994). At the end of the first 10 days of composting, virus-isolation efforts showed that HPAI virus had been inactivated in all 20 tissue samples, compared with controls stored at -70°C were positive for HPAI.

12.3.2 Survival in manures and chicken faeces

The detection of H5N1 influenza virus in bird faeces in poultry markets (Shortridge *et al.* 1998) raised the question of the stability of the virus. These authors studied the stability of H5N1 in faeces collected from wild birds. When faeces with initial titres of 3.5 to 4.5 log EID_{50} (A/Hong Kong/156/97 H5N1) per g were dried at room temperatures (25°C), infectivity had declined to undetectable levels by day 1. The survival rate was greater in wet faeces at 25°C, declining to 2.5 log on the first day and 1.5 log after 4 days. At higher temperatures (35°C) infectivity dropped to undetectable levels by the second day. When moist chicken faeces were stored at low temperatures (4°C), virus remained viable for an extended period of time with no detectable loss of infectivity over 40 days.

Lu *et al.* (2003) studied the inactivation time of H7N2 in chicken manure at different temperatures. The virus was effectively inactivated by field chicken manure in less than a week at ambient temperature of 15°C to 20°C. However, in SPF (specific pathogen-free) chicken manure the virus survived for 19 days at ambient temperature. Moreover at 4°C the virus was still active in field chicken manure after 20 days and in SPF chicken manure after 23 days.

12.3.3 Inactivation of AIV in water

Breban *et al.* (2009) cite decay rates for AIV in water and use them directly in a predictive model. The decay rates are summarised in Table 12-5.

Table 12-5: Decay rate for AIV in the environment used by Breban *et al.* (2009)

Environmental conditions for wildfowl	Log_e (per year)	Log (per year)	Log (per day)
Breeding grounds during the summer	5	2.2	0.006
Breeding grounds during the winter	1.3	0.56	0.001
Wintering grounds during the winter	5	2.2	0.006
Wintering grounds during the summer	50	21.7	0.06

12.4 Overview of risks of AIV to livestock through application of composted catering waste

The scope of the work here is to briefly examine the risks of avian influenza virus (AIV) through compost and to make recommendations on any further work. There are numerous routes of exposure of poultry (including backyard and housed) to AIV. These include wild birds, agency of man e.g. hands, fomites and vehicles, and contamination of food and water supplies (Gale *et al.* 2009). Harder *et al.* (2009) provide an example of three outbreaks of H5N1 highly pathogenic avian influenza (HPAI) virus in backyard chickens in Germany in 2007 through direct exposure to frozen and uncooked duck meat which was infected. Asymptomatic (silent) infection had occurred in the ducks which were slaughtered and frozen before entering the food chain.

Those poultry which are housed are unlikely to be exposed to compost unless it is used for bedding material or litter and this is not currently permissible in the UK. In contrast, free-range and backyard poultry are more likely to be exposed through access to fields previously amended with compost. The risks posed to backyard poultry from compost produced from catering waste are likely to be insignificant compared to the risks from exposure to migratory wild birds. This is because backyard poultry may be directly exposed to secretions (oropharangeal and faecal) which contain high titres of virus from AIV-infected birds. These secretions may be on the pastures on which the poultry graze and may get into the water and grain supplies. Unlike composted catering waste, the AIV in oropharangeal and faecal secretions from wild birds will not have been inactivated in any way.

It is, however, difficult to directly compare the risks through compost with the risks from other routes such as exposure through indirect or direct contact with raw poultry meat.

These routes include feeding of meat scraps as in Germany (Harder *et al.* 2009), or transfer of meat from scavenging animals (e.g. gulls and rodents) which might carry pieces of raw meat into a poultry house. Human handling of raw meat is also a route, particularly if they come in contact with poultry. Thus, poultry may be exposed to AIV from owners' handling raw infected poultry meat products and then handling the poultry, or the poultry feed (i.e. hand-to-feed contamination), or even the poultry's drinking water utensils and bedding.

To examine the risks through compost in greater detail, a quantitative risk assessment is undertaken for H5N1 HPAI virus. This is based on the model for Newcastle disease virus (Section 8.5). Since H5N1 is lethal in chickens it is unlikely that H5N1-infected chicken carcasses enter the food chain. H5N1 in ducks may be asymptomatic (i.e. with silent infection) and in Germany, H5N1 infected ducks have previously entered the food chain (Harder *et al.* 2009). The risk assessment here assumes that 10,000 H5N1 HPAI virus-infected ducks enter the food chain each year. Live weights of ducks are 1.25 kg for mallards up to 4.9 kg for Peking ducks (Cherry and Morris 2009).

The main route would be through clinically normal ducks (i.e. those not showing clinical symptoms) being imported legally into GB. Sick ducks would not pass the inspections at abattoirs in the EU. While sick ducks have muscle titres of 4 to 6 log EID_{50}/g (Table 12-3), clinically normal ducks have lower titres of 2.0 to 3.4 log EID_{50}/g. As a worst-case, it is assumed here that the legally imported duck meat has 10^4 EID_{50}/g. Assuming there are 2,000 g of meat per duck with 10^4 EID_{50}s per gram, then 2.0 x 10^{11} EID_{50}s enter the food chain. Assuming 2.8% (as for poultry meat, Table 2-3) of the raw duck meat is composted (i.e. 280 H5N1-infected duck carcasses) then 5.6 x 10^9 EID_{50}s are composted.

Preliminary analysis of unpublished APHA data for H5N1 infection in chickens suggests the chicken oral ID_{50} is approximately 10^3 EID_{50}s. Thus 5.6 x 10^6 poultry oral ID_{50}s are composted. With composting removing 4.61-log, a total of 138 chicken oral ID_{50} remain in the compost, giving an arithmetic mean concentration of 3.5 x 10^{-5} chicken oral ID_{50} per tonne of compost. Using decay data from Breban *et al.* (2009) of 0.006 log per day for AIV on wildfowl wintering grounds during the winter (Table 12-5), the individual risk of AIV is 5.53 x 10^{-8} per chicken per year. This assumes that the compost is surface applied, that there is no grazing interval, and that the chicken is consuming only compost every day for a period of 1 year. It gives some reassurance that the risks to backyard chickens are very low.

In relation to the risks of AIV to pigs, some decay of the avian influenza virus will occur during the no grazing ban (which probably could not be enforced for backyard poultry flocks).

13.0 Brief examination of the risks to livestock from pig circoviruses (PMWS and PDNS)

Porcine circovirus type 2 (PCV2) is consistently associated with two diseases of British pigs - porcine dermatitis and nephropathy syndrome (PDNS) and post-weaning multisystemic wasting syndrome (PMWS), although not all pigs infected with PCV2 develop PMWS (Ghebremariam and Gruys 2005). It is acknowledged that additional conditions are required for the disease to be severe in growing pigs (Madec *et al.* 2008). Co-factors are thought to act as triggers. The spread of such triggers, which may or may not be infectious, could have played a role in PMWS dissemination via normal national and international trade. The co-factors could be another infectious agent or a non-infectious management change such as use of pesticides (Woodbine *et al.* 2006).

Among six different clinical manifestations involving respiratory, enteric, nervous and reproductive signs, PMWS is the most important and studied disease. PMWS was first reported in Canada in 1991 (cited in Woodbine *et al.* 2006) and PCV2 was discovered in 1998.

PCV2 related diseases have been reported throughout the world (Madec *et al.* 2008; Rose *et al.* 2012) and horizontal transmission is widely documented with contact between pigs as the main route of transmission. However, experimental inoculation of PCV2 to pigs does not give consistent results, and severe clinical symptoms, as encountered in the field, are rarely obtained. Allan *et al.* (2007) reported the distribution of PCV2 genotypes taken from PMWS-affected and PMWS-nonaffected farms in Ireland over a 9 year study period. At least two distinct genotypes of PCV2 have been circulating on pig farms in Ireland. Allan *et al.* (2007) conclude that there does not appear to be a direct relationship between infection with the different genogroups of PCV2 and the development of PMWS.

Therefore although it may be possible to complete a risk assessment for PCV2, a risk assessment for the disease PMWS is not yet feasible. Most of the risk factors identified in surveys relate to poor biosecurity and inadequate hygiene/husbandry/herd management. No clear relationship with genotype and strain virulence is apparent, and isolates detected in PMWS-positive pigs could also be detected in healthy pigs from healthy farms. Colostrum composition and colostrum intake may be key components of disease expression, although losses are greatly reduced by applying appropriate hygiene and husbandry practices. Transmission of PCV2 to sows and piglets has been demonstrated through semen from infected boars (Gava et al. 2008).

13.1 Risk factors

Woodbine *et al.* (2006) studied the epidemiological risk factors for PMWS in GB between August 2003 and August 2004. PMWS initially occurred in the south of England, moving west and then northwards. The pattern of spread included long distance and local spread around infected farms. The greatest risk was associated with having a large number (>600) of sows. During the FMD outbreak, when animal movement restrictions were in place, the greatest risk was associated with farms with a grower/finisher herd nearby and/or where visitors were allowed onto farms. In Sweden, intensity of rearing, disease prevention measures and immaturity of the piglets appear to be important as predisposing factors to PMWS (Wallgren *et al.* 2007). Rose et al. (2003) reported that in French farms, large pens in weaning facilities increased the risks of PMWS while long empty periods in weaning facilities, treatment against external parasites and housing the sows in collective pens (as opposed to individual pens) during pregnancy decreased the risks.

Rose et al. (2012) have reviewed the epidemiology and transmission of PCV2. Direct contact is the most efficient infectious route due to the simultaneous exposure of susceptible pigs to contaminated respiratory, digestive and urinary secretions. The probability of transmission is

strongly limited by distance between infectious and susceptible animals. According to Rose et al. (2012) farm to farm transmission is restricted to the introduction of infected animals or infected animal products such as semen. The amount of PCV2 DNA in nasal and faecal samples from infected pigs on Spanish farms ranged between 4 to 6 $\log_{10}$ genomic copies per swab with up to 8 and 10 $\log_{10}$ genomic copies for faecal and nasal swabs samples, respectively (Rose et al 2012). In Canada, around 7 $\log_{10}$ genomic copies of PCV2 were present per gram of pooled faeces from PMWS/PDNS affected pigs herds. In contrast, for non-affected herds only 2 $\log_{10}$ genomic copies per gram were recorded (Rose *et al.* 2012). A significant difference in the quantity of PCV2 genome load was apparent between PMWS/PDNS-affected animals and non-affected animals. Rose et al. (2012) cite a "within-pen" R0 value of 8.9 which means that introduction of an infected pig into a naive susceptible population of pigs will directly infect 8.9 other pigs.

Rose *et al.* (2012) note that the risks of PCV2 to pigs through other routes such as feed ingredients are unknown. The use of spray-dried plasma on farms represents a potential risk and PCV2 was transmissible to naïve pigs when given experimentally produced spray-dried plasma (cited in Rose et al 2012). Other sources of potential introduction and persistence are fomites and other animal species, including rodents (Rose *et al.* 2012). PCV2 can be recovered from dead mice and rats, but the practical significance of this is unknown.

13.1.1 Oral transmission of PCV2 to pigs through meat

Some work has been completed recently on the transmission through meat of porcine circovirus type 2. Opriessnig *et al.* (2009) reported that naïve pigs fed for three consecutive days with either skeletal muscle, bone marrow, or lymphoid tissues from PCV2-infected pigs all became PCV2 viraemic. Opriessnig *et al.* (2009) conclude that pork products are a possible source of introduction of PCV2 into pig populations. Their results indicate that uncooked lymphoid tissues, bone marrow, and skeletal muscle from PCV2 viraemic pigs contain sufficient amounts of infectious PCV2 to infect naïve pigs by the oral route. However, as noted by Rose *et al.* (2012) delayed seroconversion through the oral route suggests this route is not as efficient as direct contact with infected pigs. Rose *et al.* (2012) write that the significance of the oral route is unknown.

13.2 Source term

Opriessnig *et al.* (2009) collected skeletal muscle, bone marrow and lymphoid tissues from pigs experimentally inoculated with PCV2 14 days post-inoculation. Lymphoid tissues contained the highest amount of PCV2, bone marrow contained a lower amount of PCV2 and skeletal muscle contained the lowest amount. Segales *et al.* (2005) reported that tracheo-bronchial swabs had the higher PCV2 load followed by serum, tonsillar, nasal, faecal, and finally urinary swabs in a study of 146 pigs (of which 42 were PMWS affected, 29 were subclinically infected, 75 were non-PMWS and PCV2 negative by in-situ hybridisation). Viral loads were higher in pigs with PMWS. Segales *et al.* (2005) concluded that PCV2 is excreted through respiratory (nasal and tracho-bronchial) and oral (tonsillar) secretions, urine and faeces of both PMWS and non-PMWS affected pigs, with higher viral loadings being associated with the presence of PMWS lesions. Gava *et al.* (2008) reported that several tissues (mainly the lymph nodes, bone marrow and spleen) were positive by PCR for PCV2 in 10 of 12 piglets born of sows impregnated with semen of PCV2 positive boars. In 8 of the 12 piglets tissues including the lymph nodes, tonsils and bulbourethral glands were positive by immunohistochemistry. Rose *et al.* (2012) cite a study in which between 6 and 12 $\log_{10}$ PCV2 genome copies/ml were detected in serum and liver from five gnotobiotic pigs experimentally infected with PCV2. PCV2 infects the mammary gland of sows and is shed in milk (Park *et al.* 2009; Ha *et al.* 2009). After intranasal inoculation of pregnant sows, PCV2 was detected in milk from day 1 of lactation for 27 days.

13.3 Thermal stability of porcine circovirus type 2

PCV2 is a non-enveloped virus and is more thermo-resistant than enveloped viruses such as avian influenza virus (Blumel *et al.* 2008). O'Dea *et al.* (2008) determined the effect of heat treatment on an Australian strain of PCV2. An indication of viability was determined by a combination of reverse transcriptase PCR to detect viral RNA and immunohistochemistry to visualise viral capsid antigen. PCV2 retained infectivity when heated at 75°C for 15 min but was inactivated by heating at 80°C and above for 15 min. The immunohistochemistry results (Table 1 of O'Dea *et al.* 2008) show no inactivation of PCV2 after 15 min at 56°C compared to 24°C with significant inactivation after 15 min only occurring at 70°C or above. Indeed O'Dea *et al.* (2008) demonstrated 3.06 log inactivation is 15 minutes at 70°C. Welch *et al.* (2006) reported similar findings with no inactivation of PCV2 after 30 min until temperature of 70°C or above (wet heat). Water content is important, and dry heat for 30 min had limited effect on PCV2 even up to 120°C (Welch et al. 2006). Of great significance in the study by Welch *et al.* (2006) is the 1.33-log reduction of PCV2 observed during pasteurisation at 60°C for 24 hours (wet heat). It is tempting to extrapolate this to a 2.6-log inactivation in 48 h, which would almost satisfy EC 208/2006 in terms of the validation requirement for inactivation of thermoresistant viruses by proposed novel composting and biogas processes. It is concluded that composting at 60°C for 48h will have some effect on inactivation of PCV2 and may achieve significant removals. In this respect, it is interesting to note that a 3-log inactivation of PCV2 is achieved more rapidly than for parvoviruses at 70°C (Donaldson et al. 2011). Parvoviruses are given as an example for thermo-resistant viruses in EC 208/2006.

13.4 Overview of risks of PCV2 through composting

There is clear evidence that PCV2 is present in pig tissues including muscle, bone marrow and mammary gland, and pig milk. Indeed Opriessnig *et al.* (2009) have demonstrated that uncooked pig meat products (including bone and skeletal muscle) can infect naïve pigs through the oral route. The temperature inactivation data available for PCV2 suggests that composting (60°C for 48 h) could achieve significant reductions in the risks from any PCV2 in raw pig meat, and may even approach the 3-log inactivation required for validation of novel processes in EC 208/2006. Further inactivation would be anticipated in two stage composting, with a second period of 48 h at 60°C. There is some evidence that PCV2 is inactivated more rapidly than parvoviruses (albeit at 70°C). Although the risks of transmission of PCV2 to pigs through composted catering waste may be low relative to other routes, it cannot at this stage be demonstrated that the risks are negligible. In this respect it should be noted that Rose *et al.* (2012) conclude there is potential for the transmission of PCV2 through several indirect routes such as oral, aerosol, fomites and vaccine routes. However, Rose *et al.* (2012) add that "The risk represented by these routes must be weighed compared to the reservoir represented by the pig population, the high transmissibility of the virus between pigs through direct contacts and indirectly via pig secretions and resistance in the environment".

The magnitude of any risk through compost would reflect loadings of virus in pig meat in GB. A quantitative risk assessment cannot be undertaken at this stage because of lack of quantitative data on virus loadings in pig meat and on dose-response. Similarly, a risk assessment for PMWS is not yet feasible as the exact role of PCV2 in these diseases is not clear.

Pasteurisation at 70°C for 1 h may achieve 1.5 log reduction (based on Figure 3A of Welch *et al.* 2006) which is similar to the 1.33 log reduction achieved by 24 h at 60°C (see Figure 1 of Welch *et al.* 2006). The reduction in risks of PCV2 transmission through composting at 60°C for 2 x 48 h may be of similar magnitude to that through pasteurisation at 70°C for 1 h, although this has not been formally assessed, and the effects of particle size must also be considered.

14.0 Brief examination of the risks of porcine parvoviruses

Porcine parvovirus (PPV) is ubiquitous among swine throughout the world (Mengeling 1986). In major swine-producing areas such as the Midwestern United States, infection is enzootic in most herds, and with few exceptions sows are immune. The sero-epidemiologic data indicate that exposure to PPV is common. The most common routes of infection for postnatal and prenatal pigs are oronasal and transplacental, respectively (Mengeling 1986) and Truszczynski (2008) notes that pigs may be infected through ingesting or inhaling virus-loaded secretions. Cowart and Casteel (2001) write that the virus is present in practically 100% of swine herds worldwide and survives in the environment for 16 – 20 weeks. In male pigs and nonpregnant females, the animal seroconverts and eliminates the virus with no clinical signs (Cowart and Casteel 2001). The major and usually only clinical response to infection with PPV is maternal reproductive failure (Mengeling 1986) with mummified foetuses being born. Contaminated premises are probably the major reservoirs of PPV. Infected pigs transmit PPV for about 2 weeks after exposure, while the pens in which they were kept remain infectious for at least four months (Mengeling 1986.) Truszczynski (2008) notes that PPV is amongst the most resistant viruses to environmental factors, heat and disinfectants and concludes that since PPV infection is frequent and cannot be eradicated, losses are considerable.

14.1 Presence in meat

A search of literature has not revealed any papers that provide data on PPV titres specifically in meat. However, Wilhelm *et al.* (2005) reported PPV genomes of 10^{12} to 10^{15} per 10^{6} cells in organs from infected piglets after transplacental infection. The organs studied included the small intestine, heart, lung, liver, kidney, spleen, thymus and gonads. In young pigs and probably older breeding stock, the virus replicates extensively and is found in many tissues and organs with a high mitotic index, but otherwise can be considered pantropic, and as a result is probably present in most secretions and excretions during the acute phase of infection (Mengeling 1975). Viral antigen is especially concentrated in lymphoid tissues (Mengeling 1986). During acute infection the virus is shed by various routes, including semen (Mengeling 1986). Pogranichniy *et al.* (2008) found that follicular fluid samples from abattoir-obtained pig ovaries tested positive for PPV in six of 49 animals. Feeding mummified foetuses to pigs promotes infection (Cowart and Casteel 2001). It is concluded that catering waste containing raw pork products, particular kidney, liver, heart or gonad, could contain PPV.

14.2 Resistance to heat

Parvoviruses are non-enveloped viruses and therefore more resistant to heat than enveloped viruses (Blumel *et al.* 2008). Animal parvoviruses including porcine parvovirus (PPV) have been used as surrogates for human parvoviruses to validate different methods of heat inactivation during manufacture of blood plasma products.

Porcine parvovirus

Blumel *et al.* (2008) demonstrated a slow but significant inactivation of PPV at temperatures of 80°C with a 4-log reduction over 48 hours in conditions of low residual moistures (dry heat). Inspection of Figure 2 of Blumel *et al.* (2002) suggests that 0.5 to 1.0 log inactivation of PPV may occur after 60 minutes at 60°C. Sofer *et al.* (2003) cite a 3.4-log inactivation of PPV in 10 hours at 60°C. Indeed, 10 hours is considered a requirement for PPV inactivation at 60°C (see Sofer *et al.* 2003). The time-temperature conditions for composting are based on all meat achieving at least 56°C for at least 4 hours in each composting step. Lund *et al.* (1996) reported a 5-log inactivation of PPV in 48 hours at 55°C in a thermophilic biogas reactor. A literature review based on thermostability plots concluded that the time for a 3-log inactivation of porcine parvovirus is approximately 35 hours at 56°C (Donaldson *et al.* 2011). It is concluded that the long time periods (48 hours) at 56°C to 60°C prescribed for

composting in the Animal By-Products Regulations (2011) may contribute to considerable inactivation of PPV.

Sahlstrom *et al.* (2008) have assessed the effect of pasteurisation, as set by the European Regulation EC1774/2002 on selected pathogens. Although they demonstrated a 3.15-log inactivation of PPV in 1 hour in aqueous medium at 70°C, Sahlstrom *et al.* (2008) concluded that heat-resistant viruses such as porcine parvovirus were not completely inactivated by heat treatment at 70°C for 30 min or 60 min.

Other parvoviruses

It should be noted that there is considerable variation between parvoviruses from different species in their resistance to heat. Human parvovirus is more sensitive to pasteurisation at 60°C than animal parvoviruses (Blumel *et al.* 2002). Indeed animal parvoviruses widely withstood pasteurisation of albumin, while human parvovirus was inactivated. Thus, PPV was widely resistant at 60°C, while human parvovirus B19 underwent greater than 4-log reduction after 10 minutes. Sauerbrei and Wutzler (2009) have tested a number of viruses for resistance to dry heat. While poliovirus type 1 was inactivated by treatment at 75°C for 1 hour, the infectivity of bovine parvovirus was little affected by exposure to these conditions. However, over longer periods of time, inactivation of bovine parvovirus does occur at temperatures used for composting. Thus Srivastava and Lund (1979) report a 4.0-log inactivation of bovine parvovirus at 60°C in 24 hours. Moreover, Durham and Johnson (1984) demonstrated a >3.0-log reduction in infectivity of bovine parvovirus at 56°C in 48 hours in aqueous buffer medium. However, at 70°C (aqueous buffer) Durham and Johnson (1985) observed no inactivation of bovine parvovirus within the first 2 hours, although >4.0-log inactivation occurred after 8 hours at 70°C.

A literature review based on thermostability plots concluded that the time for a 3-log inactivation of porcine parvovirus is approximately 35 hours at 56°C compared to approximately 20 hours for bovine parvovirus (Donaldson *et al.* 2011). According to the thermostability plots of Donaldson *et al.* (2011) the time for a 3-log inactivation for porcine parvovirus was longer than that for bovine parvovirus at both 56°C and 60°C, and porcine parvovirus appeared to be the most thermoresistant of the 14 viruses studied. It is interesting to note that the degree of inactivation of human parvovirus B19 (see Figure 1 of Blumel *et al.* 2002) varies greatly between 52°C and 60°C. Thus, at 60°C there was a greater than 4 log inactivation after about 1 min, while at 54°C there was only 3 log inactivation after 60 minutes, with virtually no inactivation at 52°C (Blumel *et al.* 2002).

14.3 Overview of risks of porcine parvovirus

Belschner and Love (1984) note that a piggery is much better off with parvovirus endemic than to be free of it and live with the prospect of its introduction, but add that if the virus is not present in a piggery then every precaution should be taken to keep it out. PPV is ubiquitous and thus at the national level, a case of PPV infection is very different to a case of an exotic virus such as FMDV in GB. Indeed, one form of prevention actually involves natural infection of gilts (immature female pigs) before breeding (Cowart and Casteel 2001) by grinding up PPV-infected material such as mummified stillborn pigs and foetuses (which died in pregnancy from PPV-infection) and faeces and feeding to the gilts. The results of this method are questionable and imply the potential danger of disseminating more harmful agents in a herd (van Leengoed *et al.* 1983). However, being endemic is not a reason for allowing further spread of pathogen and if PPV is not present in a piggery then every precaution should be taken to keep it out (Bleschner and Love 1984).

Pigs in acute phases of infection shed the virus in faeces (Mengeling 1986), which could contaminate the meat during carcass preparation, and high levels of viral DNA have also been detected in heart, liver and kidneys of infected piglets (Wilhelm *et al.* 2005). Thus, PPV

is likely to be present in pig meat, although lack of information on loadings of infectivity precludes a quantitative risk assessment being undertaken here. However, significant inactivation (>4.0-log) of PPV may occur over the time scales of composting (2 x 48 hours) at temperatures of 56°C to 60°C. Lund *et al.* (1996) reported a 5-log inactivation of PPV in 48 hours at 55°C in a thermophilic biogas reactor. A literature review based on thermostability plots concluded that the time for a 3-log inactivation of porcine parvovirus was approximately 35 hours at 56°C (Donaldson *et al.* 2011). Thus, long periods of time (8 to 24 hours) seem to be important in the inactivation of bovine parvoviruses at both 56°C and 70°C (see Durham and Johnson 1985). It is concluded that the long time periods (48 hours) at 56°C to 60°C prescribed for composting in the Animal By-Products Regulations (2011) should achieve >3.0 log inactivation of PPV.

In conclusion, composting according to the Animal By-Product Regulations (2011) should greatly reduce the levels of any PPV in catering waste and should achieve the >3.0-log inactivation required for novel transformation approaches in EC208/2006. Belschner and Love (1984) write that the main ways in which porcine parvovirus can be introduced are through pigs that are actively infected and excreting the virus and by faecal contamination of introduced pigs, clothing or boots of personnel. The inactivation of PPV by composting should minimise the risks from pig meat in compost compared to these routes. However, although the risks through compost are lower than through other routes, it cannot at this stage be demonstrated that the risks are negligible. The magnitude of the risk would reflect loadings of virus in pig meat in GB. A quantitative risk assessment cannot be undertaken at this stage because of lack of information on loadings of PPV infectivity in meat. In this respect PPV-free pig farms could consider not taking catering waste-derived compost until it is possible to quantify those risks using national GB data.

15.0 Brief examination of the risks to livestock from MRSA

Methicillin-resistant *Staphylococcus aureus* (MRSA) primarily causes human disease, and animals have only recently been considered as a source of infection (Lewis *et al.* 2008). Given the importance of *Staph. aureus* as a cause of mastitis in cattle and the widespread usage of intramammary antibiotics in cattle, it is perhaps not surprising that the first isolations of MRSA from animals were in milk from mastitic cattle in the 1970s (see Leonard and Markey 2008). Since then, MRSA has been found in a variety of other domestic species including dogs, cats, horses, sheep, pigs and chickens (cited by Leonard and Markey 2008). Pigs can constitute a separate MRSA reservoir and be a source of a novel and rapidly emerging type of MRSA in humans, namely MRSA clonal complex (CC) 398 (Armand-Lefevre *et al.* 2005). Clinical infection by MRSA CC398 was described in the daughter of a pig farmer in the Netherlands in 2004, with 23% of pig farmers being seropositive (cited in Lewis *et al.* 2008). A study in 2005 in France identified pig farming as a risk factor for increased nasal colonisation with *Staph. aureus* (cited in van Duijkeren *et al.* 2007). Although transmission appears to be primarily between animals, indistinguishable isolates have been found in their human contacts, particularly those with occupational exposure (Lewis *et al.* 2008). There is relatively little information on the levels of MRSA in pigs. Indeed it was only recently that pigs were identified as a source of CC398 in Denmark (Lewis *et al.* 2008). The European Union baseline survey on the prevalence of MRSA in breeding pigs, initiated in January 2008, is an important step in addressing the size of the reservoir in pigs.

15.1 Risk factors to livestock

Supplier farms that sell MRSA-colonised pigs to other farms play a role in spreading the organism. It was recently reported that 209 (39%) of 540 finishing pigs at Dutch slaughterhouses were MRSA-positive. Antimicrobial drugs used on farms may select for CC398 (cited in van Duijkeren *et al.* 2007). No data on prevalence levels in GB pigs have been found.

15.2 Presence in meat

Large numbers of MRSA were cultured from skin lesions of piglets with exudative epidermitis in the Netherlands (van Duijkeren *et al.* 2007). It is believed the MRSA were clinically relevant in the skin condition. Exudative epidermitis is a skin disease normally caused by *Staph. hyicus* which was not detected in the lesions (van Duijkeren *et al.* 2007). *Staph. aureus* is normally located on the skin and mucosa, and as such, loadings in fresh meats may be low due to singeing and scolding of skin prior to carcass splitting during processing. However, pork skin is often attached to chops, for example, and furthermore, *Staph. aureus* could grow if inoculated on meat stored at room temperature through contact with the fingers of an infected person.

15.3 Inactivation by heat

There is evidence that *Staph. aureus* is more temperature resistant at 64°C and 70°C than *Salmonella enterica* and *Listeria monocytogenes*. Li *et al.* (2005) reported decimal reduction times (D-values) of 0.193 min, 0.364 - 0.440 min and 1.304 – 1.768 min for *Salmonella* serotypes, *L. monocytogenes* and *Staph. aureus,* respectively, in high solid egg mixes at 64°C. They concluded that the *Staphylococcus* strains were 6.2 to 11.7 times more heat resistant than *S. enterica* serotypes.

Kennedy *et al.* (2005) investigated the thermal inactivation of *Staph. aureus* and the impact of chilled storage on thermotolerance. This scenario would be particularly relevant for composting of raw meats which may have been stored in refrigerators for considerable periods. Those authors found that chilling did not increase the resistance of *Staph. aureus* to thermal destruction during subsequent thermal processes such as cooking. Moreover, they reported that the thermal inactivation of *Staph. aureus* follows first order kinetics with maximum calculated D-values of 127.9 min at 50°C, 21.1 min at 55°C and 6.6 min at 60°C.

Using the D-value of 21.1 min at 55°C and assuming first order kinetics, it is calculated that a greater than 11 log reduction of *Staph. aureus* could occur after 4 h of composting at 55°C. It is concluded that composting for 60°C for 2 x 48 hours (as set out by Gale 2002) would achieve the required 4.61 log reduction of MRSA allowing for by-pass.

15.4 Overview of risks of MRSA to livestock

It is suggested here that the risk of colonisation of pigs through contact with scraps of MRSA-infected pig skin following composting of catering waste is likely to be remote, particularly when compared with the risks from direct contact of pigs during sales. This is because singeing and scalding during processing of the pig carcass together with destruction of the *Staph. aureus* during composting will greatly reduce any risks of MRSA to pigs from composted catering waste.

Although *Staph. aureus*, may be more temperature resistant than *Salmonella enterica* serotypes, available evidence suggests that composting for 60°C for 2 x 48 hours (as set out by Gale 2002) would achieve log reductions in *Staph. aureus* comparable to those of salmonellas, even allowing for by-pass of the two step composting process.

16.0 Discussion

16.1 Measures of risk presented in the risk assessment

The quantitative risks are presented here in two ways, namely the group risk as represented by the number of infections nationally per year, and the annual risk to the individual animal.

For the individual farmer, the more relevant measure is the risk to the individual animal grazing or housed for one year on land to which compost has been applied. This is the individual risk. As an example, an individual risk of 10^{-6} per animal per year equates to a 0.1% chance each year of having an infected animal on a farm which holds 1,000 animals each and every day of the year. Exchange of individual livestock between farms does not change the risk because pathogens do not act cumulatively. The criterion is that livestock must average 1,000 head per day on the farm over the one year period, and must have access only to compost-amended soils every day for that year. In the approach adopted here, the individual risk is not affected by the density of livestock on the farm, and applies equally to each and every animal which spends a whole year on the farm. Thus, for example, if stock densities are higher, it is assumed that each animal will still consume the same amount of soil/compost per year.

For regulators and risk managers, the more relevant expression of risk is the number of infections predicted to occur nationally per year – or alternatively, its reciprocal, which is the time (expressed in years) expected to pass on average between each infection in all livestock across GB as a whole.

16.2 Overview of the approach for calculating exposure to livestock in the risk assessment

The calculation of the number of infections in GB as a whole is complicated by the uncertainty associated with the number of animals grazing on land to which compost has been applied, and the length of time for which they graze. In the original compost risk assessment, this was addressed by assuming England and Wales comprise a single field of 9,537,300 ha within which all the livestock randomly graze, and to which 500,000 tonnes of compost is applied with a rate of 10 tds/ha to 50,000 ha. For this revised assessment, a higher application rate of 20 tds/ha is used. It is estimated (Section 2.4.1) that if all the catering waste produced in the GB were composted, it would produce 3,913,200 tonnes dry solids (tds) of compost (dry weight), which at an application rate of 20 tds/ha would cover 195,660 ha. Of this it is assumed (based on GB land use statistics for June 2010, see Table 7-2) that 58.2% i.e. 113,897 ha, are grassland used for grazing of livestock.

Knowing the soil ingestion rates (per animal per day) allows the proportion of compost ingested by livestock across GB over a given period of time to be calculated. These assumptions give worst-case estimates of total compost consumption because 95th percentile soil consumption values together with the maximum "landscape scale" livestock densities are used. It is not appropriate to use actual "farm field" densities of livestock for calculating the total number of exposed livestock because the risk assessment would, in effect, be assuming that compost is being selectively applied only to grassland that is all grazed, all year. One way of avoiding such an excessively precautionary approach is to use "landscape scale" grazing densities, since these should better reflect the densities of livestock on the whole mosaic of grazed compost-amended grassland and non-grazed compost-amended grassland. Thus, although a maximum "farm field" stocking rate for pigs of 20 sows/ha has been reported by Cronin (1996) with sheep at 20/ha (English Beef and Lamb Executive, pers. comm. 12 June 2009) and beef cows at 6.1/ha (Defra 2009b), these values have not been used here.

Therefore, it is assumed that each of those 113,897 ha of grassland in GB has 20 tds compost applied and a livestock density of 2.8 pigs, 5.1 sheep or 1.59 cattle for the whole year. These livestock densities are the maximum recorded for the Defra 2005 England

Census and represent the maximum number of animals recorded in a given census area, i.e. they are 'landscape scale' livestock densities.

Using stocking densities for pigs of 2.8/ha, sheep of 5.1/ha and cattle of 1.59/ha, the model assumes 1.17%, 3.74% and 1.9%, respectively, of the total 3,913,200 tds of compost (dry weight) produced from catering waste annually in GB is consumed when surface-applied by pigs, sheep and cattle respectively (Table 7-4). This is based on there being no dilution in the soil for surface-applied compost over the whole one year period such that grazing livestock ingest compost to the complete exclusion of soil. Tilling into a depth of 10 cm (assumed for minimum tillage) or to a depth of 25 cm (plough depth) gives dilution in the soil and decreases the exposures by 75-fold and 188-fold, respectively.

16.3 Summary of worst-case assumptions

The risk assessment is based on worst-case assumptions to overcome uncertainty. These are summarised here:

- The revised risk assessment (as in the original) assumes that all the meat discarded in GB through catering waste over a period of one year goes to compost.
- On the grassland to which catering waste-derived compost is applied, livestock graze all year at maximum livestock densities recorded in the 2005 Defra Census for England (Table 3-14).
- 95th percentiles for soil consumption by cattle and sheep were used (Table 3-12), and the maximum in the case of pigs.
- In the case of surface-applied compost, livestock ingest compost to the complete exclusion of soil, every day for a period of one year. There is thus no dilution of the compost in the soil for surface-applied compost.
- In total, 6.8% of the catering waste-derived compost produced each year is assumed to be ingested by farm animals if surface applied.
- 95th percentiles are used for the amounts of BSE and scrapie infectivity entering the food chain. For exotic viruses, loadings are calculated on the basis of the infected carcasses being at the highest viraemic state together with 95th percentiles for the amount of infected meat estimated to be illegally imported into GB.
- It is assumed there is no inactivation of BSE by composting and just 0.69-log inactivation of scrapie infectivity. Recent experiments suggest composting and mesophilic anaerobic digestion, in particular, may remove significant amounts of scrapie infectivity.
- The risk assessments presented here for NDV and AIV assume 10,000 infected carcasses enter GB per year illegally. This is 15 tonnes of infected chicken meat and 20 tonnes of infected duck meat for NDV and AIV, respectively. Estimates of the amounts of pig meat infected with exotic pig viruses and illegally imported into GB are in the order of a few hundred kilograms per year. Amounts of illegally imported poultry meat infected with AIV and NDV would presumably be of similar magnitude.
- The number of *T. gondii* cases is calculated assuming that all 596,728 sheep grazing at 5.1 sheep/ha on the 117,005 ha of grassland (UK) treated with compost at 20 tds/ha are pregnant ewes, and that grazing ewes are pregnant all year.
- Although decay and dilution of pathogens in the soil are accommodated, no allowance is made for leaching of those pathogens to lower soil layers.
- Decay data used were taken from experiments performed during winter months or at low temperatures for which pathogen decay rate is lower than might be expected when compost is applied during the main grass growing season.
- No decay in soil allowed for BSE, scrapie or SVDV.
- No more than 5-log decay allowed on land over period of one year.

16.4 Overall results

The individual risks for CSF, ASF, FMD, SVD, NDV, AIV, *T. gondii*, BSE and scrapie are set out in Table 16-1. The predicted number of years between infections in livestock is set out in Table 16-2 for each pathogen. For scrapie the number of infections predicted per year is set out in Table 16-3.

Table 16-1: Overall summary of the individual risks of disease (risk per head per year). *Note predicted individual risks apply to an animal spending all day, every day for 1 year on land to which compost has been applied once at 20 tds/ha.

Pathogen	Surface applied (no dilution in soil)		Minimum tillage to 10 cm depth	
Grazing ban	**None**	**2 month**	**None**	**3 week**
CSF	0.92×10^{-6}	0.76×10^{-9}	1.22×10^{-8}	0.88×10^{-9}
ASFV	1.1×10^{-10}	1.7×10^{-12}	1.4×10^{-12}	3.4×10^{-13}
FMDV cattle[1]	0.4×10^{-9}	0.52×10^{-12}	0.5×10^{-11}	0.5×10^{-12}
SVDV	3.7×10^{-15}	3.7×10^{-15}	4.9×10^{-17}	4.9×10^{-17}
NDV	5.3×10^{-7}	$9.1x\ 10^{-8}$	7.0×10^{-9}	3.8×10^{-9}
AIV	5.7×10^{-8}	ND	ND	ND
[2]*T. gondii*	8.9×10^{-5}	1.7×10^{-5}	1.2×10^{-6}	0.67×10^{-6}
BSE	0.8×10^{-6}	0.8×10^{-6}	1.1×10^{-8}	1.1×10^{-8}
Classical scrapie (95th percentile)	1.34×10^{-4}	1.34×10^{-4}	1.8×10^{-6}	1.8×10^{-6}
Atypical scrapie (95th percentile)	1.65×10^{-5}	1.65×10^{-5}	2.2×10^{-7}	2.2×10^{-7}

[1]Highest risk amongst livestock categories considered (cattle, sheep and pigs) reflecting amount of soil ingested
[2]abortion in pregnant sheep
ND, not done as grazing ban and tilling in to 10 cm difficult to enforce for backyard poultry

Table 16-2: Overall summary of results. Predicted number of years between cases of disease in GB

Pathogen	Surface applied (no dilution in soil)			Minimum tillage to 10 cm depth	
Grazing ban	**None**	**3 week**	**2 month**	**None**	**3 week**
CSF	3.4	48	4,110	256	3,579
ASFV	29,574	123,400	1,858,000	2,218,000	9,254,000
FMDV sheep	7,773	80,771	5,392,000	583,000	6,058,000
SVDV	8.6×10^{8}	8.6×10^{8}	8.6×10^{8}	6.4×10^{10}	6.4×10^{10}
T. gondii[‡]	0.019	0.335	0.1	1.4	2.5
BSE	7	7	7	511	511

[‡]abortion in pregnant sheep for UK

Table 16-3: Overall summary of results for scrapie. Predicted number of infections in GB per year

Pathogen	Surface applied (no dilution in soil)			Minimum tillage to 10 cm depth	
Grazing ban	None	3 week	2 month	None	3 week
Classical scrapie (95th percentile)	14.7	14.7	14.7	0.195	0.195
Atypical scrapie (95th percentile)	9.6	9.6	9.6	0.13	0.13
Total (classical and atypical)	24.3	24.3	24.3	0.325	0.325

16.5 Discussion of results for each pathogen

Exotic mammalian livestock viruses – CSFV, ASFV, SVDV and FMDV

As found in the original 2002 risk assessment, the risk posed (to pigs) from CSFV was the highest predicted for the four exotic viruses CSFV, ASFV, SVDV and FMDV. Indeed, the risks from ASFV, SVDV and FMDV can be dismissed as negligible. The individual risk of CSFV for a pig housed in a field for one year to which compost has been surface applied is low, at 0.9 x 10^{-6} per pig per year with no grazing ban. Therefore a farm with 1,000 pigs housed on land for a year immediately after application (i.e. no grazing ban) of surface-applied compost would expect a 0.09% chance of a case of CSF over that year.

For GB as a whole, however, this translates into one CSF infected pig every 3.4 years if no grazing ban is implemented (assuming the pig density is 2.8/ha across all 113,897 ha of grassland to which 58.2% of the 3,913,200 tds of catering waste-derived compost has been surface-applied). This reduces to one infected animal every 4,110 years if pigs are not allowed to graze for a period of 2 months on land to which the compost has been surface-applied. This is the grazing ban period recommended from the original risk assessment for pigs and in the Animal By-Products Regulations (2011). The individual risk is 0.76 x 10^{-9} per pig per year for surface-applied compost with a 2 month grazing ban. The chance of a case of CSF on a farm with 1,000 pigs all year is remote at 0.00008%. Tilling in to a depth of 10 cm with a grazing ban for pigs of 3 weeks predicts one CSF infected animal every 3,579 years in GB as a whole.

The net reduction assumed in the risk assessment for exotic livestock viruses (FMDV, CSFV, ASFV, SVDV, AIV and NDV) by the two stage composting process is 4.61-log. This is based on the premise that 6-log reduction in the pathogen can be achieved after 4 h at 56°C as represented by the minimum conditions of the "hot part" of composting at the centre of a 40 cm diameter particle. While this has been demonstrated for SVDV (see Table 4-3 and Turner and Williams 1999) and ASFV (Plowright and Parker 1967), experiments to date have not used sufficiently high starting titres or have not been run for long enough time periods to demonstrate 6-log reduction for CSFV and FMDV (Turner and Williams 1999; Turner *et al.* 2000). The maximum inactivation demonstrated for CSFV is >4.2-log after 5 min at 55°C (Turner *et al.* 1999) and >5.0-log for FMDV after 36 min at 50°C (Kamolsiripichaiporn *et al.* 2007). For the baseline conditions it is not unreasonable to assume that 56°C for 4 h will achieve a 6-log inactivation of FMDV and CSFV. For FMDV, this is supported by the finding of Mateo *et al.* (2007) that thermostable variants of FMDV do not exist. There is also some evidence that FMDV is inactivated by heat less rapidly in meat compared to aqueous media and slurries, although no experiments have been conducted at temperatures of <63°C.

With a 3 week grazing ban and tilling to 10 cm depth, the predicted risks of infection of livestock with CSFV or FMDV in GB are still negligible (i.e. one CSF case in 714 years). In

conclusion therefore, even if 6-log inactivation of FMDV and CSFV cannot be achieved in the "hot part" of each composting step, the predicted risks to livestock from CSFV and FMDV are still acceptably low using the log inactivation rates which have been demonstrated experimentally. The bulk medium is at 60°C for 48 h, where greater inactivation would be achieved. Furthermore the meat fraction is composted twice such that the bulk experiences 60°C for 2 x 48 h.

The sensitivity analyses presented in Section 9.5 clearly support the case for a 2 month grazing ban for pigs as set out in the Animal By-Products Regulations (2011).

Newcastle disease (NDV) and avian Influenza virus (AIV)
In terms of exposure it is considered that composts would not be used in pastures for commercial free-range poultry operations, and certainly not in commercial housed poultry operations. Exposure should therefore focus on the estimated 38,000,000 backyard chickens in GB. It is not possible to estimate the number of backyard poultry which could be exposed to compost, although the proportion is likely to be small. Therefore predictions of the overall number of poultry infected through composted catering waste are not undertaken here.

The risk assessments for NDV and AIV focus on the individual risk to poultry. It should be noted that chickens are selective in what they eat and would preferentially eat any pieces of meat (potentially) remaining in the compost and therefore assuming that poultry randomly eat compost/soil may not be appropriate. This contrasts with cattle and sheep which randomly ingest soil and compost through eating grass. In this respect, consideration should be given to the nature of any meat components in compost.

The risks posed to backyard poultry from NDV and AIV in compost produced from catering waste (according to the Animal By-Product Regulations (2011)) are likely to be insignificant compared to the risks from other routes. In particular, there are routes through which backyard poultry could be exposed to raw poultry meat. Thus, on the backyard poultry owners' premises, unintentional exposure of poultry to catering waste could occur through access to a "home" compost heap containing kitchen waste or even to bin bags. Intentional feeding of raw meat to backyard poultry (although illegal) does occur in GB, although to what extent, is not known. Some idea can be gleaned from internet chat rooms. It is therefore difficult to directly compare the risks through compost produced under the Animal By-Product Regulations (2011) with the risks to poultry from other routes of indirect or direct contact with raw poultry meat.

There are no data available on the amount of poultry meat which is infected with NDV and which enters the food chain in GB. A "What If" analysis is performed to test the impact of 10,000 NDV-infected chicken carcasses entering the GB food chain in a year. It should be noted that 10,000 chicken carcasses is about 15 tonnes of infected poultry meat per year which is considered unrealistically worst case. The predicted risk of NDV to an individual chicken living for a year on land to which compost has been applied is low at 0.5×10^{-6} per chicken per year without a "grazing" ban and for undiluted surface applied compost. The relatively slow decay rate used for NDV in soil (at temperatures of 3 to 6°C) equates to a 1-log decay in 80 days. This means that the implementation of a three week ban on chicken grazing has relatively little impact, roughly halving the annual risk. Enforcing a grazing ban for non-commercial backyard poultry flocks may be difficult. A two month ban reduces the risk fivefold at temperatures of 3°C to 6°C.

The HPAI virus is enveloped and will be rapidly inactivated by the heat of the composting process. Sufficient data are available to perform a quantitative risk assessment for H5N1 HPAI virus. As an extreme worst-case, it is assumed that 10,000 H5N1-infected ducks, albeit clinically-normal, enter the food chain in GB each year. This represents 20 tonnes of

infected duck meat which is unrealistic. (Compared to "infected but clinically-normal" ducks, sick ducks have higher viral titres in the muscle, but would not be expected to be imported to GB because of export and import checks).

Assuming that the compost is surface applied and that there is no grazing restriction, an individual risk of H5N1 of 0.57 x 10^{-7} per chicken per year is calculated. This provides some reassurance that the risks to backyard chickens are low. Chicken meat has higher H5N1 titres than duck meat. However, chickens are rapidly killed by H5N1 and it is assumed here that meat from dead chickens could not be legally imported into GB (because of inspection at abattoirs in the EU). An assessment of the risks from legal importation of chicken meat has therefore not been undertaken here. Illegal imports of H5N1-infected meat are likely to be insignificant compared with the 20 tonnes used here for legal meat. By comparison, estimates of the amounts of pig meat infected with exotic pig viruses and illegally imported into GB are in the order of a few hundred kilograms per year. Amounts of illegally imported poultry meat infected with AIV and NDV would presumably be of similar magnitude, although no quantitative risk estimates are available for poultry meat.

BSE

Using the 95th percentile estimate of 260 bovine oral ID_{50} units exiting the abattoir to enter the food chain per year (APHA BSE Control Model version 5), the risk assessment predicts an individual risk of 0.8 x 10^{-6} per bovine per year. This is based on surface applied compost, with cattle consuming only compost and no soil every day for a year. This individual risk is over 100-fold lower than that predicted in 2001 for BSE in cattle through sewage sludge applied to grazing land (Gale and Stanfield 2001). Nationally it is estimated that there would be one BSE infected animal every 7 years through surface-applied compost. This is based on the assumption that *cattle eat 1.9% of all the food waste compost produced in GB, which is a worst-case assumption.*

The number of confirmed cases in GB in 2002, for which the source term for the risk assessment was estimated, was 445 (Defra 2012). Thus one additional infection every 7 years represents a very small proportion. It is concluded here that the risks of transmission of BSE to cattle in GB through application of composted catering waste to land are insignificant even using worst case assumptions.

The mean estimate of the amount of BSE infectivity going into the food chain each year is 2.6-fold lower than the 95th percentile used here and would predict one BSE infection every 18 years for surface applied compost. With tilling in to the soil to a depth of 10 cm, the model predicts one BSE infected animal in GB every 511 years (using the 95th percentile for the amount of BSE infectivity entering the food chain). These calculations assume that there is no reduction in BSE prevalence from that observed in the 2002 cohort and do not consider the on-going reduction in BSE prevalence that has been observed since 2002. Since 1992 the number of confirmed cases of BSE in GB has declined from 36,680 to zero in 2010 and 2011. The current risks through compost would therefore be negligible.

Scrapie

A risk assessment developed at the APHA estimated 95th percentiles for the number of sheep oral ID_{50} units of classical and atypical scrapie entering the food chain each year. It should be noted that ID_{50}s for atypical scrapie in brain and spinal cord are not available, and the value used is assumed to be the same as for classical scrapie. The assessment accommodated the SRM controls in sheep. Assuming 5.89% of sheep meat (cooked and raw) and 100% of sheep spinal cord are discarded in kitchens, it is estimated that 14,806 and 1,816 sheep oral ID_{50} units for classical and atypical scrapie, respectively, enter composting systems per year.

Assuming compost inactivates 80% of the scrapie agent, the risk assessment predicts individual risk of 1.34×10^{-4} and 1.65×10^{-5} per sheep per year for classical and atypical scrapie, respectively. This risk is calculated for surface applied compost, with sheep consuming only compost and no soil every day for a year. Nationally it is estimated that there would be 14.7 classical scrapie infections and 9.6 atypical scrapie infections per year in GB. These calculations are based on an estimated 580,875 sheep grazing for a whole year on land to which 58.2% of all the catering waste-derived compost has been applied. It is assumed that in total, those sheep eat 3.74% of the total compost produced annually from catering waste. With tilling in to the soil to a depth of 10 cm, the model predicts 0.195 classical scrapie infections and 0.13 atypical scrapie infections in GB every year, i.e. 0.325 scrapie infections in total.

Compared to the 67,619 sheep infected with scrapie (classical and atypical) estimated annually to be going to the food chain in GB, the extra 0.325 infections predicted from tilled-compost is insignificant.

The risk assessment assumes that sheep eat 0.69 kg of soil per day every day for a period of one year. A recent risk assessment for Defra (Adkin and Kosmider 2011) assumed that sheep eat a mean of 0.0937 kg soil per day. Using this more realistic value the predicted number of classical and atypical scrapie infections through compost tilled to 10 cm depth in the soil was 0.027 and 0.017 per year, respectively. For surface applied compost this gave 2.0 and 1.3 classical and atypical scrapie infections, respectively, per year.

The baseline risk assessment assumes composting removes 0.69-log of scrapie infectivity. Recent experiments suggest composting and mesophilic anaerobic digestion, in particular, may remove much larger proportions of scrapie infectivity (Section 8.7.4). The sensitivity analysis (Section 9.10) predicts just 0.006 scrapie (classical and atypical) infections per year if the 2.43-log removed reported by mesophilic anaerobic digestion is used in the risk assessment for removal by composting. Using data for thermophilic conditions would reduce the risks a further 10-fold according to published data, although this would be limited to 2.7-log by the modelled 0.2% by-pass (Figure 10). Thus, composting may be a major barrier for scrapie.

It is important to note that scrapie is not a human pathogen, and unlike BSE, it is known from epidemiological evidence to be transmitted between sheep and through the environment. The extra scrapie infections predicted through compost are insignificant.

Endemic faecal-oral bacteria

Due to the lack of dose-response data for faecal-oral bacterial pathogens in livestock, full quantitative risk assessments could not be performed. The approach therefore was to compare predicted loadings of *E. coli* O157 in compost with those predicted in stored manures (3 months storage) and primary MAD-treated sewage sludge.

Assuming as a worst-case that 4-log regrowth of *E. coli* O157 occurred in meat awaiting composting, it was estimated that the *E. coli* O157 total loadings were 6.7-fold lower than those in MAD-treated sewage sludge, and some 4,335–fold lower than for manures stored for 3 months. It is concluded that even allowing for regrowth in compost, the total loadings of faecal pathogens such as salmonellas, *E. coli* O157 and campylobacters present in composted catering waste do not exceed those of stored manure, which is currently used on 78% of farms.

There is recent evidence that *E. coli* O157 may lose virulence genes during manure storage, so presumably the same could happen during composting. Campylobacters require unusual conditions for growth and would not grow in raw meat or in the environment (Corry and

Atabay 2001). Overall it is concluded that the exposures to livestock of faecal-oral bacteria through composted catering waste are low compared to stored manures.

Porcine parvoviruses

Pigs in acute phases of infection shed the virus in faeces (Mengeling 1986), which could contaminate the meat and high levels of viral DNA have been detected in heart, liver and kidneys of infected piglets (Wilhelm *et al.* 2005). Thus, PPV is likely to be present in pig meat, although lack of information on loadings of infectivity prevents a quantitative risk assessment being undertaken here. However, significant inactivation (>4.0-log) of PPV occurs over the time scales of composting (48 hours) at temperatures of 55°C to 60°C.

Composting according to the Animal By-Product Regulations (2011) should greatly reduce the levels of PPV in catering waste. Belschner and Love (1984) write that the main ways in which porcine parvovirus can be introduced are through pigs that are actively infected and excreting the virus and by faecal contamination of introduced pigs, clothing or boots of personnel. The inactivation of PPV by composting should minimise the risks from pig meat in catering waste-derived compost compared to these routes.

While the risks through compost are lower than through other routes, it cannot at this stage be demonstrated that the risks are negligible. The magnitude of the risk would reflect loadings of virus in pig meat in GB. A quantitative risk assessment cannot be undertaken at this stage because of lack of information on loadings of PPV infectivity in meat.

Porcine circoviruses

There is clear evidence that PCV2 is present in pig tissues including muscle, bone marrow and mammary gland, and pig milk. Indeed Opriessnig *et al.* (2009) have demonstrated that uncooked pig meat products (including bone and skeletal muscle) can infect naïve pigs through the oral route. The temperature inactivation data available for PCV2 suggest that composting (60°C for 48 h) could achieve some reduction in the risks from any PCV2 in raw pig meat, and could (with extrapolation of 1.33 log in 24 h at 60°C) achieve 2.66-log reduction over 48 hour periods. The time-temperature combination of 60°C for 48 h achieves >3.0-log reduction for porcine parvovirus and indeed, there is some evidence that PCV2 is inactivated more rapidly than parvoviruses (albeit at 70°C).

Although the risks of transmission of PCV2 to pigs through composted catering waste may be low relative to other routes, it cannot at this stage be demonstrated that the risks are negligible. The exact role of PCV2 in post-weaning multisystemic wasting syndrome (PMWS) is not clear, and a risk assessment for PMWS is not yet feasible. Pasteurisation at 70°C for 1 h may achieve 1.5 log reduction which is similar to the 1.33 log reduction achieved by 24 h at 60°C.

Clostridium botulinum

The risk of botulinum intoxication to cattle through compost would be low because composting, unlike silage production, is an aerobic process such that regrowth of any *Clostridium botulinum* bacteria present in the meat should not occur during composting. Also, at temperatures of 56°C – 60°C, any bacteria germinating from spores would be inactivated. Furthermore, any botulinum toxin is likely to be diluted during the composting and soil application processes, such that cattle exposures may be below the threshold dose, although this has not been formally assessed here.

It is concluded that the risks to grazing livestock from *C. botulinum* in compost which has been tilled into a depth of 10 cm are low. However, it cannot be ruled out that spores present in compost could not multiply up in anaerobic silages produced from grass.

Methicillin-resistant Staphylococcus aureus (MRSA)

Although *Staph. aureus*, may be more temperature resistant than *Salmonella enterica* serotypes, available evidence suggests that composting for 60°C for 2 x 48 hours (as set out in the Animal By-Products Regulations 2011) would achieve log reductions in *Staph. aureus* comparable to those of salmonellas even allowing for by-pass of the two step composting process. The risk of colonisation of pigs through contact with scraps of MRSA-infected pig skin after composting of catering waste is likely to be remote in relation to the risks from direct contact between pigs during sales. This is because singeing and scalding during processing of the pig carcass together with destruction of the *Staph. aureus* during composting will greatly reduce any risks of MRSA to pigs in composted catering waste. It is concluded that further consideration of the risks of MRSA through compost is not required.

Risks of abortion from Toxoplasma gondii infection in sheep

There are no dose-response data for *Toxoplasma gondii* that offer a clear improvement on the dose-response approach used in the original 2002 risk assessment. In that report, a dose-response curve was used for the risk of abortion in pregnant ewes. Data from a recent study in the UK suggest that less than 0.1% of UK cat litter is wrongly disposed of in catering wastes. Assuming a worst-case that 1% of UK cat litter contaminates catering waste, then with a 3 week grazing ban and tilling to 10 cm, catering waste compost would contribute 0.4 abortions from *T. gondii* infection in sheep in the UK per year. This is 0.11% of the 375 abortion cases confirmed in 2007 in sheep. For surface applied compost with no grazing ban the individual risk of 8.9×10^{-5} per sheep per year translates into 53 still births per year in the UK. This is worst-case in assuming all those sheep grazing on land with compost are not only pregnant all year, but also ingest compost to the exclusion of soil for a year. It should be noted that although 375 cases of *T. gondii* abortion were confirmed in 2007 in sheep in GB, many cases are unreported. In the majority of sheep flocks there will almost always be a low level of abortion with 3% to 4% losses being accepted without investigation.

Conclusions

- The results of this revised risk assessment support the Animal By-Products Regulations (2011), and there is no need to tighten the composting parameters for catering waste or to increase the length of the current grazing bans.
- There are no new data which suggest the time-temperature combination and particle size recommendations need amending. However, the original risk assessment allowed for pathogen dilution in the soil through leaching to a depth of 10 cm. In this respect, minimum tillage on agricultural land would further minimise (relative to surface-applied compost) any residual risks from BSE, *T. gondii* and *C. botulinum*. Indeed, if compost is tilled in to a depth of 10 cm, then the grazing ban for pigs could be reduced from 2 months (currently) to 3 weeks as currently for cattle and sheep.
- Composting process by-pass significantly increases any risks and must be minimized by ensuring the compost process operators continue to adopt and implement robust HACCP plans.
- Although outside of the scope of this risk assessment, the complete prevention of direct feeding of catering waste to livestock is extremely important.
- Predicted risks to individual livestock from FMDV, ASFV and SVDV are negligible at $<10^{-9}$ per animal per year, even with no grazing ban and for surface application of compost (such that livestock ingest compost with no soil for a year).
- Risks of BSE transmission to cattle through composted catering waste are remote.
- Compared to the 67,619 scrapie-infected sheep (classical and atypical) estimated to be entering the food chain annually in GB, the extra infections (classical and atypical combined) per year predicted from compost are insignificant.

- For surface applied compost, it is recommended that the grazing ban remain 2 months for pigs, as the model predicts one case of CSF every 48 years with just a 3 week grazing ban, reducing to one case every 4,110 years with a 2 month grazing ban.
- Although parvoviruses and circoviruses are more resistant to heat inactivation processes than the other viruses considered here, there is evidence to suggest that significant inactivation (>3-log for parvovirus, and perhaps up to 2.6-log for porcine circovirus) may be achieved over 48 h at 60°C. The second composting step would give additional inactivation. Although the risks of transmission of PPV and PCV2 to pigs through composted catering waste may be low relative to other routes, it cannot at this stage be demonstrated that the risks are negligible. This is because quantitative risk assessments cannot be undertaken at this stage due to lack of information.
- Avian viruses, namely AIV and NDV, are enveloped and are inactivated by heat. Commercial free-range and housed poultry are unlikely to be exposed to compost. The risks posed to backyard poultry from NDV and AIV in compost are likely to be insignificant compared to the risks from other routes.
- Even allowing for regrowth in the meat and compost, the levels of pathogens such as salmonellas, *E. coli* O157 and campylobacters present in composted catering waste would be well below those of stored manure, which is currently used on 78% of farms.
- Predicted risks of abortion in pregnant sheep from *Toxoplasma gondii* from cat litter contamination of kitchen waste are very low compared to reported abortion rates in the UK.
- The risks to grazing livestock from *Clostridium botulinum* in compost which has been tilled into a depth of 10 cm are low.
- It is concluded that further consideration of the risks of MRSA through compost is not required.

17.0 References

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