RISK ASSESSMENT: USE OF COMPOSTING AND BIOGAS TREATMENT TO DISPOSE OF CATERING WASTE CONTAINING MEAT

Final Report to the Department for Environment, Food and Rural Affairs

Risk Assessment: Use of Composting and Biogas Treatment to Dispose of Catering Waste Containing Meat

Final Report to the Department for Environment, Food and Rural Affairs

Report No: DEFRA

May 2002

Author: Dr Paul Gale

Contract Manager: Dr Paul Gale

Contract No: 12842-0

DEFRA Reference No:

Contract Duration: October 2001 to May 2002

This report has the following distribution:
DEFRA – 10 copies
Contract Manager – 2 copies

Any enquiries relating to this report should be referred to the Contract Manager at the following address:

WRc-NSF Ltd, Henley Road, Medmenham, Marlow, Buckinghamshire SL7 2HD.
Telephone: + 44 (0) 1491 636500 Fax: + 44 (0) 1491 636501

CONTENTS PAGE

Page

LIST OF TABLES

LIST OF FIGURES

Glossary

"**Biogas plant**" means a plant in which biological degradation of products of animal origin is undertaken under anaerobic conditions for the production and collection of biogas.

"**Catering waste**" means all waste food originating in restaurants, catering facilities and kitchens, including central kitchens and household kitchens. In terms of waste types, catering waste would includes B – K as set out below. B would be classified as catering, because it *could* also contain food.

A	Civic Amenity Site Greenwaste
B	Source separated garden waste
C	Source-separated plant (fruit; veg) uncooked
D	Source-separated plant (fruit; veg) cooked
E	Source-separated meat uncooked
F	Source-separated meat cooked
G	Catering establishments
H	Commercial kitchens
I	Mechanically-separated organics from mixed household waste (MSW.)
J	Waste from food retailers
K	Mixed household waste (MSW)

"**Composting plant**" means a plant in which biological degradation of products of animal origin is undertaken under aerobic conditions.

"**Enclosed**" in this report means vermin and bird-proof (as from the point of pathogens) and does not refer to gas exchanges (odours).

Greenwaste includes A and sometimes B and C in addition.

"**Meat fraction**" – waste fraction for composting which contains meat derived from two sources, namely:-

1. waste stream containing the meat which has been separated at source by the waste producer from the residual stream; and

2. residual black bag waste which has not been separated at source and will include meat as well as other waste materials.

"**Non-meat fraction**" – waste fraction for composting which should be free of most of the meat because waste producer has been instructed to exclude meat by source separation.

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

"**Turning**" – the process of redistributing the material comprising a windrow – to be defined by a standard operating procedure (SOP).

"**Maturation**" – the mesophilic phase of composting which follows the thermophilic phase characterised by establishment of natural fungi.

"**Windrow**" – trapezoidal heaps.

EID_{50} – egg ID_{50} (for Newcastle disease)

ID_{50} – Dose which when given to each and every member of a population infections half of the members of that population.

IDU, infectious dose unit (a hypothetical term)

$TCID_{50}$ – Tissue culture ID_{50} – measure of virus titre

Pfu, plaque-forming unit (measure of virus titre)

BSE, bovne spongiform encephalopathy;

TSE, transmissible spongiform encephalopathy;

ASF(V), African Swine Fever (Virus)

CSF(V), Classical Swine Fever (Virus)

FMD(V), Foot and Mouth Disease (Virus)

SVD(V), Swine Vesicular Disease (Virus)

MGRT; minimum guaranteed retention time (biogas)

HRT; hydraulic retention time (biogas)

DRG, dorsal route ganglia

HSE, Health and Safety Executive

"Tds", tonnes dry solids

VTEC, Vero-toxigenic *Escherichia coli*

CNS, Central Nervous System

OTMS, Over Thirty Month Scheme (cattle)

SRM, Specified Risk Material,

MHS, Meat Hygiene Service,

EXECUTIVE SUMMARY

A proportion of meat from domestic households and catering establishments will be discarded uncooked with catering waste. A risk assessment is developed here to determine the risks to cattle, sheep, pigs and chickens of infection from pathogens potentially present in that meat after the catering waste has been composted and applied to land. In addition, the risks to humans utilising compost and consuming crops grown in those fields to which compost has been applied are considered. The risk assessment has focused on TSE agents, the exotic pig viruses, *E. coli* O157, campylobacters, salmonellas, Newcastle disease and parasites. The overall conclusion is that it is acceptable to apply composted catering waste to land provided: -

1. All steps are taken to eliminate any by-pass of the composting/biogas process, including ensuring that:-

- Raw catering waste material is not keep on livestock farms;

- Birds and small mammals do not gain access to the raw material;

- Raw material is delivered to a housed reception;

2. A two-barrier composting system is used for the “meat” fraction.

3. For each composting barrier, the catering waste reaches a temperature of 60°C for two days during composting, with the composting process being continued for at least 14 days;

4. The first treatment barrier be it “in-vessel” or windrow is housed or enclosed;

5. Windrows are turned at least three times;

6. “Dirty” end is kept separate from the “clean” end; i.e. different tools and equipment are used to handle the final product and the raw material;

7. Biogas is performed at 57°C; MGRT = 5 h; HRT = 19 days

8. The maximum particle size for composting is <40 cm diameter. This includes large joints of meat, e.g. discarded after freezer failures. For biogas, a maximum 5 cm (diameter) particle size is required;

9. Animals are not allowed to graze on land to which composted catering waste has been applied for a period of 2 months.

The risk assessment approach

A risk assessment is developed with the Source – Pathway – Receptor approach. Environmental risk assessment is not concerned with the complete elimination of a pathogen by any one barrier, but relies on a multiple barriers approach. The barriers include exclusion of meat at source, the composting/biogas processes, stock-pilling/storage, decay in the soil, dilution in the soil, in addition to the fact that only a small proportion (model assumes 1%) of meat is discarded in the kitchen uncooked.

Source Terms

For BSE, scrapie and *E. coli* O157, the risks are calculated on the basis of the number of bovines and sheep slaughtered in the UK. For BSE, account is also taken of imported bovine carcasses and processed meat. For the purpose of BSE and scrapie it is assumed that 90% of dorsal route ganglia (DRG) and 100% of spinal cord in lamb chops, respectively, present in the food chain are discarded to catering waste. For *E. coli* O157 is it assumed that 0.01% (w/w) of meat sold in shops is cow/sheep faeces. It is interesting to note that the predicted *E. coli* O157 concentration is 1.4 cfu g^{-1} beef, which is in good agreement with the median concentration of 1.5 cfu g^{-1} of beef measured in burgers in an outbreak in Canada.

For campylobacter and salmonella, the risks are calculated on the basis of published data on bacterial loadings per chicken carcass and the fact that 615 million broilers are slaughtered annually in the UK.

For exotic agents, the Source Term is based on "What if?" scenarios. Thus for Trichinellae, CSF and FMD, the model addresses the question of "What if 10,000-infected pig carcasses entered the food chain each year?". Although unrealistically worst-case for illegally imported meat, such scenarios could occur during outbreaks of FMD and CSF. For SVD and ASF, the risks are based on 1,000-infected pig carcasses entering the food chain annually.

For *Clostridium botulinum*, the starting point is that 4.18% of bacon contains spores.

For *Toxoplasma gondii*, the Source Term is based on an estimate of faeces from 15,000 infected domestic cats being disposed of to "black-bag" waste through cat litter.

Composting process parameters

It is proposed here that the composting process (windrow or "in-vessel") should achieve a temperature of 60°C for two days. The literature data reviewed here demonstrate that at 55°C considerable destruction of FMDV, CSFV and ASFV occurs within 10 – 15 minutes. Furthermore aeration processes at 50°C give complete inactivation over 48 h. However, for SVDV in one slurry experiment a temperatures of 56°C was required for 2 hours to achieve a 3-log reduction. The "60°C for 2 days" is chosen because it takes 40 h for a sphere of diameter 40 cm to reach a temperature of 56°C at the centre when the surrounding temperature is 60°C. This is analogous to a large leg of pork with the bone in. The bone marrow contains high CSFV loadings in a CSF-infected pig carcass and would take 40 h to reach 56°C if the outside temperature is maintained at 60°C.

It is shown here that a temperature of 70°C for 1 hr according to the EU Regulations is only acceptable if the particle size is <6 cm in diameter. Indeed the EU Regulations specify <12 mm. It is the 2 day time period of composting proposed here which eliminates the requirement for a 12 mm minimum particle size at 60°C. This is important because such a small particle sizes would tend to impair the aerobic processes of composting.

Biogas processing parameters

For biogas, the particle size must be less than 5 cm (diameter). The thermophilic biogas system must achieve a digestion temperature of 57°C with MGRT of 5 h and HRT of

>19 d. When operated correctly removals of 4 – 6 logs have been demonstrated at operational scale. Mesophilic digestion (36 – 38°C) is not appropriate for treatment of catering waste containing meat.

By-pass of the composting process (or biogas process)

Of critical importance is the degree of by-pass of the processes. Indeed, the net pathogen destructions achievable by any process are ultimately limited by the degree of by-pass. By-pass forms the basis of estimating the net pathogen destruction by composting/biogas in this risk assessment. There is some quantitative information on by-pass for thermophilic biogas systems albeit under conditions of operational failure. By-pass of the 60°C/2 day conditions for windrows can be modelled mathematically according to the number of turns. It is calculated here that at least three turns of a windrow are required to ensure <0.2% of the raw material remains in the "cold" part. For "in-vessel" composting, the requirement is that >99.8% of the raw material achieves 60°C for two days. (A 99.8% destruction, or 0.2% survival, is equivalent to a 2.7-log reduction as set out in Table 1). The biogas plant must be designed and operated to ensure <0.02% by-pass of untreated material. This is to give a net 3.7-log (5,000-fold) destruction of pathogens by biogas (Table 2) and can be achieved at operational scale.

Stock-piling of compost/Storage of biogas product

The storage period recommended is 18 days. This is to allow for a 1-log decay of CSFV. During this process for composts, there will be some heat generation and greater destructions than just 1-log would be anticipated in practice.

A credit system for modelling the barriers

The credit system is based on log removals. Thus a 1-log removal is a ten-fold destruction, a 2-log removal represents 100-fold destruction, 2.7 logs is 500-fold, 3.7-logs is 5,000-fold.

Source separation

Source separation is the actions 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 the waste fraction for composting which should be free of most of the meat because waste producer has been instructed to exclude meat by source separation.

The Meat fraction is the waste fraction for composting which contains meat derived from two sources, namely:-

1. waste stream containing the meat which has been separated at source by the waste producer from the residual stream; and

2. residual black bag waste which has not been separated at source and will include meat as well as other waste materials.

Composting of "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 Meat Exclusion at Source, Composting/Biogas and Stock-piling. Meat Exclusion at Source is assumed to be 90% efficient, i.e. source-separate "non-meat" waste contains 10% of the total uncooked meat waste. The barriers are set out in Table 1 for composting and Table 2 for biogas.

Table 1 A credit system for the barriers for composting "non-meat" waste.

Process (Barrier)	Credits (log-reduction)
Meat Exclusion at Source	1.0
Composting process*	2.7
Stock-piling (18 days)	1.0
Total	4.7

*Windrow or "in-vessel"

Table 2 A credit system for the barriers for biogas treatment of "non-meat" waste.

Process (Barrier)	Credits (log-reduction)
Meat Exclusion at Source	1.0
Biogas	3.7
Total	4.7

Composting the "meat" fraction

Waste containing meat must be composted by a two barrier process. First an "In-vessel" process in which <0.2% fails to reach 60°C for 2 days, and secondly a windrow. The windrow need not be housed as it is a secondary barrier. The barriers are set out in Table 3.

Table 3 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

Biogas treatment of the "meat" fraction

A two barrier process comprising biogas digestion and a storage is set out in Table 4. A storage stage of the finished product of 18 d is required to allow for a 1-log decay of CSFV.

Table 4 A credit system for the barriers for biogas treatment of the "meat" fraction.

Process (Barrier)	Credits (log-reduction)
Meat Exclusion at Source	0.0
Biogas	3.7
Storage (18 days)	1.0
Total	4.7

Putting these barriers together

The composting processes outlined in Table 1 and Table 3 are put together in a single event tree in Figure 1 to model the overall reduction of infectivity in meat in catering waste by composting. In total 2.36 x 10^{-5} infectious dose units (IDU) from each IDU in the catering waste remain in the compost. This is equivalent to a 4.62-log reduction overall.

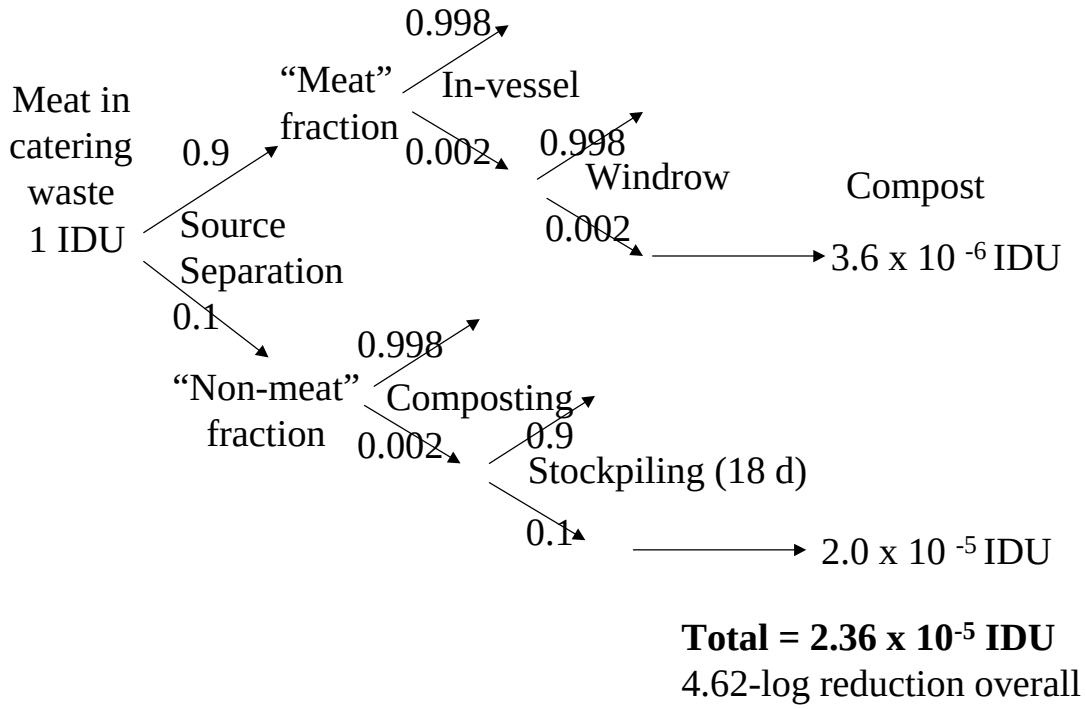

Figure 1 Composting as set out in Tables 1 and 3 achieves a 4.62-log reduction of infectivity in catering waste.

Similarly for biogas, combining the processes set out in Tables 2 and 4 into an event tree (Figure 2) shows that overall the net reduction of infectivity in catering waste is 4.42-logs.

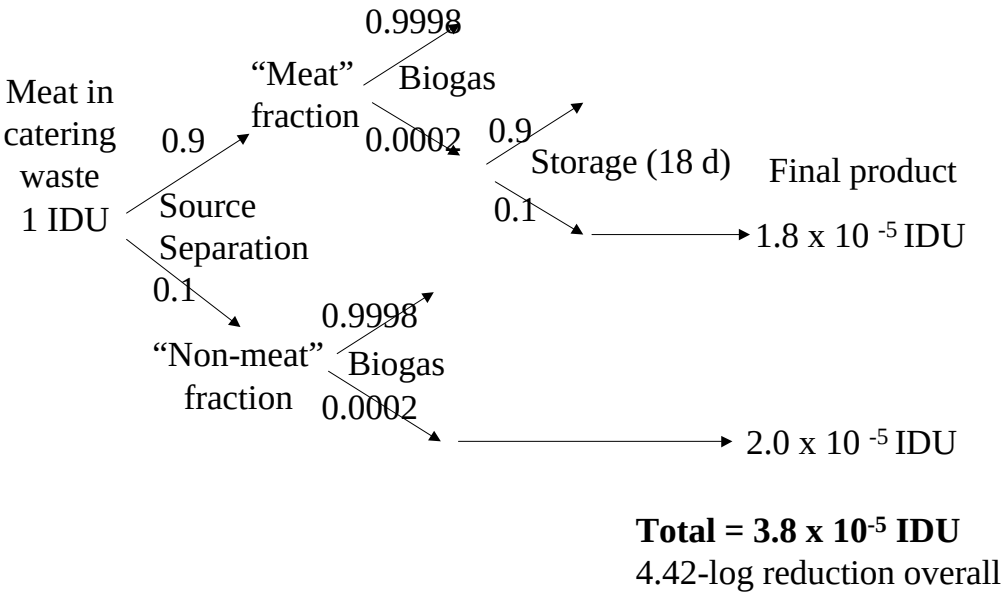

Figure 2 Biogas treatment as set out in Tables 2 and 4 achieves a 4.42-log reduction of infectivity in catering waste.

Modelling the 2 month no grazing ban

For FMDV, CSFV and ASFV the model allows for a 5-log decay with time as specified by available data. This is shown for FMDV in Figure 3. The risk to grazing cattle (ingesting 0.41 kg soil cow^{-1} d^{-1}) is calculated from the cumulative exposure between days 61 and 426.

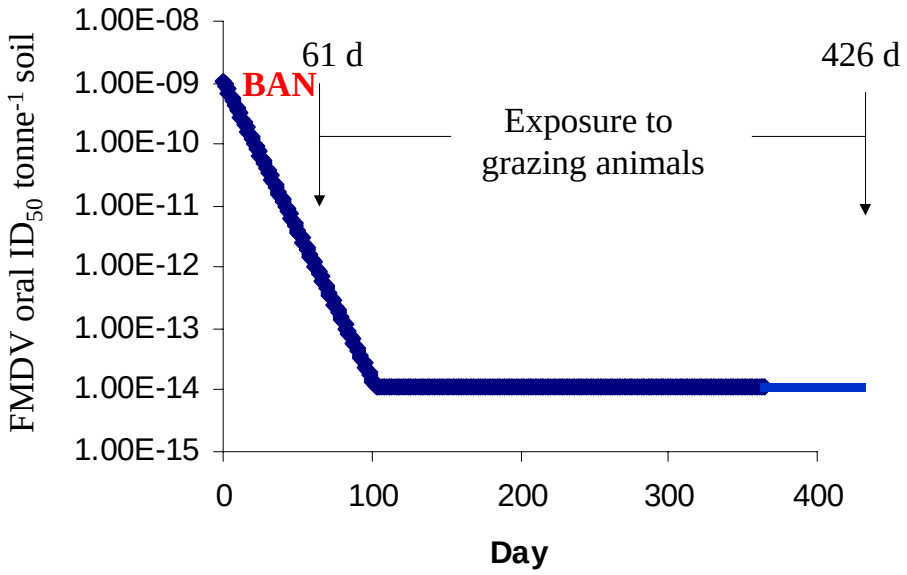

Figure 3 Modelling annual exposure of grazing animals and the effect of the 2 month no grazing ban

Summary of the predicted risks

The results of the risk assessment are presented in Table 5. It should be noted that the annual numbers of animal infections are predicted on the assumption that 0.52% of the England/Wales herd/flock are housed or graze on land to which compost has been applied.

Table 5 Summary of risks for each pathogen from composting of "non-meat" fraction – assumes 4.7-log credits by Source Separation and composting according to Table 1. To model for composting of all meat (as set out in Figure 1) multiply risks by a factor of 1.2. To model for biogas treatment of all meat (as set out in Figure 2) multiply risks by a factor of 1.9.

Pathogen	Quantitative Risk – *additional number of cases per year assuming 0.52% of farm animals graze on land to which compost has been applied	Qualitative assessment of risks
Toxoplasma gondii	<1 additional pregnant ewes abort per year (from cat faeces in MSW)	The risks to sheep and goats from *Toxoplasma gondii* from application of composted catering waste to land are considered low in the light of the risks posed by farm yard cats, which may defecate directly in the feed.
***Scrapie**	0.14 additional cases in sheep year^{-1}	Assumes composting and stock-piling has no effect on scrapie prion
***BSE**	Remote risks:-0.0034 cases of BSE in cattle year^{-1} in England/Wales. Risk to humans eating unwashed crops of 0.7 x 10^{-10} person^{-1} year^{-1}	Dorsal Root Ganglia (DRG) are main source of infectivity in catering waste – assumes composting and composting has no effect on BSE agent
***FMD**	Very low risks to grazing animals; Stock-piling and 2 month grazing ban important during FMD outbreak	Low infectivity of virus through oral route, inactivation in muscle, and removal of bone serve to reduce risk
***CSF**	Assuming 10,000 "bone-in" CSF infected carcasses imported per year, model predicts 0.0032 cases year^{-1}	Virus is highly infectious through oral route; huge titres in bone marrow of infected pigs. Stock-piling barrier is important. Enforcement of 2 month grazing ban is crucial, particularly during an outbreak of CSF.
***ASF**	Assuming 1,000 "bone-in" ASF-infected carcasses imported per year, model predicts very low risks to pigs; need to consider exposure through skin.	Lower risk of importation than FMD and CSF – Less decay on land than FMD and CSF; more infectious than FMD, removal of bones greatly reduce risks; Enforcement of 1 year grazing ban is important.
***SVD**	Remote risk through oral	Low risk of importation

Pathogen	Quantitative Risk – *additional number of cases per year assuming 0.52% of farm animals graze on land to which compost has been applied	Qualitative assessment of risks
	route; low risk even through skin exposure	Relatively low levels of infectivity in pork: Note model assumes no decay in the soil
Newcastle Disease	Model assumes 100,000 ND-infected chickens enter food chain annually. Predicted risks to chickens are at least an order of magnitude lower than the risks to cattle, sheep and pigs calculated for FMDV	
Trichinella spiralis	Very low risk to humans, even from direct ingestion of compost. Using very worst case assumptions, the model predicts that for every 10,000 *T. spiralis*-infected porcine carcasses entering the food chain (illegally) there will be one case in pigs from composted catering waste	Meat inspection very effective; hence very low incidence in meat in UK, life cycle does not appear to include environmental stage.
Campylobacter	Very low risks; 7.5×10^{-8} person^{-1} year^{-1} for consumption of unwashed and uncooked root crops; The risks to a gardener ingesting a gram of compost are $<10^{-6}$ person g^{-1} ingested.	Higher levels and prevalence in poultry and more infectious to humans than salmonella, but no growth in waste meat.
***E. coli* O157**	Loading on land in England/Wales from composted catering waste is >5,000-fold lower than for manures and >40-fold lower than for treated sewage sludge. Risk to a gardener ingesting 1 g of compost could be as high as 0.5×10^{-4} person^{-1} g^{-1}.	*E. coli* O157 in meat products, mainly beef, is not a rare contaminant. However, ID_{50} may be high in both humans and cattle. Model allows for 4-log growth of *E. coli* O157 in catering waste
Salmonellas	The risks from salmonella	Model assumes chicken carcasses

Pathogen	Quantitative Risk – *additional number of cases per year assuming 0.52% of farm animals graze on land to which compost has been applied	Qualitative assessment of risks
	on soil through application of compost containing catering waste are at least 1,000-fold lower than from application of conventionally-treated sewage sludge. The risks to a gardener ingesting a gram of compost are in the order of 10^{-6} person g^{-1} ingested – but assumes no growth in the compost.	bought at retail have been scalded. Salmonella levels on chickens at retail are low. A major source of uncertainty is the degree of growth in the meat after being discarded to the catering waste – the model allows for 6-log growth on the meat. No allowance is made for regrowth in the compost.
***C. botulinum* spores**	Recommendation that warning label is put on compost sold for home use to ensure infants under 6 months are kept away.	Infants under 6 months of age highly susceptible to *C. botulinum* spores; composting/biogas will not inactivate the spores, may multiply in the meat in the catering waste. Some 4% of vacuum-packed bacon estimated to be positive for spores. "Estimated" spore loading in compost lower than for some soils in The Netherlands.
***C. botulinum* toxin**	No risk	Toxin may well be generated in rotting meat discarded to waste, but the composting process will inactivate the protein to some degree, and dispersion of the toxin molecules should ensure any exposures are below the threshold dose
Plant pathogens	Risks from specific pathogens have not been addressed.	Further consideration will be given; controls in existing guidance on composting should be followed.

Could composted catering waste spread endemic infections to uninfected animal herds?

The question of whether the application of composted catering waste to agricultural land could spread the infection to uninfected herds and flocks is not tackled directly. Instead the risks relative to those from spreading of sewage sludge and manures are compared. Predicted concentrations of salmonellas in soil from application of compost are at least 1,000-fold lower than those predicted for application of conventionally-treated sewage sludge. This puts the risks of spread of infection to uninfected herds into perspective. For *E. coli* O157 and salmonellas, quantitative risk assessment is complicated by the uncertainty in the potential for regrowth, both in the raw catering waste and in the composted product. In the case of campylobacters, no

growth will occur on the meat or in the compost. This, together with the barriers, namely composting, decay and dilution in the soil, would mean that the application of composted catering waste to land would be a minor pathway for campylobacter compared to other routes.

Animal Health - Classical Swine Fever Virus – why both a 2 month ban and multiple composting barriers are important.

The model assumes that 10,000 CSF-infected pig carcasses enter the food chain in a year. Although unrealistically worst-case for illegally imported material, it is not inconceivable that such challenges could occur during an outbreak of CSF in the UK. An acceptable risk would be <1 new CSF case every 50 years, i.e. <0.02 pigs infected year^{-1}.

Composting System

Table 6 demonstrates the importance of both the second barrier (i.e. windrow for "meat" fraction) and the 2 month no gazing ban for composting systems.

Table 6 Predicted risks of CSF to pigs in UK from application of compost-treated meat to land.

Composting as in Figure1	Grazing ban status	Risk pig^{-1} y^{-1}	CSF cases* (pigs y^{-1})
With second barrier, i.e, 4.62-log reduction	2 month ban	1.6 x 10^{-7}	0.005
Without second barrier, i.e. 2.70-log reduction	2 month ban	1.3 x 10^{-5}	0.43
With second barrier, i.e, 4.62-log reduction	no ban	1.8 x 10^{-4}	5.9
Without second barrier, i.e. 2.70-log reduction	no ban	0.015	489

*assumes 0.52% of UK pigs are housed on land to which compost applied

Biogas system

The predicted risks to pigs from CSFV are set out in Table 7. In the case of biogas, both the 2 month no grazing ban and the additional 18 d storage for "meat" fraction are required.

Table 7 Predicted risks of CSF to pigs in UK from application of biogas-treated meat to land.

Biogas as in Figure 2	Grazing ban status	Risk pig^{-1} y^{-1}	CSF cases* (pigs y^{-1})
With 18 d storage, i.e, 4.42-log reduction	2 month ban	2.5 x 10^{-7}	0.008
Without 18 d storage, i.e. 3.698-log reduction	2 month ban	1.3 x 10^{-6}	0.043
With 18 d storage, i.e. 4.42-log reduction	no ban	2.8 x 10^{-4}	9.3
Without 18 d storage, i.e. 3.698-log reduction	no ban	1.5 x 10^{-3}	49

*assumes 0.52% of UK pigs are housed on land to which compost applied

The extra 18 d storage stage only reduces the risk by 5.4-fold. However, it is a back-up barrier and reduces the frequency of a CSF case from once every 23 years to once every 125 years.

Public health – salmonellas, *E. coli* O157, and campylobacters – the case for two barrier composting/biogas

Table 8 compares the predicted risks to humans from ingestion of a gram of compost producted from feedstock containing meat. In the case of campylobacters and salmonellas, two stage composting and biogas with storage is important to bring the risks down to the order of 10^{-6} person^{-1} year^{-1}, which is an accepatable risk according to the HSE.

Table 8 Predicted individual risks (infections gram^{-1}) to humans ingesting 1 g of compost produced from material containing meat. Note it is assumed that 18 d stock-piling (Figure 1) and 18 d storage (Figure 2) give a 1-log reduction of these pathogens.

Process	Net removal (logs)	Campylobacter	Salmonella	*E. coli* O157*
Two barrier composting (Figure 1)	4.62	0.5 x 10^{-6}	1.3 x 10^{-6}	1.5 x 10^{-7} (0.6 x 10^{-4})
One barrier composting (Figure 1 minus 2nd windrow)	2.70	0.4 x 10^{-4}	1.1 x 10^{-4}	1.3 x 10^{-5} (0.035)
Biogas/stoarge (Figure 2)	4.42	0.8 x 10^{-6}	2.0 x 10^{-6}	2.4 x 10^{-7} (1.0 x 10^{-4})
Biogas with no storage	3.70	0.4 x 10^{-5}	1.1 x 10^{-5}	1.3 x 10^{-6} (0.0035)

*Risks calculated using two different dose-response models

Comparison with landfill routes

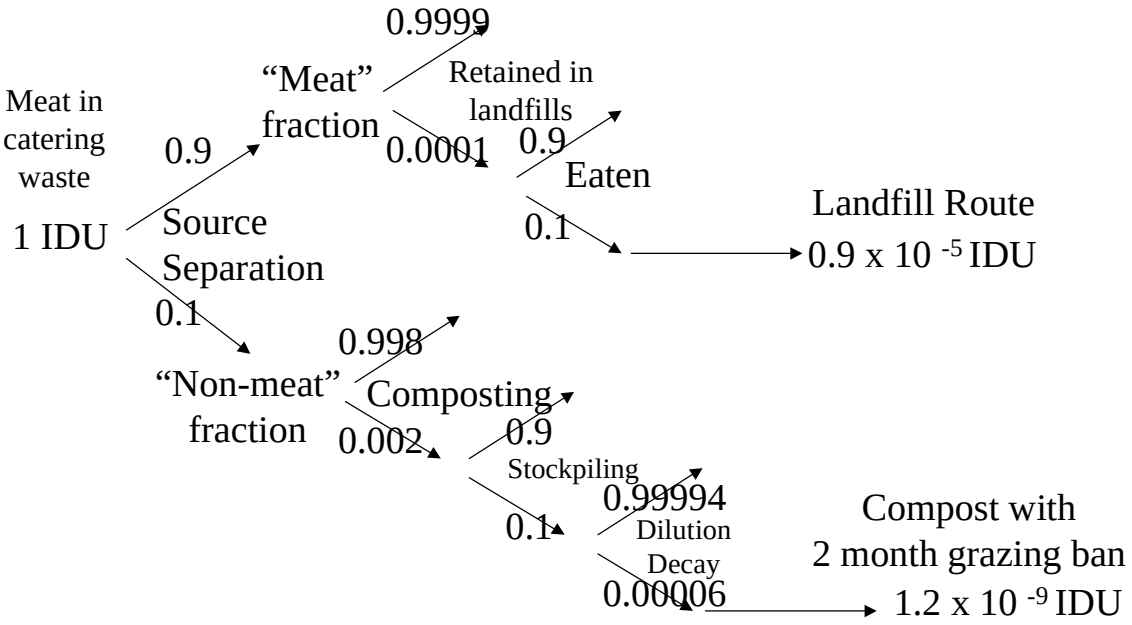

Figure 4. Prototype event tree for comparison of exposures to animals through landfill disposal and composting with a 2 month grazing ban. Note; 1) model assumes source separation, such that 90% of the meat goes to landfill; 2) no data are currently available for how much meat is removed from landfills by scavenging gulls.

The composting route for catering waste *potentially* presents lower risks to grazing animals than disposal through land-fill. A prototype model is set out in Figure 4. This is because composting offers extra control points. First, the raw catering waste will be delivered to enclosed receptions where birds and animals cannot gain access. Second, a no-grazing period can be enforced after application of the compost to land.

The significance of the ultimate "by-pass"

The illegal action of feeding raw catering waste directly to pigs would by-pass the 9-log process (set out in Figure 4) comprised of the multiple barriers of source separation/composting and dilution/decay in the soil (from the 2 month no grazing period). If just 0.01% of the meat in catering waste in the UK were applied direct to land (with no 2 month grazing ban), the risks of an FMD or CSF outbreak would increase by some 100,000-fold. This represents feeding catering waste direct to pigs and would in effect remove the benefits of applying the composting and biogas processes set out in Figures 1and 2, respectively. The net reduction in risks across the UK by implementing a multiple barriers composting process with a grazing ban would no longer be 9-logs but just 4-logs. The "take home" message is that if just a small proportion of catering waste was illegally fed directly to pigs, then there would be no point in the rest of UK composting the waste – simply tilling all catering waste produced in the UK into the land and applying the 2 month grazing ban would not present a higher risk.

Conclusions

1. The risks from prion diseases (BSE and scrapie) both to animals and humans are remote, even allowing for no destruction by composting/biogas/stock-piling and no decay in the soil.
2. It is concluded that the risks posed to animal health from the application of composted catering waste to land are acceptably low, providing the two barrier composting and biogas processes as set out in Figures 1 and 2, respectively, are used, and a 2 month no grazing ban is enforced.
3. The no grazing ban (2 month) and 2nd barriers in the composting/biogas processes are essential for CSF, and are also important during FMD outbreaks.
4. The 2nd barrier is important in reducing the risks from campylobacter and salmonellas to acceptable levels if compost is ingested.
5. *Clostridium* spores will not be destroyed by composting or biogas. The risk assessment cannot eliminate *Clostridium botulium* spores in compost as a potential risk to infants (under 6 months of age). However, the spores levels predicted in compost are no higher than reported for some soils.
6. The composting approach outlined here provides more control points than landfilling. Composting could potentially present lower risks to animal health than the current practice of disposal of catering waste to landfill.
7. The risks to human health from consumption of crops grown on land to which compost has been applied are very low.

Notes on the risk assessment

The models described in Figures 1 and 2 allow for composting of all of the uncooked meat (i.e. both the “meat” and “non-meat” fractions), which is discarded each year to catering waste in the UK. The risks predicted in Table 5 are based on the assumption that the Source-Separated “non-meat” fraction of **all** catering waste in the UK is composted (either by “in-vessel” or a windrow) and that compost is then applied randomly to land across the UK. The model assumes that any pathogens surviving the composting process are contained in the 500,000 tonnes of compost currently produced annually in the UK. Clearly, if composting expands within the UK, then the total tonnage of compost produced will increase greatly, thus lowering the pathogen concentration (per tonne) in the compost. In this sense the predicted risks to individual humans ingesting compost as presented in Table 5 are too pessimistic and would decrease in magnitude. In contrast, the estimates of the numbers of infected animals will remain constant because the greater tonnage of compost will be spread over a greater surface area of land, potentially exposing a greater number of animals (albeit to lower individual risks).

1. Introduction

The Animal By-Products Order 1999, as amended, requires that all catering wastes that could contain or have been in contact with meat or other products of animal origin be disposed of so that livestock and wild birds cannot gain access to them. Since swill-feeding is banned, this material is currently being disposed of primarily to landfill or incineration. Land-filling is not a sustainable option in the long term. There is, therefore, increasing pressure to find more sustainable disposal routes. One option is the use of composting and Biogas plants to treat the materials, with land-spreading to dispose of the compost or residues.

The Government strongly supports the composting of waste. It is expected to be a vital tool in helping the UK meet stringent targets for reducing landfill of biodegradable municipal waste (BMW) under the Landfill Directive. By 2020 the amount of BMW landfilled must be reduced to 35% of that produced in 1995. Composting is also expected to be a vital component of meeting *Waste Strategy 2000* targets for recycling and composting. The Government has set targets to recycle or compost at least 25% of household waste by 2005, rising to at least 33% by 2015. New EC rules, coming into force in mid-2002, will permit the use of composting and Biogas plants to dispose of low-risk (category 3) animal by-products, manure, gut contents and catering wastes containing meat. The Animal By-Products Regulation will require that the material is reduced to a particle size of no more than 12 mm and then treated, in a closed unit, so that all the material in the unit reaches a temperature of 70°C for 60 minutes. Providing the material complies with certain microbiological standards, the compost or residues may then be spread to non-pasture land. Manure and gut contents may also be treated in this way and spread to any type of land. Category 2 animal by-products (e.g. fallen stock) could only be used in a composting or Biogas plant if they had first been rendered to 133°C at 3 bar pressure for 20 min. Category 1 material (e.g. Specified Risk Material) could not be used at all.

1.1 Objectives

The overall objective of this project is to determine the risks to animal, public and plant health from the land application of various categories of animal by-products and catering wastes containing meat.

More specifically, the objectives are:-

1. To compare the risks from the following three options

- Maintain the current ban on the use of composting and biogas to dispose of animal by-products and catering waste containing meat;

- Adopt the new EC rules;

- Adopt specific UK standards;

2. To determine any minimum standards that might be needed to reduce those risks to an acceptable level.

1.2 Project appreciation

The objective “adopt specific UK standards” requires the alternative disposal strategies to be examined with respect to their ability to prevent the spread of a range of pathogens identified as being of concern to human, animal and public health. Given the wide range of pathogens of concern and the number of treatment options to be considered, the only feasible approach is to carry out a series of microbiological risk assessments.

The approach taken for environmental risk assessment is the Source, Pathway, Receptor approach. The Source Term defines the reservoirs of infectivity, and includes the frequency of infections and the loadings of pathogen in the catering wastes and other animal by-products studied. The Receptor Term defines the animal, human and plant categories that may be exposed. Central to the Receptor Term are dose-response data (i.e. how susceptible to infection is each species), and information on the nature of the disease. In this respect, information on the transmissibility through secondary spread is of major importance. For example, horizontal transmission of BSE (from cow-to-cow) occurs at such low frequency that it has not yet been detected by epidemiology. In contrast, foot and mouth disease rapidly spreads both within and between herds.

The Pathway Term defines the routes by which the various receptors might be exposed to pathogens in the Source Term. Some pathways will be well understood through epidemiological studies from outbreaks and sporadic cases. Other pathways will not have been identified, either because they are rare events or because epidemiological studies have not yet been able to separate them out.

Central to the Pathway Term are the barriers, which protect or attenuate exposure. There are two types of barrier. The pathway barriers include rendering, composting, decay on the soil. Dilution in the soil is also important if the composted waste is sub-surface injected. The biomedical barriers control how infectious the microbiological agent is to the receptors. Examples of biomedical barriers include the species barriers in BSE, acquired protective immunity in *C. parvum* infection and the natural gut microbiota in Salmonella infection (see Gale, 2001).

The pathways and pathway barriers will be presented in the form of event trees. An example of an event tree is shown in **Figure 1.1**. This defines the pathways and barriers for transmission of BSE agent from abattoirs to agricultural land through the application of sewage sludge. The fractions define the proportion of BSE infectivity through the pathway and must add up to 1.0 for the arrows coming from each node.

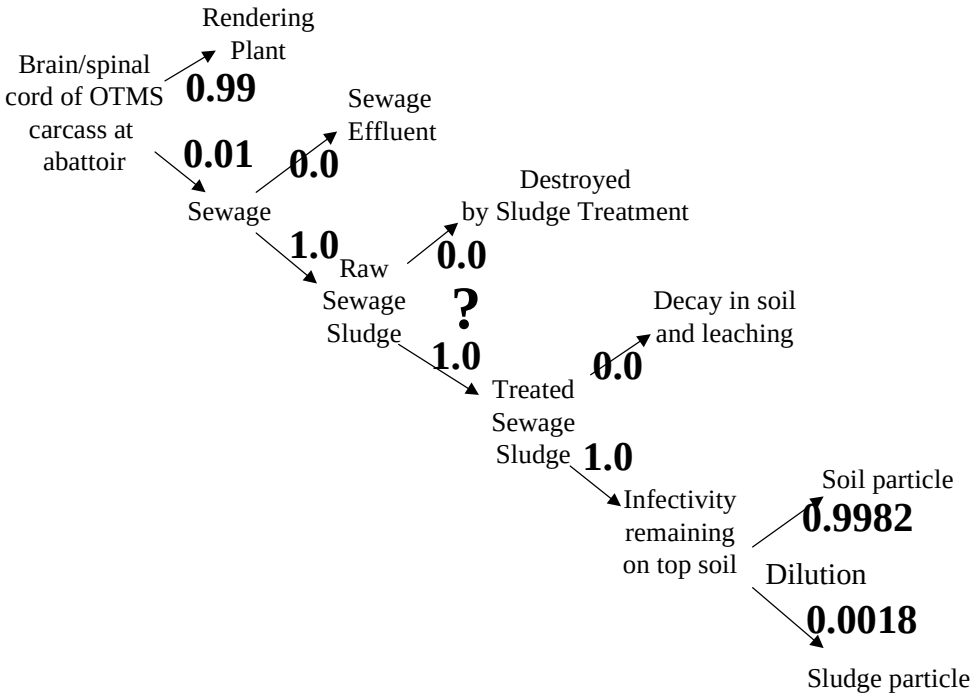

Figure 1.1 Event tree for BSE agent from abattoir to soil through application of sewage sludge (from Gale & Stanfield, 2001).

1.3 Variation and uncertainty

There will undoubtedly be natural variation in the Source, Pathway and Receptor Terms. Natural variation in concentrations of pathogens is accommodated in risk assessment either by using Monte Carlo simulations or more simply, by using the arithmetic mean exposure (Haas, 1996; Gale and Stanfield, 2000; Gale and Stanfield, 2001). Indeed, the risk assessment for BSE in sewage sludge was carried out simply by estimating the arithmetic mean concentration for BSE in sewage sludge in England and Wales.

1.4 General Approach

The approach for environmental risk assessment is based on the Source – Pathway – Receptor approach.

The risk assessment involves gathering information and modelling seven areas. These are:-

1. What is the prevalence of pathogen occurrence in cattle, sheep, pork and poultry food products in the UK. This is relatively easy to assess for diseases such as scrapie and BSE, but is very difficult to address for the exotic pathogens such as FMDV and CSFV. Indeed, levels will fluctuate depending on the incidence in illegally imported meats and whether there is an outbreak in the UK;

2. What is the pathogen loading in the different tissues of infected animals;

3. What happens to individual animal tissues at abattoirs, for sheep, cattle, pigs and poultry;

4. What happens to these different tissues in catering outlets (as catering waste) and domestic kitchens (as MSW);

5. What happens to pathogens during the various composting/biogas processes;

6. How much decay of pathogens is there in the soil; and

7. Dose-response

2. Source Term

"Catering waste" means all waste food originating in restaurants, catering facilities and kitchens, including central kitchens and household kitchens.

Household kitchens will tend to discard kitchen waste to the dustbin for collection by the council. The meat component of municipal solid waste (MSW) is therefore considered.

2.1 Household consumption of meat

Meat consumption from a survey of 14,584 households by DEFRA is presented in **Table 2.1**. The average consumption of uncooked meat across households was 1,208 g person^{-1} week^{-1}.

The total annual consumption of meats by household in the UK is therefore 1,208 x 52 x 60 x 10^6 g = 3.77 million tonnes year^{-1}.

Table 2.1 Household consumption of meats by households in UK (Source DEFRA)

Meat	Average Consumption (grams person^{-1} week^{-1})
Beef and veal	55
Mutton and lamb	68
Pork	720
Uncooked bacon and ham	71
Uncooked poultry	214
Bacon and ham cooked	41
Cooked poultry	39
Total	1,208

2.2 Meat discarded to the waste bin in household surveys

Some preliminary results of the bin-survey carried out in the Wales are presented in **Table 2.2** for the summer months and **Table 2.3** for the winter months.

Table 2.2 Household waste survey (summer). 250 households sampled.

Waste category	Mean wt (kg)	
	Positive data	All data
Compostable kitchen waste	2.42	**2.24**
soil	6.94	**0.44**
kitchen non-compostable	1.80	**1.62**
other putrescible	1.35	**0.30**
Garden waste	5.04	**2.50**

Table 2.3 Household waste survey (winter). 156 households sampled.

Waste category	Mean wt (kg)	
	Positive data	All data
Compostable kitchen waste	2.27	**2.08**
Soil	9.29	**0.95**
Kitchen non-compostable	2.09	**1.82**
Other putrescible	0.34	**0.06**
Garden waste	5.99	**2.00**

2.2.1 Composition of the putrescible material household biowaste

Limited data on the composition of waste produced weekly by 23 households out of a total of around 1600 households participating in a biowaste source-separation scheme have been reported (Barr *et al.* University of Leeds).

The mean amount of biowaste waste produced by these households was 3.5 kg per household per week. All (99.9 %) of the biowaste was putrescible material although 23.4% of the black bin waste (total 196.1 Kg per week) collected from the same households also contained putrescible material but was observed to be composed mainly of garden waste. The biowaste was composed of fresh food (81%) and cooked food (19%) but no data was given on the type of food that comprised these two categories.

A study was carried out by Greenfinch Limited on behalf of WRc-NSF Ltd in support of this study. The contents of 8 bags (10% of total) collected from a scheme for treating source separated kitchen waste from individual households in Burford, Shropshire. The average kitchen bag weight was 3.4 kg per household per week. Uncooked fruit and vegetables comprised the greatest majority (60% by weight) of the putrescible material. Cooked meat, including bone, accounted for 12% of the total weight whilst the uncooked meat accounted for an ever smaller proportion (1%) of the total weight.

2.2.2 Summary

The three studies give remarkably good agree with between 3 and 4 kg household^{-1} week^{-1} (**Table 2.4**).

Table 2.4 Summary of total kitchen waste discarded weekly by households from three studies

Study	Kitchen waste (kg per household per week)
EA study	3.96 (winter) and 4.16 (summer)
Leeds study	3.5
Greenfinch	3.4

Assuming there are 20 million households in the UK, and on the basis that 1% of the 4.0 kg of kitchen waste discarded weekly is uncooked meat, the total uncooked meat discarded annually would be 4.0 kg x 0.01 x 20 x 10^6 x 52 x 10^{-3} = 41,600 tonnes year^{-1}.

The total annual consumption of meat in the UK is calculated as 3.77 million tonnes (**Section 2.1**). Therefore, 1.1% of meat is discarded uncooked to the bin.

It is assumed for the purpose of the risk assessment that 1% of meat purchased by households and catering establishments is discarded to the bin uncooked.

A survey undertaken on this contract by WRc-NSF of the amount of uncooked meat discarded to bins by local catering establishments is presented in the Appendix together with a similar survey undertaken for domestic households. The results for the domestic kitchen waste are presented in **Table 2.5**. No account is taken of perception bias.

Table 2.5 Summary of results of survey by WRc-NSF of 39 domestic kitchens. Note, percentages are only estimates and were not weighed.

Number of people	% of uncooked meat discarded	Product
1	20	20
1	10	10
18	5	90
7	1	7
10	0.5	5
2	0	0
Total = 39		Total = 132
		Average discarded is 3.4%

The survey suggests that the average percentage discarded is 3.4%. This is 3.4-fold higher than the 1% used in the risk assessment. However, a value of 1% is used in the

risk assessment because it appears for the survey that catering establishments discard very little uncooked meat in catering waste. This is because most of the meat is purchased pre-butchered.

2.3 Catering Outlets

Catering waste includes hotels, restaurants, pubs, popular catering, leisure, staff catering, health care, and education canteens.

2.3.1 Meat use in catering outlets in UK

The quantities of beef, lamb, pork, bacon and ham used in catering outlets in the UK are summarised in **Table 2.6**.

Table 2.6 Breakdown of meat distribution to catering outlets in UK (The Foodservice market meat monitor, 2001). Tonnes purchased per week by sector.

Outlet	Beef	Lamb	Pork	Bacon	Ham
Hotels	264.5	78.4	61.0	204.9	23.7
Restaurants	1356.4	123.5	190.6	769.5	440.9
Pubs	399.4	79.3	62.3	146.3	23.3
Popular catering	223.7	71.4	72.8	274.3	55.9
Leisure	137.4	98.9	33.9	70.7	38.2
Staff Catering	276.6	92.8	70.5	326.2	65.2
Health Care	279.1	120.2	106.9	121.2	36.8
Education	182.9	152.0	121.8	126.1	48.7
Total	3120.0	1234.9	719.8	2039.2	732.7

2.3.2 Amount of uncooked meat discarded by catering outlets

As part of this project WRc-NSF undertook a telephone survey to find out exactly how much uncooked meat is discarded by commercial catering operations. The questionnaire is presented in Appendix 1.

2.4 Other pathogen sources in household domestic waste

In addition to kitchen and food wastes, domestic waste will include:-

- Pet food;
- Dog and cat faeces (cat litter);
- Dead pets (hamsters, mice, cats?);
- Nappies.

Microbial contamination of municipal solid waste (MSW) is mainly of faecal origin (Deportes *et al.* 1998). For example, 1 – 4% of the dry weight of MSW consists of soiled disposable diapers.

2.5 Poultry

In England and Wales in 2000, some 615 million broilers were slaughtered (DEFRA). This excludes “Dead on Arrivals” and includes registered slaughter houses only. The average live weight was 2.26 kg. This is therefore 615 x 10^6 x 0.00226 = 1,389,900 tonnes.

In 2000, household consumption was 214 and 39 g person^{-1} week^{-1} of uncooked and cooked poultry (Food Standards Agency). The total consumption of poultry is therefore 253 x 52 x 60 x 10^6 g = 789,360 tonnes.

Over 700 million chickens are sold per year in the UK (cited in Harrison *et al*. 2001).

These figures appear to be in relatively good agreement.

3. Animal Tissues Composition

3.1 Cattle

In the UK in 2000, some 2.28 million head of prime cattle were slaughtered for the human food chain (data from Meat and Livestock Commission (MLC)). This included 4,700 live cattle imports, and accounted for 708,000 tonnes of beef products entering the human food chain.

In addition 307,000 tonnes of meat was imported. This comprised 202,000 tonnes of carcasses and 105,000 tonnes of processed meat.

Table 3.1 Cattle by-products (data from MLC). Organs discarded as SRM in italics.

Category	Weight (kg)
Carcase (ex K,KKCF)	***284.4***
KKCF	***10.0***
Kidneys	***1.0***
Gut contents	***80.0***
Intestinal fat	***12.0***
Caul fat	***13.0***
Intestines	***15.0***
Stomachs	***14.0***
Lungs	***3.2***
Heart	***2.0***
Lung fat	***1.4***
Trachea (weasand) & trim	***1.0***
Sweetbreads (thymus)	***0.3***
Liver, gall bladder	***7.5***
Pancreas	***0.3***
Spleen	***0.8***
Hide	***38.0***
Feet	***10.0***
Head, tongue	***14.0***
Blood	***18.0***
Cerebro-spinal fluid	
Skirt	***1.1***
Tail	***1.0***
Reproductive organs	***1.2***
Udder	***0.9***
Lymph nodes	
Waste	***6.5***

3.2 Sheep

According to MLC data, 19.14 million heads of sheep were marketed in the UK in 2000. This included 169,000 head imported. DEFRA statistics (www.defra.gov.uk) confirm 15.96 million "other sheep and lambs" and 2.42 million "ewes and rams" were slaughtered for meat in the United Kingdom in 2000.

In addition 123,000 tonnes of sheep meat was imported into the UK in 2000.

The masses of sheep-by-products (per carcass) are summarised in **Table 3.2**.

Table 3.2 Sheep by-products – weights in lambs. Organs discarded as SRM in italics. For sheep tissues multiply by a factor of 1.6. Data from MLC.

Category	Weight (kg)
Carcass (ex K,KKCF)	***17.60***
KKCF	***0.60***
Kidneys	***0.10***
Gut contents	***4.50***
Intestinal fat	***0.50***
Caul fat	***0.65***
Intestines	***1.20***
Stomachs	***1.00***
Lungs	***0.40***
Heart	***0.20***
Lung fat	***0.30***
Trachea (weasand) & trim	***0.05***
Sweetbreads (thymus)	***0.05***
Liver, gall bladder	***0.65***
Pancreas	***0.10***
Spleen	***0.10***
Fleece and pelt	***4.10***
Feet	***0.72***
Head, tongue	***1.50***
Blood	***1.70***
Cerebro-spinal fluid	
Skirt	***0.20***
Reproductive organs	***0.13***
Lymph nodes	
Waste & Tail	***0.75***

KKCF, Kidney Knob and Channel Fat

3.3 Pigs

The total number of pigs in the UK June census is 6.5 million in 2000 (MLC pig year book 2001).

DEFRA statistics (www.defra.gov.uk) confirm 12.4 million “clean pigs” and 0.32 million “sows and boars” were slaughtered for meat in the United Kingdom in 2000.

Table 3.3 Pig by-products (Data from MLC)

Category	Weight (kg)
Carcass (incl. head, feet, kidneys and flare)	62.9
Carcass (ex 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***
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***

Farez and Morley (1997) describe the various tissues in which pig viruses replicate. Pork and pork products are comprised principally of skeletal muscle, bone and fat. 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 carcase. With respect to pork portions, lymph nodes and bone marrow may not be present as a result of trimming, deboning on the cut. Many of the lymph nodes are removed through carcass trimming due to their fat-embodied location on the carcass. The blood, respiratory, GI and reproductive tracts, the head, the respective lymph nodes of these parts and the tonsils are not pork tissues.

The tissues in which the various pig viruses replicate are an important consideration in the risk assessment.

3.4 Chickens

Giblets

The giblets are defined as the heart, liver and gizzard of a poultry carcass.

A recent survey by the Food Standards Agency showed that 268 of 4,881 (5%) of chickens sampled on retail sale contained giblets.

4. Modelling The Pathways

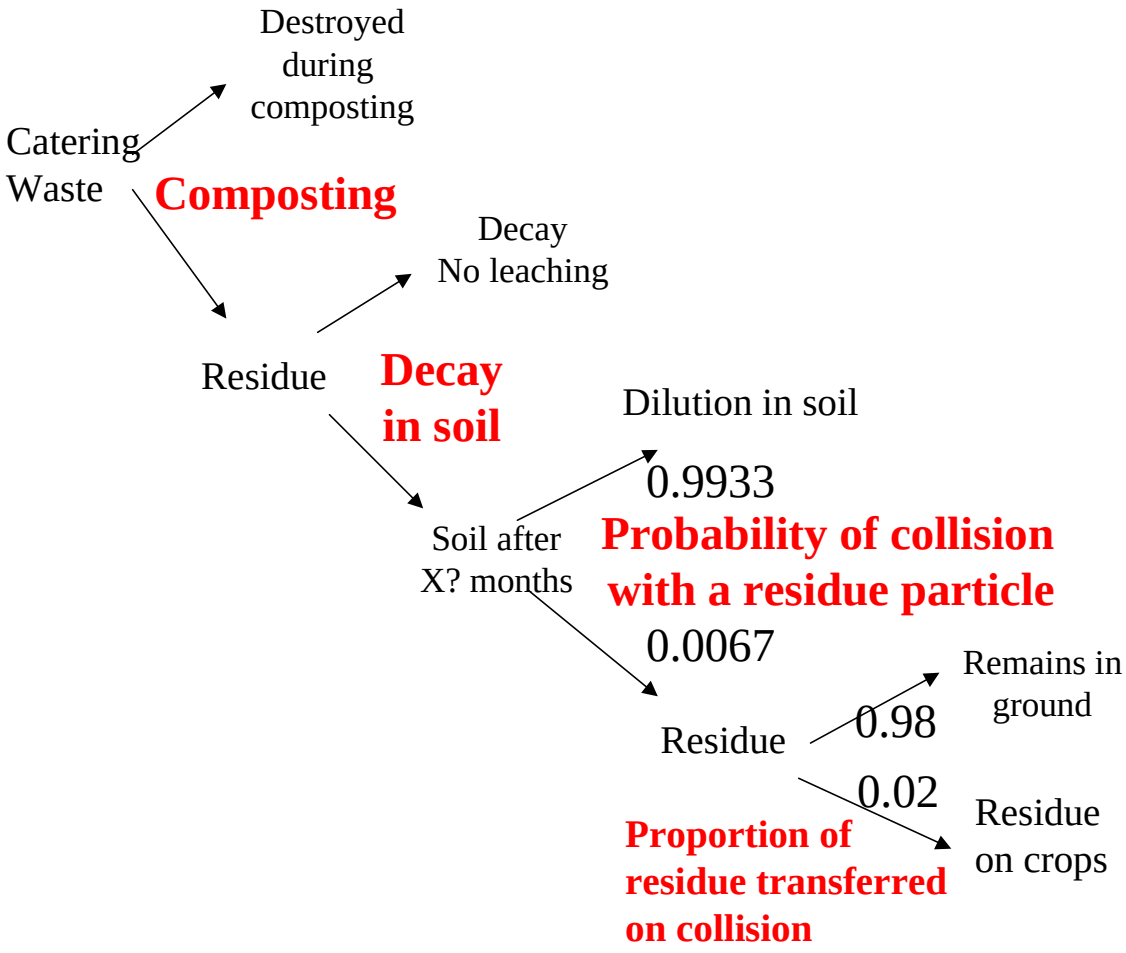

Figure 4.1 Event tree for transmission of pathogens in composted catering wastes to root crops.

An event tree outline the main barriers for pathogens in composted catering waste applies to soil is shown in **Figure 4.1.**

4.1 Growth of pathogenic bacteria in catering waste

This only applies to the bacterial pathogens (see **Section 20.2**) and includes spores of *Clostridium botulinum* after germination. Viruses, protozoa and TSE agents cannot replicate outside the host. The main problem in modelling growth of bacterial pathogens in catering waste is allowing for the competition effect of indigenous bacteria. Berry and Koohmaraie (2001) studied the influence of various levels of endogenous beef bacteria on the growth and survival of E. coli O157:H7 on bovine carcass surface tissue. This is considered in greater detail in **Section 20.2**.

4.2 Dilution in the soil

There will undoubtedly by dilution of any pathogens present in the compost once it has been applied to the soil. The question for quantitative risk assessment is estimating the dilution factor. This is relatively straight-forward for application of sewage sludge to land (Gale and Stanfield, 2001) because the sludge is tilled into the soil to a depth of 0.25 m. Compost, however, may not necessarily be tilled in, particularly for grasslands. In terms of risk assessment, dilution can be modelled as the probability of a grazing animal ingesting a soil particle, compared to the much smaller probability of ingesting a sludge or compost particle. For the purpose of risk assessment, it is assumed that any pathogens present in compost are "diluted" down to a depth of 0.1 m (i.e. 10 cm). This could occur through leaching of the pathogens during the 2 month no grazing ban, or through mechanical disruption of soil (e.g. ploughing before sowing of vegetable crops).

The dilution factor assuming 10 tonnes ha^{-1} of composted residue is tilled in to a depth of 10 cm of soil is 150-fold (**Figure 4.2**). Thus, the probability of a crop colliding with a compost particle is 0.0067 (**Figure 4.1**).

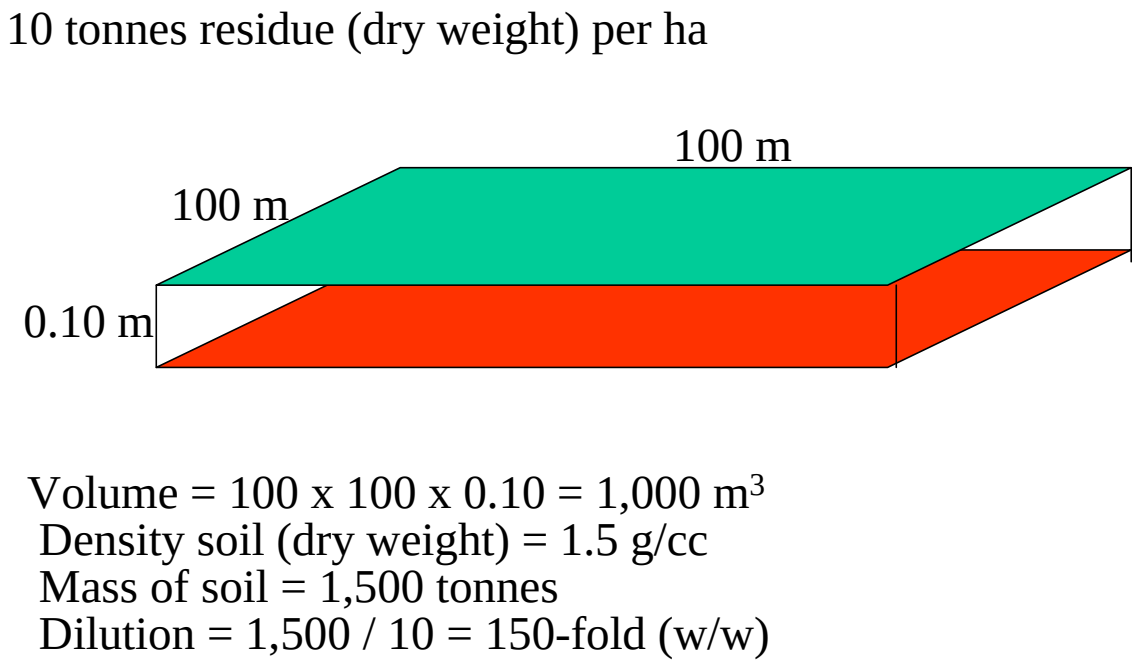

Figure 4.2 Dilution of composted catering waste residues in soil. Based on assumption that any pathogens in compost leach down to a depth of 10 cm.

4.3 Decay of pathogens on land

There is a lot of information on the decay of endemic pathogens on soil to which slurries and sludges have been applied. This is now summarised.

4.3.1 Decay of pathogens in sludge-treated soil.

Salmonellas

The die-off of salmonellas in sludge-treated soil is affected by several factors such as moisture, temperature and sunlight. Andrews *et al.* (1983) reported a T_{90} (time for 1-log decay) for the winter period of 17 d. In the summer the T_{90} was 3.7 d. Over a period of seven weeks between April and June 1976, Watson (1980) demonstrated a 2.2-log reduction in salmonellas in soil to which treated-sewage sludge had been applied. Watkins and Sleath (1981) demonstrated a 2-log reduction for salmonellas over five weeks in soil to which raw sewage sludge had been sprayed. The experiment was conducted in mid-winter. The reduction over 8 weeks may well have been greater than 2-logs since undetectable readings were recorded at 6-8 weeks. The approach adopted here in the risk assessment is conservative in not extrapolating the decay rates to the 12 month interval specified by the Safe Sludge Matrix between application of conventionally-treated sewage sludge and harvesting of root crops. The model thus allows for only 2-log decay of salmonellas (**Table 4.1)**.

Campylobacters

Unpublished studies of campylobacter survival following land spreading of dairy farm yard manure on a clay loam grassland soil demonstrated a 2.4-log destruction after 16 d (ADAS, pers. comm.). Decays of 2-log and 3-log were reported for beef farm yard manure and beef slurry after 16 d. These are comparable with a 0.74-log decline in campylobacter counts after 5 d in matured unaerated slurry sprayed to land (Stanley *et al.* 1998). The risk assessment assumes a 2-log decay of campylobacters on the soil (**Table 4.1**).

E. coli O157

Maule (1995) compared survival of cultured *E. coli* O157:H7 in soil cores, cattle slurry, cattle faeces and river water at laboratory scale. While die-off was particularly rapid in slurry, *E. coli* O157:H7 survived best in the soil cores. Counts in the soil increased slightly between day 0 and day 7. Regression analysis of the data of Maule (1995) showed a 1.05-log decay between day 14 and day 63. A 1-log decay is used in the risk assessment model (**Table 4.1**). An increase of about 1-log (10-fold) in faecal coliform counts was observed by Stanley *et al.* (1998) in slurry after application to topsoil after one day. Thereafter the counts declined. Field studies of slurry containing *E. coli* applied to land (Fenlon *et al.* 2000; Ogden *et al.* 2001) suggested T_{90} of 16 days. Strachan *et al.* (2001) allow for a T_{90} of 16 days in a model for an environmental outbreak (New Deer, Scotland, May – June 2000) of *E. coli* O157 infection.

Protozoa

Whitmore and Robertson (1995) studied the survival of *C. parvum* oocysts in sludge-amended soil mesocosms (2% w/w) At 10°C, the viability decreased from 91% to 60-70% over 30 d. Olson *et al.* (1999) demonstrated that decay of *Cryptosporidium* oocysts in soil samples was strongly influenced by temperature. Thus, a 1-log decay of oocysts required 12 weeks at 4°C, but only seven weeks for 25°C. At soil temperatures of -4°C,

however, only 50% of the oocysts had decayed at ten weeks. Viability was determined by dye exclusion. The risk assessment assumes a 1-log decay for *Cryptosporidium* oocysts in the soil (**Table 4.1**).

Giardia cysts do not survive as well as *Cryptosporidium* oocysts in the soil environment. Olson *et al.* (1999) demonstrated a greater than 1-log loss of viability in cysts at one week in unautoclaved soil at -4°C. At 25°C, a l-log loss of cyst viability occurred at two weeks. At 4°C, a 1-log loss of viability occurred at about six weeks. Hu *et al.* (1996) demonstrated destruction of *Giardia* cysts within 12 weeks following soil amendment of anaerobically digested sewage sludge. In the second trial of Hu *et al.* (1996), cysts were initially present at a concentration of approximately 600 g^{-1}. The population had declined to less than 100 cysts g^{-1} after one week, and no cysts were detectable after 12 weeks. It would therefore be reasonable to apply a 600-fold (2.78-log) reduction on the basis of the data of Hu *et al.* (1996). For the purpose of risk assessment, a 2-log destruction in the soil is allowed for over a 12 week period (**Table 4.1**). This is consistent with an extrapolation of the 4ºC data of Olson *et al.* (1999) from six weeks to a 12 week period.

Viruses

The retention and persistence of enteroviruses in soil is influenced by a number of factors including virus type, type and texture of the soil, and temperature (Straub *et al*. 1992; Sobsey *et al.* 1995). The survival of viruses is enhanced by a combination of low soil temperature and sufficient moisture. Tierney *et al*. (1977) spiked raw sewage sludge with poliovirus and monitored the decay after application to soil plots. In the winter months a 3-log decay was recorded in about 80 d, while during the summer months a 3-log decay occurred in about 7 d. Straub *et al.* (1992) applied anaerobically digested sludge, spiked with poliovirus to desert soil in laboratory studies. At 15ºC, a 1-log decay occurred in the region of 10 – 16 d depending on the soil type. The risk assessment (**Table 4.1**) uses a 3-log reduction based on the experiments of Tierney *et al*. (1977) which were conducted in Ohio (USA) and are therefore more representative with respect to the climatic conditions in the UK than the desert conditions studied by Straub *et al*. (1992).

Table 4.1 Summary of parameters for decay of pathogens in sewage sludge after application to soil.

Pathogen	Decay in soil as log_{10} units Time frame of experiment in parentheses
Salmonellas	2.0 (5 weeks; winter)
Campylobacters	2 (16 d)
E. coli O157	1.0 (49 d; 18°C)
Cryptosporidium	1.0 (12 weeks; 4°C)
Giardia	2.0 (12 weeks; 4°C)
Enteroviruses	3.0 (80 d; winter)

4.4 Receptor Term

Information supplied by growers suggested that root crops contain 2% (w/w) soil at point of harvest (Gale and Stanfield, 2001). Thus when a tonne of root crops collides with a tonne of compost residue in the event tree (**Figure 4.1**), 0.02 tonnes of compost will transfer to the root crops.

Cattle ingest 0.41 kg soil cow^{-1} day^{-1} on average (EUSES 1997). It is assumed here that sheep and lambs ingest half this amount. It is assumed that pigs ingest the same amount of soil as cattle.

4.5 Exposure to grazing cattle, pigs and sheep across England and Wales.

The Composting Development Group (1998) reported an estimated 18 million ha of agricultural land in the UK, of which 11 million ha are land where the addition of compost would have little benefit in increasing prevailing high levels of organic matter (e.g. grassland, natural grazing or woodland). They estimated that compost could be used on about 2.6 million hectares of land. A typical application rate would be 20-25 t ha^{-1}. To avoid excessive applications of phosphate, compost is unlikely to be applied more than one year in four in the longer term.

According to "The State of Composting 1999" (Stater *et al*. 2001), a total of 462,768 tonnes of composted material was produced in the UK during 1999 (see **Section 5.2**) from some 833,044 tonnes of feedstock material (see **Section 5.1**). For the purpose of risk assessment it is assumed that 500,000 tonnes of composted material containing catering waste is applied to 50,000 ha of agricultural land in England and Wales. This represents an arithmetic mean application rate of 10 tds ha^{-1} $year^{-1}$. The total tillage and grassland in England and Wales is 9.5 million ha (**Table 4.2**). Thus, composted catering waste is applied across 0.52% of total tillage and grass land in England and Wales. Assuming compost is applied at a rate of 25 tds ha^{-1}, then 500,000 tonnes could only be spread over 20,000 ha of agricultural land. On this basis, 0.21% of the total tillage and grassland in England and Wales would be used.

Table 4.2 Total tillage and grass land in England and Wales (Anon 1997)

Country	ha
England	8407700
Wales	1129600
Total	9537300

The total number of cattle, sheep and pigs in England and Wales according to the 1996 survey (Anon 1997) are presented in **Table 4.3**.

Assumption

Assuming cattle, pigs and sheep graze randomly over the total tillage and grassland in England and Wales, then 0.52% of the animals would be exposed to soil to which composted catering waste had been applied. The total numbers of animals exposed are presented in **Table 4.3**.

Table 4.3 Total number of cattle, pigs and sheep in England and Wales (Anon 1997).

	Cattle	Pigs	Sheep
England	6,805,300	6,275,400	19,089,900
Wales	1,359,900	98,800	10,874,000
Total	8,165,200	6,374,200	29,963,900
Number of animals exposed (0.52% of Total)	42,800	33,417	157,100

This is a worst case assumption in that it assumes cattle, pigs and sheep graze randomly on land across the UK to which composted catering waste may have been applied.

Furthermore, a proportion of pigs may be housed on intensive systems inside and therefore not exposed to land to which compost has been applied.

5. Amount Of Material Composted In The Uk

5.1 Total feedstock material

The overall picture for composting in the UK is one of continued expansion. One of the key challenges facing the industry over the next few years is the continued expansion required to provide an alternative to landfill for biodegradable waste to contribute to national statutory recycling and composting targets and EU Landfill Directive obligations.

In 1999, Slater *et al.* (2001) report there were a total of 90 operators running 197 sites and processing approximately 833,044 tonnes of material within the UK. Approximately 74% of this material came from municipal sources and 26% came from non-municipal sources. Of the 618,517 tonnes of municipal waste composted, 72% was green waste from bring sites, 17% was green waste from Local Authority parks and gardens and only 7.5% was collected from the kerbside. Municipal waste composted was comprised of 80% (493,520 tonnes) household waste and 20% (124,997 tonnes) non-household waste.

5.2 Types of composted product

Slater *et al.* (2001) report that 462,768 tonnes of composted material was produced in the UK during 1999, accounting for 55% of the total feedstock material. The quantities and proportions of composted material are presented in **Table 5.1**.

Table 5.1 Quantity and proportion of composted material

Composted product	Tonnes	%
Mulch	166,772	36.0
Soil conditioner	164,480	35.5
Landfill cover/remediation	66,132	14.3
Growing area	43,126	9.3
Top soil	9,000	2.0
Other	13,258	2.9

6. Particle Size, Temperature And Time For Pathogen Destruction By Composting And Biogas

6.1 Objectives

The objectives of this section are to:-

1. Review data for heat inactivation data of pathogens for different time/temperature regimes;

2. Consider the effect of particle size in relation to surrounding temperature;

3. On the basis of the most resistant pathogens, define a minimum temperature;

4. Identify a time/temperature combination to be achieved by composting/biogas;

5. Consider the degree of by-pass of different composting/biogas processes; and

6. Formulate credit system of log-removals for use in the risk assessment.

BSE and scrapie prions and *C. botulinum* spores are excluded because for the purposes of risk assessment they are considered not to be affected by the temperatures achievable by composting and biogas processes.

6.2 Introduction

The on going discussions have also been noted concerning the need to achieve a balance between pathogen kill, through the use of high temperatures, and obtaining a stabilised compost.

The use of high temperatures can apparently lead to a loss of the soil conditioning properties of compost and the use of lower process temperatures has been advocated by some. It is proposed that where lower temperatures are used the indigenous microflora of the material being composted will be preserved and that competition for nutrients created by this natural microflora will cause pathogen numbers to decline. Microbiologically this appears to be a sound argument in that composting processes will be an alien and therefore inimical environment for pathogens. The indigenous microbes will be better suited to this environment and will flourish at the expense of any pathogens present through competition for nutrients and predation. Furthermore, the indigenous microbes are important in preventing salmonella regrowth in compost (Sidhu *et al*. (2001)).

6.3 Heat inactivation of exotic viruses

Turner *et al.* (2000) report data on decontamination of pig slurry containing exotic viruses of pigs (namely FMDV, Aujeszky's disease virus and CSF) by heat inactivation. The work demonstrated the suitability of thermal treatment in ensuring the safety of pig slurry following a disease outbreak.

6.3.1 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 6.1**. The net destructions, calculated on pooling both the slurry and medium data, are presented in **Table 6.1**. The data show that even at the lower temperatures of 55°C and 60°C considerable inactivation occurs 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. It should be noted that during the composting process, meat in catering waste will be exposed for much longer time periods than 10 minutes.

Table 6.1 Net destruction of FMDV over 10 minutes. Data from Turner *et al.* (2000). Data averaged for medium and pig slurry counts.

Temperature	0 minutes	10 minutes	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

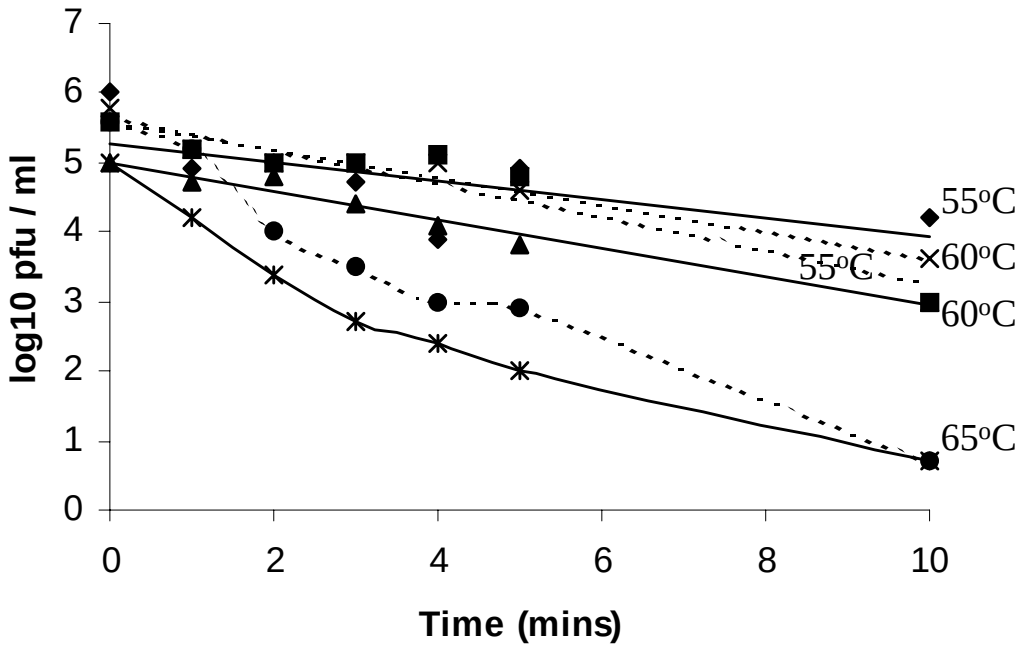

Figure 6.1 Laboratory scale thermal treatment of FMDV incubated at different temperatures with time in pig slurry (solid line) and Glasgow Eagles medium (dashed line). Data from Turner *et al.* (2000).

6.3.2 Classical Swine Fever Virus

The heat inactivations of CSFV in pig slurry and medium at three temperatures are plotted in **Figure 6.2**. Data are plotted from Turner et al. (2000). A destruction of over 4-logs in 5 minutes was reported for CSFV at 55°C (**Table 6.2**), with greater destructions apparent at higher temperatures.

Table 6.2 Net destruction of CSFV over 10 minutes. Data from Turner *et al.* (2000). Assumes starting titre for experiments at 60°C and 65°C is 7.0 $\log_{10}$. Data averaged for medium and pig slurry counts.

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

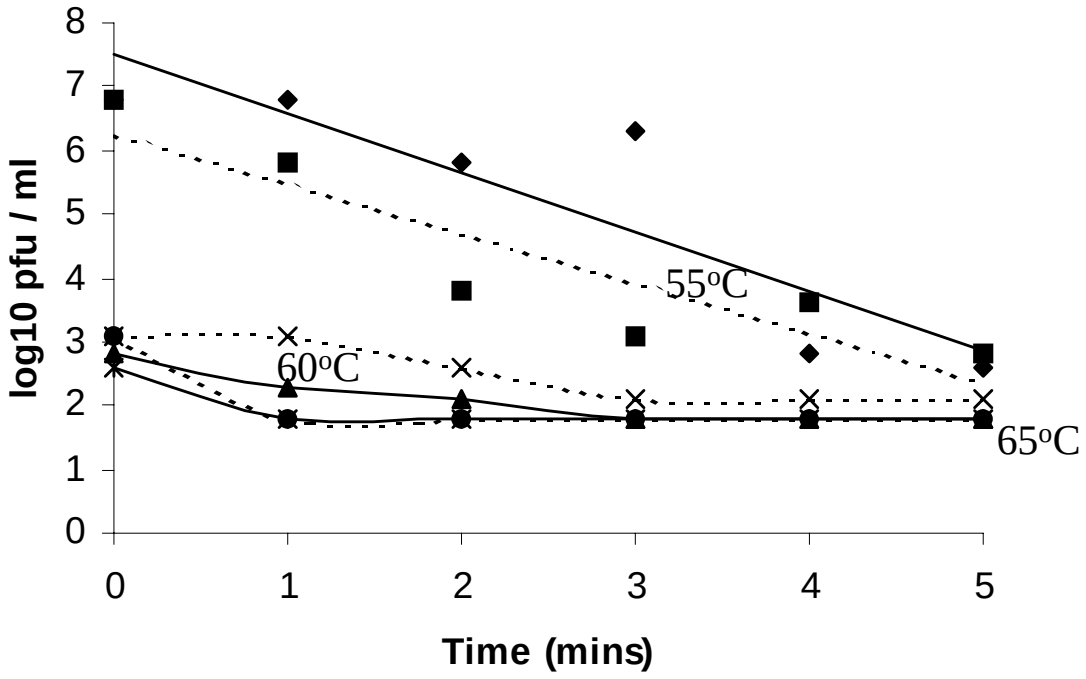

Figure 6.2 Laboratory scale thermal treatment of Classical Swine Fever Virus incubated at different temperatures with time in pig slurry (solid line) and Glasgow Eagles medium (dashed line). Data from Turner *et al.* (2000). Starting titre for all experiments was measured at 7.0 $\log_{10}$.

6.3.3 Aujeszky's disease virus (ADV)

Heat inactivation of ADV at 55°C, 60°C and 65°C in Glasgow Eagles medium and pig slurry is shown in **Figure 6.3**. It is apparent that the medium has a protective effect. The net log-inactivations for ADV in the medium are presented in **Table 6.3**. The greatest protective effect is observed at 55°C. Indeed in pig slurry a 6-log removal was observed after 15 minutes (**Figure 6.3**), compared to just a 2.5-log inactivation in Glasgow Eagles medium (**Table 6.3**).

Table 6.3 Net destruction of Aujeszky's disease virus (ADV) over 15 minutes. Data from Turner *et al.* (2000). Data averaged for medium counts (and not pig slurry).

Temperature	0 minutes	10 minutes	Net destruction	log net destruction
55°C	1,995,262	6,309	316	2.5
60°C	2,511,886	16	158,489	5.2
65°C	630,957	16	39,810	4.6

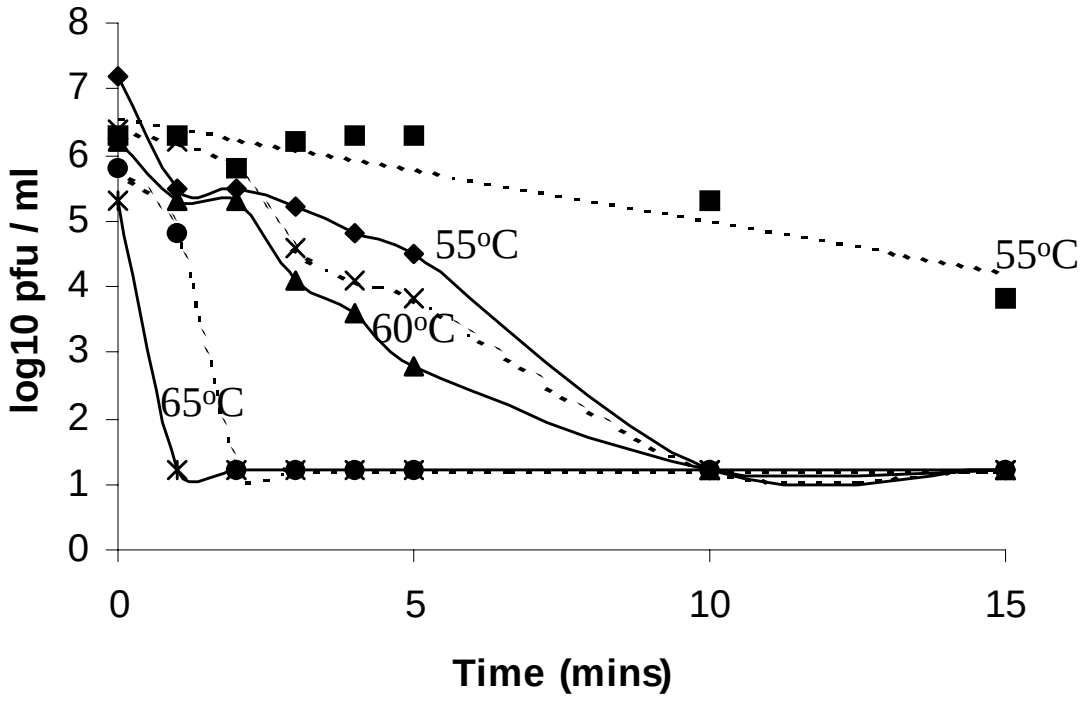

Figure 6.3 Laboratory scale thermal treatment of Aujeszky's Disease Virus (ADV) incubated at different temperatures with time in pig slurry (solid line) and Glasgow Eagles medium (dashed line). Data from Turner *et al.* (2000).

6.3.4 African Swine Fever and Swine Vesicular disease

Plowright and Parker (1967) demonstrated rapid initial destructions of 7-log and 5-logs of ASFV at 56ºC 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 6.4**). In slurry, however, a temperature of 56°C only achieved a 3-log reduction in 2 h (**Table 6.5**).

Table 6.4 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 6.5 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

6.3.5 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. Peaks 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 (cited in Senne *et al.* 1994).

6.3.6 Conclusions

Turner and Burton (1997) review the inactivation of viruses in pig slurry. They conclude that the most suitable treatments are the use of heat at about 60°C for up to 30 minutes, or the application of an appropriate concentration of chemical, such as NaOH or CH_2O (formaldehyde). At 55°C considerable destruction of pig viruses occurs within 10 – 15 minutes. Furthermore, 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. Thus:-

- FMDV was undetectable after aeration at a pH of 8 at 50°C for 48 h.
- ADV needed 5 h aeration at 40°C for inactivation, and;
- SVD needed 48 h aeration at 40°C.

6.4 Destruction by composting of bacterial pathogens

Lung *et al.* (2001) spiked *E. coli* O157:H7 and *Salmonella enteritidis* into raw compost feed 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 Windrow piles are presented in **Table 6.6**.

Table 6.6 Effect of Windrow (at laboratory scale) on bacterial pathogens spiked into sewage sludge. Data from Horan and Lowe (2001).

Pathogen	55C / 4 h	40C / 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 enteriditis	>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 salmonella in windrow composting of pig manure. However, their data question the efficiency of windrows. They demonstrated a drop 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 MSW by a windrow composting process over 14 days. Counts of total streptococci decreased by 5-logs.

6.5 By-pass: Variation and uncertainty in the net pathogen destruction by composting or biogas

In terms of the treatment barriers, defining the degree of both "within-batch" (spatial variation) and "between-batch" (temporal variation) will be important. For example, a treatment process which typically gave a 6-log destruction, would only give a 2-log reduction if it failed completely with a frequency of 1% (**Table 6.7**). Thus, if 1% of the raw material by-passed the process, it would effectively wipe out 4 of the 6-log removal. This illustrates the benefits of minimising "within-batch" and "between-batch" variation in the composting process. The effect of by-pass is less 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 6.7**).

6.5.1 Key point for risk assessment:

It is not so much whether a 2, 3, 4, …6, or even 7-log destruction of pathogens by composting can be achieved under laboratory conditions, but how much material by-passes the process.

Table 6.7 Effect of "within-batch" and "between-batch" variation (e.g. from short-circuiting and dead spaces in a digester) on the net destruction of pathogens.

Treatment conditions	% by-passing treatment and receiving 0-log destruction	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

An example of how the destruction ratios for microorganisms vary in presented in **Table 6.8**. *E. coli* were placed in bags at different locations. The destruction ratios varied between 1.6 and >5.8-logs.

Table 6.8 Fate of *E. coli* in bags placed in situ in aerated static piles (Data from Horan and Lowe 2001).

Sample	log-removal
C (coolest)	1.6
D (medium)	2.2
E (hot)	>5.8
F (hot)	>5.8

6.6 "In-vessel" process

Temperature data for an "in-vessel" composting unit taken on one day during the hotter stages of processing are plotted in **Figure 6.4**. It is apparent that there is greater variation at the edges and that the average temperature is lower. A more detailed study of the temperatures near the edges demonstrated that temperatures of 60°C are achievable even 10 cm from the edge, although the temperatures are some 5°C lower than further in (**Figure 6.5**).

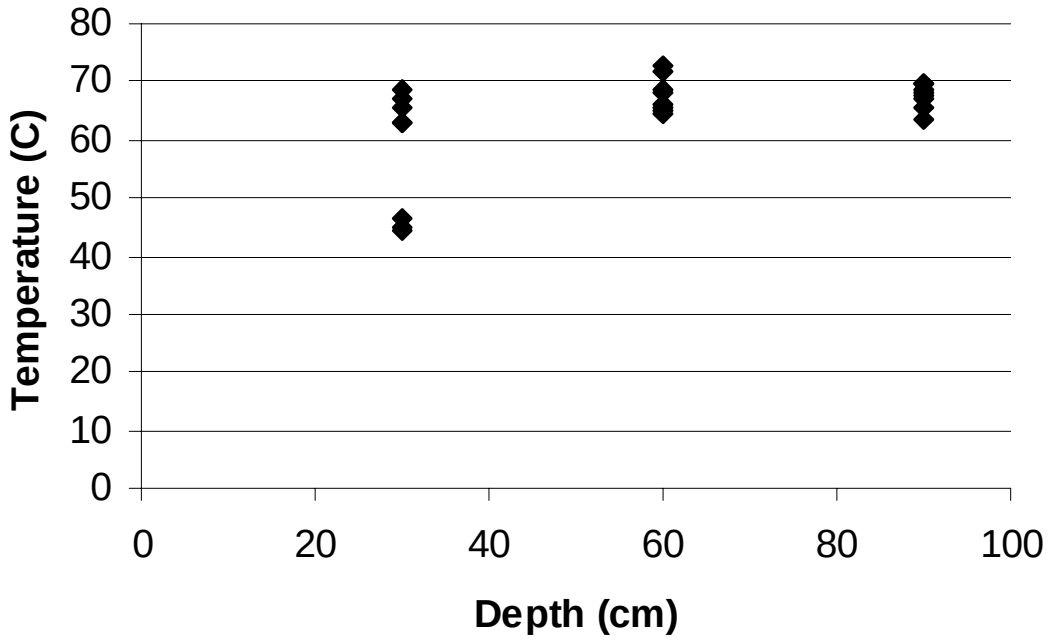

Figure 6.4 Temperature measurements for an "in-vessel" system taken on one day during the hotter stages of processing. Data kindly provided by Dr Joe Short of London Remade Organics Eco-site.

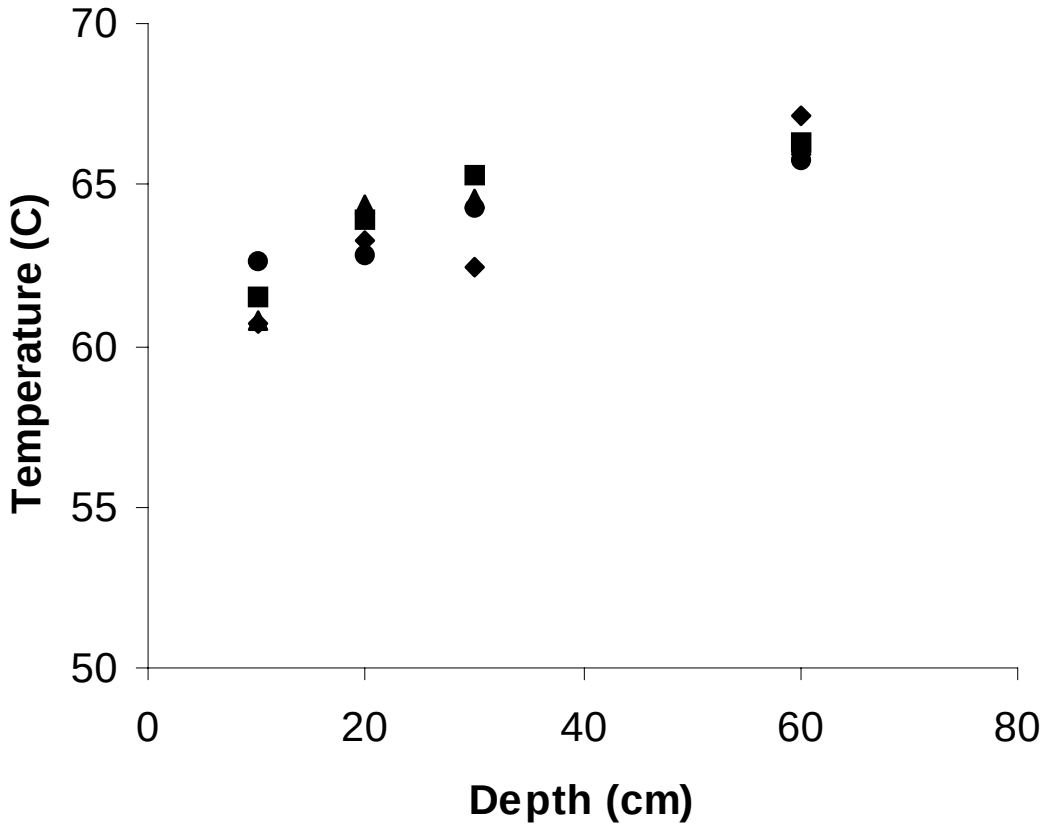

Figure 6.5 Temperature measurements recorded for an "in-vessel" system taken at 1.5 days. Data kindly provided by Dr Joe Short of London Remade Organics Eco-site.

Senne *et al*. (1994) demonstrated differences in temperature between the different layers in a laboratory scale composting process. Peaks temperatures for the upper layer were 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.

6.6.1 Edge effects could contribute a considerable by-pass for "in-vessel" systems.

Consider an "in-vessel" system that is 2 m in height. Assuming 10 cm did not get up to temperature, then 0.1/2.0 (i.e. 5%) of the contents would be below temperature. Thus, the net destruction could never be greater than 95%.

6.7 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 that "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 organic 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 6.9**. They concluded that predominantly thermophilic conditions were maintained in the windrows throughout processing and virtually all material was subjected to the commonly recognised 55°C for three days which ensures the destruction of potential pathogens in organic material.

Table 6.9 Percentage cross sectional area of windrows reaching certain temperatures throughout green organic processing (Joshua *et al*. 1998).

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

6.7.1 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 1 $$n_i = n_0\left[f_l + f_h e^{(-k_d \Delta t)}\right]^N$$

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 1** is simplified by assuming that a given proportion (π) of the pathogens survive in the high temperature zone after a time *t*. Thus:-

Equation 2 $$n_i = n_0\left[f_l + f_h \pi\right]^N$$

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 6.9**) 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 2** in **Table 6.10**. These are plotted in **Figure 6.6**. It is apparent that the for destructions of 2-logs or more in the high temperature zone, then the net destruction is controlled by the number of turns.

Table 6.10 **Log_{10} 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 2).**

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**
	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 6.10).

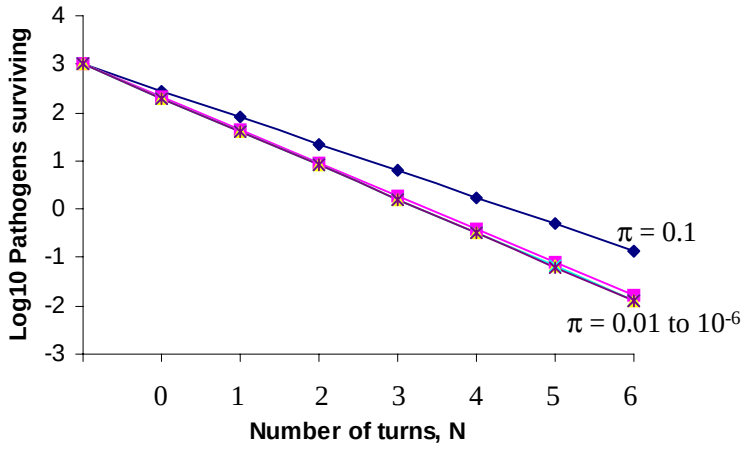

Figure 6.6 **Effect of number of turns of a windrow on pathogen survival. Model assumes 80% of pathogens are in a high temperature zone where proportion (π) of pathogens surviving.**

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 6.10**). Thus three turns reduces the counts for 3-logs to 0.79-logs.

Recommendation

The windrow should be turned at least three times to achieve a >2.7-log destruction.

6.8 Destruction of pathogens by thermophilic biogas systems

The risk assessment relies almost entirely on data for thermophilic biogas reported by Bendixen (1999). Some T_{90} values (the time to achieve 90% kill) for biogas are given in **Table 6.11**. The increased rate of decay at the thermophilic temperatures compared to mesophilic temperatures is significant. Similarly, although the data for slurry systems is incomplete, the performance of the Biogas system at mesophilic temperatures appears significantly better.

Table 6.11 T_{90} values for Selected Microorganisms in Biogas and Slurry Systems. Data from Bendixen (1999).

Organism	Biogas System		Slurry System	
	53°C (h)	35°C (d)	18-21°C (weeks)	6-15 °C (weeks)
S typhimurium	0.7	2.4	2.0	5.9
E coli	0.4	1.8	2.0	8.8
Mycobacterium paratuberculosis	0.7	6.0		
Enterococci	1.0	2.0		
Clostridium perfringens	NR	NR	NR	NR
Bacillus cereus	NR	NR		
Gastro worms	1-4	2.0		
Ascaris worms	1-4	21-35		
Lungworm	1-4	<7		

Mesophilic biogas digestion gives reductions of 1 – 2-logs, often below 1 (Bendixen 1999). Indeed for one plant studied by Bendixen (1999), destruction of FS faded out to nothing. It is concluded that mesophilic digestion is not appropriate for treatment of catering waste.

Bendixen (1999) reported the average faecal streptococci (FS) counts for four thermophilic biogas digestion plants. These are presented in **Table 6.12**. Removals for the digestion process ranged between 2.76-logs and 4.78-logs. For three of the four plants, FS counts increased between the digestion tank and the storage tank, due to comtamination. This is a classic case of by-pass of the raw material into the treated product (see **Section 6.8.1**).

Table 6.12 Average counts of FS in four thermophilic biogas plants (Table IV from Bendixen 1999)

Plant no.	Conditions (digestion temp; MGRT; HRT)	Receiver	Digestion tank	Storage tank	log reduction	
		(1)	(2)	(3)	(1) to (2)	(1) to (3)
II	56°C; 3 h; 20 d	364,000	620	8,000	2.76	1.66
V	53C;2 h; 14 d	201,000	56	900	3.56	2.34
VIII	53C; 5 h; 19 d	610,000	<10	2,100	4.78	2.46
IX	52C; 4 h; 15 d	2,400,000	1,600	700	3.18	3.54
Net destruction - logs					3.17	2.12

Table 6.13 Daily removals of faecal streptococci between the receiver tanks and the digestion tank from Table VI of Bendixen (1999). The conditions for this plant (no. VIII) were thermophilic digestion tanks (53°C) with mean guaranteed retention time (MGRT) of 5 h and hydraulic retention time (HRT) of 19 days.

Date	Log-removal	Proportion surviving
Sept 1992	4.83	0.0000148
Oct 1992	5.18	0.0000066
Nov 1992	4.18	0.0000661
Jan 1993	4.36	0.0000437
Feb 1993	4.95	0.0000112
Mar 1993	4.93	0.0000117
Mar 1993	5.23	0.0000059
April 1993	4.72	0.0000191
May 1993	4.72	0.0000191
June 1993	4.04	0.0000912
July 1993	5.04	0.0000091
Aug 1993	4.72	0.0000191
Sept 1993	3.38	0.0004169
Oct 1993	4.56	0.0000275
Nov 1993	4.75	0.0000178
Dec 1993	4.3	0.0000501
Jan 1994	4.52	0.0000302
Feb 1994	4.57	0.0000269
Mar 1994	4.65	0.0000224
May 1994	4.56	0.0000275
June 1994	4.04	0.0000912
Aug 1994	4.94	0.0000115
Oct 1994	5.04	0.0000091
Net		0.000045 (4.34-log)

Bendixen (1999) reported the daily variation in faecal streptococci removal ratios for a Biogas plant No VIII. The conditions for this plant were thermophilic digestion tanks (53°C) with mean guaranteed retention time (MGRT) of 5 h and hydraulic retention time (HRT) of 19 days. Daily removals varied between 3.38-logs and >5.23-logs between the receiver tanks and the digestion tanks (**Table 6.13**). The net removal by thermophilic

digestion was 4.34-logs. (The value of 4.62 quoted by Bendixen (1999) is actually the geometric mean removal an inappropriate as discussed in Gale and Stanfield (2000)).

6.8.1 Operational by-pass of biogas systems

Of greater interest is the fact that salmonella levels increased in the storage tanks due "a leakage or operational error resulting in transfer of untreated biomass to the storage tank in November 1992".

Table 6.14 Daily removals of faecal streptococci between the receiver tanks and the storage tank from Table VI of Bendixen (1999). The conditions for this plant (no. VIII) were thermophilic digestion tanks (53°C) with mean guaranteed retention time (MGRT) of 5 h and hydraulic retention time (HRT) of 19 days.

Date	Log-removal	Proportion surviving
Sept 1992	3.53	0.000295
Oct 1992	4	0.0001
Nov 1992	0.82*****	0.151356
Jan 1993	2.69	0.002042
Feb 1993	2.8	0.001585
Mar 1993	2.63	0.002344
Mar 1993	2.75	0.001778
April 1993	2.46	0.003467
May 1993	3.07	0.000851
June 1993	2.39	0.004074
July 1993	3.06	0.000871
Aug 1993	1.92	0.012023
Sept 1993	2.78	0.00166
Oct 1993	2.26	0.005495
Nov 1993	3.43	0.000372
Dec 1993	1.88	0.013183
Jan 1994	3.37	0.000427
Feb 1994	2.65	0.002239
Mar 1994	2.47	0.003388
May 1994	4.56	2.75E-05
June 1994	4.04	9.12E-05
Aug 1994	4.04	9.12E-05
Oct 1994	3.53	0.000295
Net		*0.009 (2.04-log) 0.0025 (2.60-log)

*poor removal due to leakage; Note changing Nov 1992 value of 0.82-logs to 3.0-logs gives a net removal of 2.6-logs.

This a classic case of by-pass and reduced the arithmetic mean removal to just 2.04-logs (**Table 6.14**). Indeed changing this November value from 0.82-log removal to 3-logs gave a net removal of 2.6-logs. Furthermore, according to Bendixen (1999) "During the following months, minor irregularities caused small contamination with untreated biomass. The operational procedures have been improved" and 4.1 to 6.3-log reductions are observed when the plant is operated correctly (**Table 6.15**).

Table 6.15 Faecal streptococci counts in biogas plant VIII after improvement of operational procedures to eliminate minor irregularities causing contamination with raw material.

Sampling Date	Receiver tanks	Digestion tanks	Storage tanks	log_{10} reduction
March 1998	1,300,000	<5	<5	5.4
May 1998	140,000	<5	<5	4.4
July 98	>9,000,000	<5	<5	>6.3
November 98	62,000	<5	<5	4.1

6.8.2 Determining a net removal for thermophilic biogas plant

It is apparent that for thermophlic biogas plants, a net removal of 2.12 logs (**Table 6.12**), or 2.60-logs (**Table 6.14**) could be used for poorly operated plants or 4.1 to 6.3-logs (**Table 6.15**) for well-operated plants. This net removal depends entirely on the degree of contamination of the digested material with raw material.

For the purpose of risk assessment a value of 2.7-logs is used to fit in with the windrow removal.

6.9 Summary of pathogen destructions by composting and biogas

Table 6.16 Net removal ratios for the processes and storage

Barrier	Net removal
In-vessel	2.7-log
Windrow	2.7-logs
Stockpiling/storage	1.0-logs
Biogas	3.7-logs

The model considers the composting process in terms of barriers. These are: -

1. “in-vessel” process – assumes< 0.2% of raw material does not get up to 60°C – gives 2.7-log reduction.

2. windrow maturation – assumes at least three turns (**Table 6.10**) – gives 2.7-log reduction.

3. Biogas – assumes <0.02% of raw material by-passes the process giving 3.7-log removal; removals of >4.4 log are achievable at operational scale (**Table 6.15**).

4. storage/stockpile prior to sale for 18 days gives 1-log reduction of CSFV.

Log-removals are summarised out in **Table 6.16**.

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

The approach for risk assessment is outlined in **Table 6.7**. In the hot area, the process should be able to achieve almost complete destruction (i.e. 6-log for the purposes of **Table 6.7)**. In this respect a temperature of 56°C for 4 h would be required to give a 6-log destruction of SVDV in slurry. This is based on data in **Table 6.5** which show a 3-log destruction of SVDV at 56°C in 2 h. For most bacterial pathogens a 55°C for 4 hour gives 6-log destruction (**Table 6.6**). It is assumed that composting has no affect on BSE agent or scrapie agent.

6.10.1 Proposed UK standard for composting - A temperature of 60°C for 2 days.

Haug (1993) estimated the heat transfer times into spherical compost particles. These are shown in **Table 6.17**.

Table 6.17 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 6.17**, 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 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 to 20°C. Thus,

Equation 3 $\mathbf{(T - T_0)/(T_1 - T_0) = 0.9}$

if T_1 = 60°C. To reach this condition requires a time of 40 h for a sphere of 40 cm diameter (**Table 6.17**).

Consider a leg of pork with a bone in from an FMDV-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 the catering waste bin e.g. after a freezer failure, it is appropriate to define the composting conditions to deal with such challenges.

A temperature of 60°C for 2 days is therefore recommended for composting as this will ensure the centre of such joints is up to 56°C.

6.10.2 The EU conditions (70°C for 1 hr)

Assuming a value of T_1 = 70°C in **Equation 3**, then T = 65°C.

According to Haug (1993), the equation relating to the time (t hours) for a sphere of radius R (cm) to meet **Equation 3** is:-

Equation 4 $kt / \rho cR^2) = 0.3$

where k (thermal conductivity)= 3 cal/h-cm^2, ρ (mass density) = 1 g/cm^3 and c = 1 cal/g. Thus, it may be calculated that a sphere of 2.9 cm radius will reach a core temperature of T_1 = 65°C after t = 50 minutes. Such a temperature will give a >4-log destruction of FMDV (**Table 6.1**), CSFV (**Table 6.2**), and SVDV (**Table 6.5**) within 10 minutes (i.e. 1 hour in total) and is therefore appropriate. However, it is limited to particles of diameters of <6 cm.

The EU conditions are therefore not appropriate for dealing with large (40 cm diameter) pieces of meat such as legs of pork. However, if the particle size is reduced to <6 cm diameter, the EU conditions are appropriate.

6.10.3 Biogas (57°C; MGRT = 5 h)

To achieve a core temperature of 56°C, the maximum particle size should be 5 cm in diameter. This is calculated for a system where the surrounding temperature is 57°C.

Thus,

Equation 5 $(T - T_0)/(T_1 - T_0) = 0.97$

if T_1 = 57°C, T0 = 20°C and T_1 (which is required at the core of the sphere) = 56°C. To reach this condition requires a time of 50 minutes for a sphere of 5 cm diameter (**Table 6.17**).

According to Haug (1993), the equation relating to the time (t hours) for a sphere of radius R (cm) to meet **Equation 5** is:-

Equation 6 $kt / \rho cR^2) = 0.4$

where k (thermal conductivity)= 3 cal/h-cm^2, ρ (mass density) = 1 g/cm^3 and c = 1 cal/g. Thus, it may be calculated that a sphere of 2.5 cm radius will reach a core temperature of T_1 = 56°C after t = 50 minutes.

By setting the GMRT to 5 h, this will leave 4 h 10 min at 56°C, which is sufficient to give a 6-log kill of SVDV (**Table 6.5**).

It is concluded that the maximum particle size for biogas is 5 cm diameter.

7. A Credit System For Modelling The Barriers In Composting And Biogas Treatment Of Catering Waste

7.1 Source separation and definitions

Source separation is the actions 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 the waste fraction for composting which should be free of most of the meat because waste producer has been instructed to exclude meat by source separation.

The *Meat* fraction is the waste fraction for composting which contains meat derived from two sources, namely:-

1. waste stream containing the meat which has been separated at source by the waste producer from the residual stream; and

2. residual black bag waste which has not been separated at source and will include meat as well as other waste materials.

7.2 Composting/biogas of 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 Meat Exclusion at Source, Composting/Biogas and Stock-piling. Meat Exclusion at Source is assumed to be 90% efficient, i.e. source-separated "non-meat" waste contains 10% of the total uncooked meat discarded to catering waste. The barriers are set out in **Table 7.1** for composting and **Table 7.2** for biogas.

Table 7.1 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
Stock-piling (18 days)	1.0
Total	4.7

*Windrow or "in-vessel"

Table 7.2 A credit system for the barriers for biogas treatment of the "non-meat" fraction.

Process (Barrier)	Credits (log-reduction)
Meat Exclusion at Source	1.0
Biogas	3.7
Total	4.7

7.3 Composting the "meat" fraction

The meat fraction of catering waste must be composted by a two barrier process. First an "In-vessel" process in which <0.2% fails to reach 60°C for 2 days, and secondly a windrow. The windrow need not be housed as it is a secondary barrier. The barriers are set out in **Table 7.3**.

Table 7.3 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

7.4 Biogas treatment of the "meat" fraction

A two barrier process comprising biogas digestion and a storage is set out in **Table 7.4**. A storage stage of the finished product of 18 d is required to allow for a 1-log decay of CSFV.

Table 7.4 A credit system for the barriers for biogas treatment of catering waste containing meat.

Process (Barrier)	Credits (log-reduction)
Meat Exclusion at Source	0.0
Biogas	3.7
Storage (18 days)	1.0
Total	4.7

8. COMPOSTING AND BIOGAS - PUTTING THE BARRIERS TOGETHER

8.1 Note

This section describes processes to accommodate all the uncooked meat (i.e. both "meat" and "non-meat" fractions) discarded to catering waste in the UK.

8.2 Composting

The composting processes outlined in **Table 7.1** and **Table 7.3** are put together in a single event tree in **Figure 8.1** to model the overall reduction of infectivity in meat in catering waste by composting. In total 2.36 x 10^{-5} infectious dose units (IDU) from each IDU in the catering waste remain. This is equivalent to a 4.62-log reduction overall.

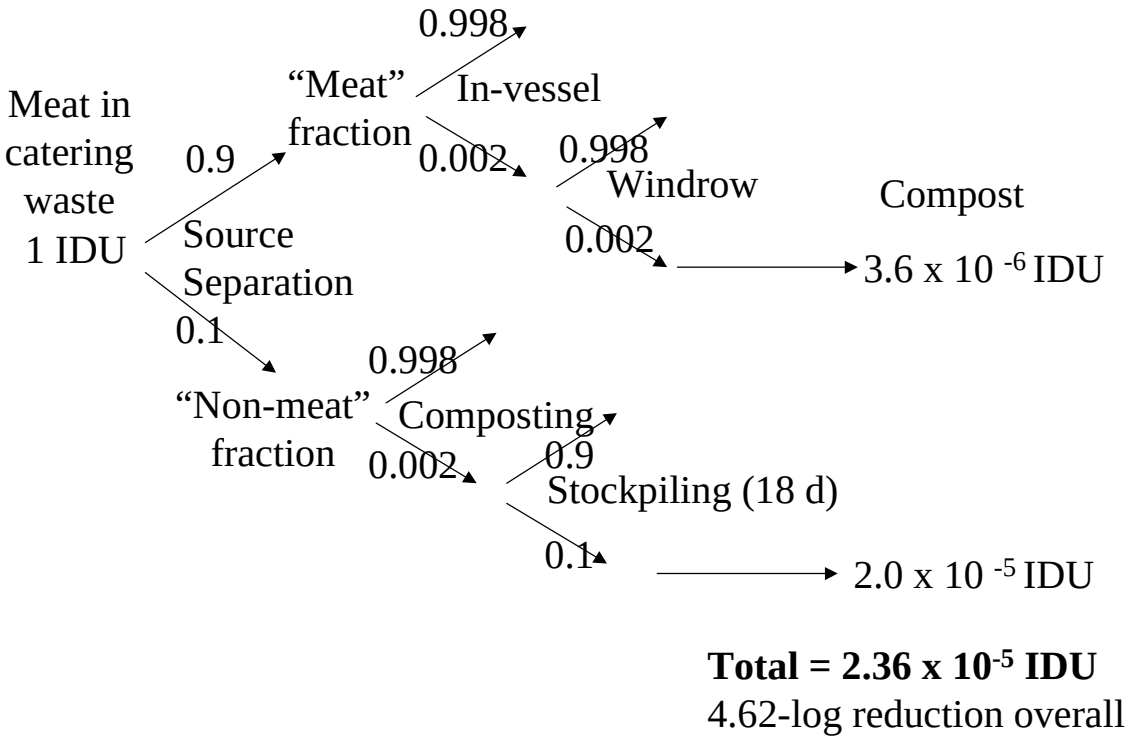

Figure 8.1 Composting as set out in Table 7.1 and Table 7.3 achieves a 4.62-log reduction of infectivity in catering waste.

8.3 Removing the Windrow (2nd barrier) and 18 d stock-piling stages from composting increases the risks by 83-fold

Removing the second stage, i.e. windrow stage in composting of the "meat" fraction reduces the net pathogen destruction from 4.62-logs (**Figure 8.1**) to 2.73-logs (**Figure 8.2**). If this windrow stage is omitted then there is little point in having the 18 d

stockpiling stage for the “non-meat” residue, since omission of this stage reduces the net destruction by just 0.3-logs (2-fold) to 2.70-logs (**Figure 8.3**).

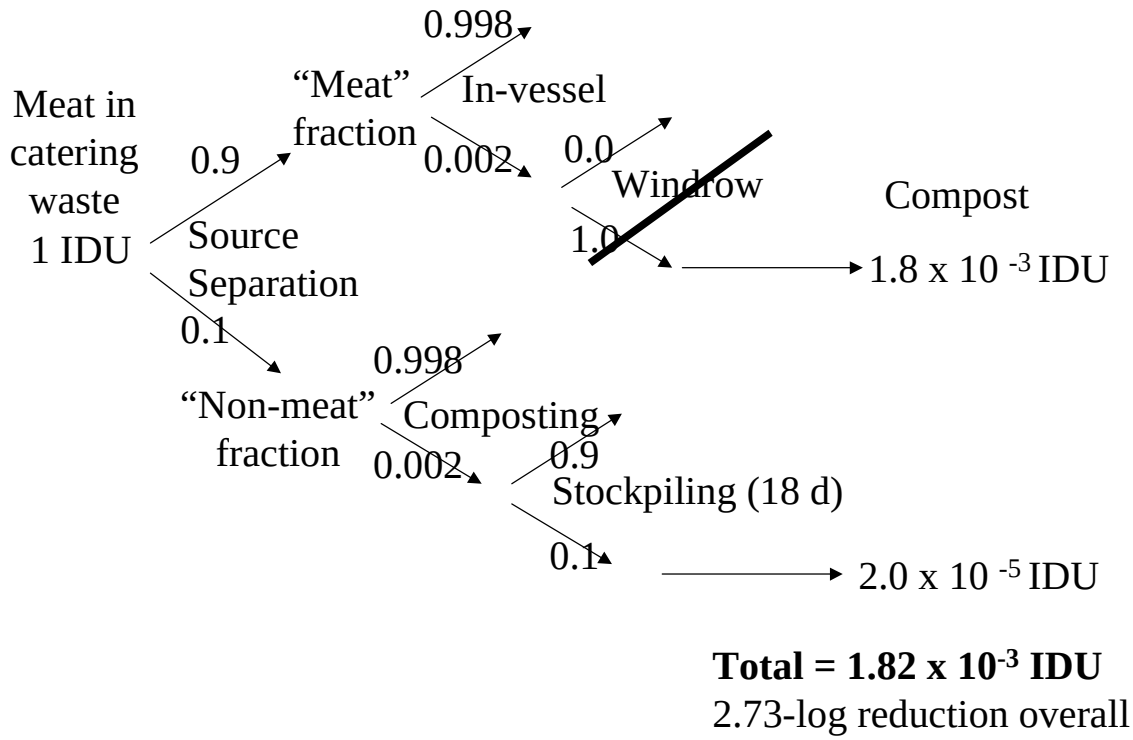

Figure 8.2 **Composting - Failure or omission of the windrow 2nd stage for the “meat” fraction reduces the net destruction of composting to 2.73-logs**

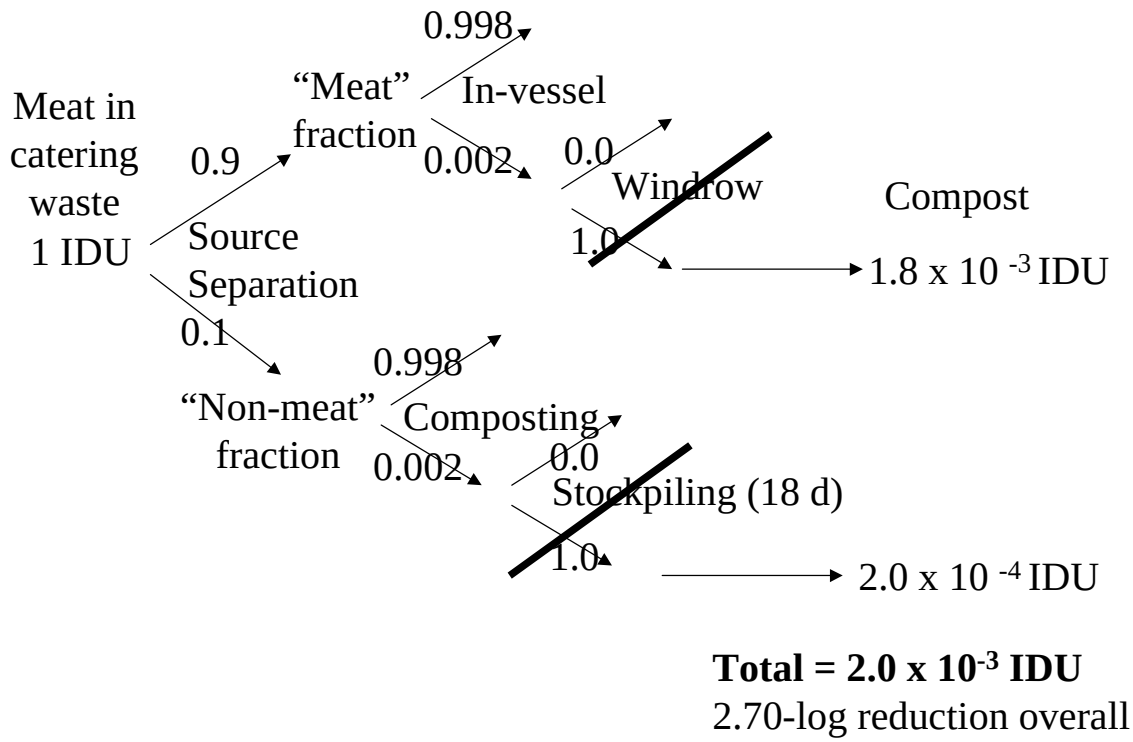

Figure 8.3 Composting - Stock-piling of the "non-meat" fraction is of little point if there is no windrow second stage for the "meat" fraction.

Thus adding in windrow as a second stage for the "meat fraction" and the stock-piling stage for the "non-meat" fraction increases the net pathogen destruction by 83-fold from 2.70-logs to 4.62-logs.

8.4 Biogas

For the biogas process, combining the processes set out in **Table 7.2** and **Table 7.4** into an event tree (**Figure 8.4**) shows that overall the net reduction of infectivity in catering waste is 4.42-logs.

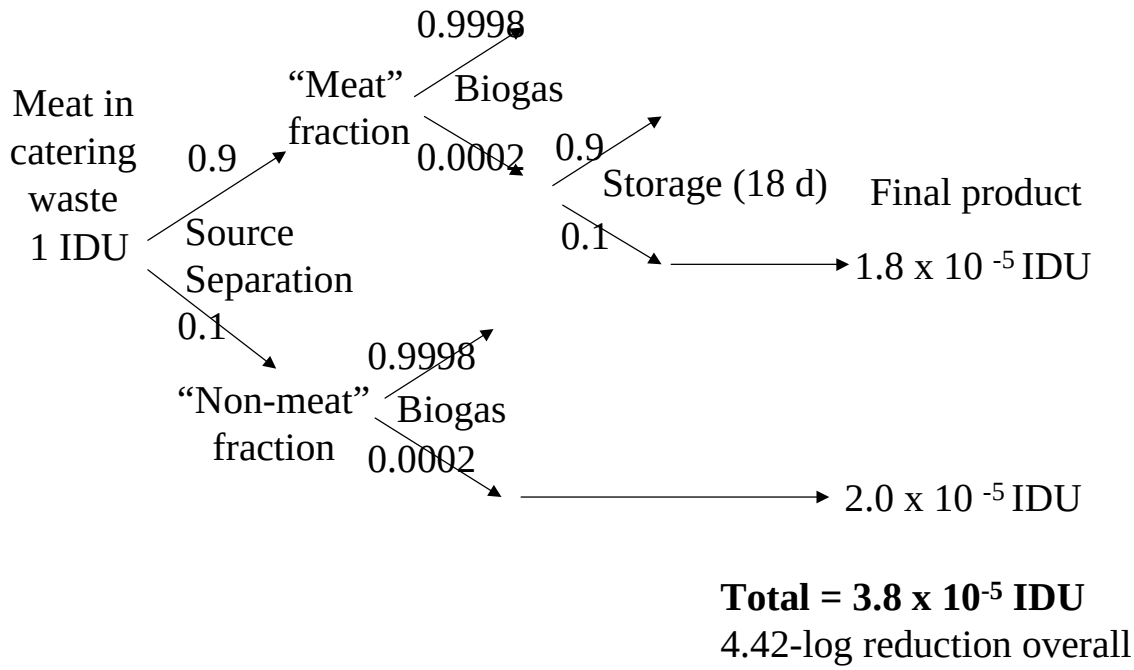

Figure 8.4 **Biogas treatment as set out in Table 7.2 and Table 7.4 achieves a 4.42-log reduction of infectivity in catering waste.**

8.5 Storage (18 days) of the "meat" fraction gives a further 5-fold reduction in the overall risk for the biogas process

Removal or failure of the 18 d storage process for the "meat" fraction reduces the net removal from 4.42-logs (**Figure 8.4**) to 3.7-logs (**Figure 8.5**). This is a 0.72-log reduction, which is equivalent to a fivefold loss in efficacy.

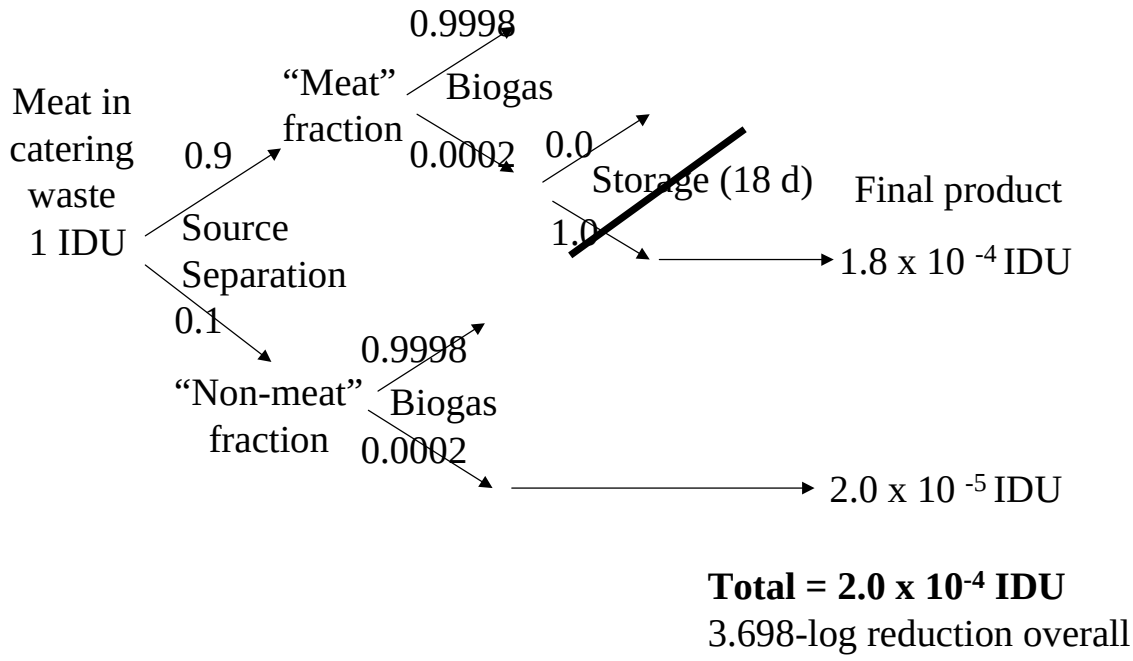

Figure 8.5 Biogas process - Omission of the 18-day storage stage for the "meat" fraction results in a 3.7-log overall reduction by biogas.

Adding the storage stage to the "non-meat" fraction has little effect increasing the net removal for 4.42-logs (**Figure 8.4**) to 4.7-logs (**Figure 8.6**), i.e less than two fold.

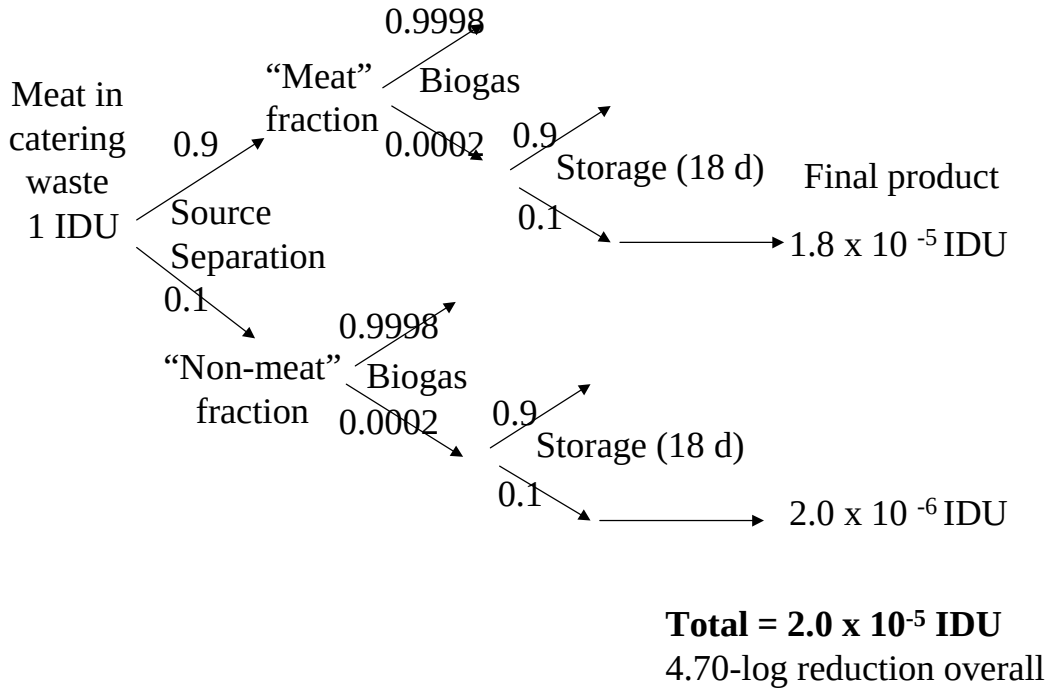

Figure 8.6 Biogas – overall effect of adding storage to the "black-bag" fraction is negligible.

9. Compare With Other Routes Of Disposal –Landfill

Department of the Environment (1995) consider the problems of birds, vermin and mud on the road at landfill sites. These are now summarised.

9.1 Bird control at landfill sites

Birds are attracted to landfill sites in large numbers, particularly where sites receive appreciable amounts of food wastes. Usually only large birds such as gulls are regarded as a nuisance. Potential concerns relating tot he presence of scavenging birds include:-

- The deposit of excrement and scraps of food on mobile plant and vehicles on-site, reducing driver's visibility and damaging nearby land;
- Causing bird-strike damage to aircraft
- The introduction of pathogens to nearby water bodies and crops
- The introduction of alien species to sensitive local habitats

Measures which can be used to mitigate bird nuisance include the employment of good landfill practice, with prompt disposal, consolidation working in small active areas and progressive covering (if appropriate) of waste, together with the use of bird scaring techniques, which include:-

- Flying birds of prey over the site
- Bird kites mimicking birds of prey
- Shell crackers – containing flare and banger
- Rope bangers
- Gas cannons
- Scarecrows – fixed or mobile
- Amplified recordings of bird distress calls
- Electronic sounds imitating calls of distress
- Bird corpses or dummies

9.2 Vermin and other pests

Landfills have potential to harbour flies and vermin, particularly where the waste contains food materials. Modern landfilling techniques including prompt emplacement, consolidation and covering of wastes in well-defined cells are effective in the prevention

of infestation by rodents and insects. Rats and flies are the main pests that require control. Sites with extensive non-operational land can become infected with rabbits.

9.3 Mud on vehicle tyres

Mud on the public highway is one of the most common causes of public complaint. Preventative measures should be incorporated into the site design to reduce the potential for mud to be carried off-site. It is therefore in the interests of the landfill operator to provide adequate wheel cleaning facilities to ensure that mud is not carried off site by vehicles.

9.4 A quantitative assessment of the pathways out of landfill.

The main barriers for composting and landfill are compared in **Table 9.1**.

To enable a quantitative risk assessment, the effectiveness of these barriers are compared for composting and landfilling. There is no data on how much meat is removed from bin-liners by gulls. In **Table 9.2**, an assumption is made that 1% of all meat in bin-liners is removed by gulls. Of this it is assumed that 90% is eaten by the gull, but that the remaining 10% is discarded somewhere outside the land fill site. In effect 0.1% of the meat entering the land landfill is taken out by birds and discarded. The approach assumes that the chance of the meat falling onto agricultural land as opposed to non-agricultural land is the same as for application of compost to agricultural vs non-agricultural land. In total therefore, land-filling only achieves a 3.0-log reduction. A bird-scrarer which reduced the number of gulls by 100-fold at the land fill, would reduce the amount of meat removed from land-fills by 100-fold. This would increase the land-fill credit to 5.0 logs. This compares to a 4.6-log reduction by the Composting process set out in **Figure 8.1**. Allow comparable at this stage, there is the 2 month grazing ban to consider for compost which cannot be controlled for landfills. Thus, the 2 month no grazing ban would enable dilution (by leaching) and decay on the soil (4.2-logs assumed in **Table 9.2**). In contrast, the meat dropped by a gull would drop onto the top of the soil, where no grazing ban could be enforced. Thus, in the extreme, the landfill route would not have any soil dilution factor or decay. According to the credit system in **Table 9.2**, composting of catering waste could achieve 8.8 credits; i.e. a 3.5-log (3,000-fold) lower risk to animal health than land-fill (even allowing for bird scarers). Thus even in well-managed landfills, the risks to animal health could be 3.5-logs (3,000-fold) higher than for composted catering waste.

An event tree is set out in **Figure 9.1** to compare the situation in which the Source-Separated “meat” fraction goes to landfill, while the “non-meat” fraction is composted according to **Table 7.1**.

It should be noted that there are more barriers through the compost/grazing ban route than through landfill, although it should be stressed that the numbers based on retention of food at landfills are not based on any data and are merely used for illustrative purposes.

Table 9.1 A qualitative comparison for control points for pathogens in uncooked meat in catering waste

Control point	Compost	Landfill
Transport from kitchen to landfill site or composting plant	Same for both processes	
Exposure of raw material to birds and wild animals	Big advantage that raw material could be delivered into negative pressure receptions, thus eliminating access – but depends on design and management of the plant – raw material stored in the open would attract birds and small mammals	Landfill open to sea-gulls and small mammals – deer also have been found on landfills.
Direct access of raw material to farm animals	Possible if composting carried out on farms – but plant could be designed and managed to prevents access	None – landfill sites are generally fenced in so that any adjacent farm animals could not get in.
Carry over of raw material to agricultural land	In theory, a small mammal or bird could transfer uncooked meat to an adjacent pig field, or alternatively get infected and then get eaten by a pig – but depends on how the plant is designed. Less scope for transmission on tyres of infected vehicles	Birds carrying meat out of landfill and discarding onto adjacent land – depends on proximity of farm to landfill site and effectiveness of bird control. Vehicle and truck tyres could carry infectivity in mud out of a landfill site and to a local farm
Treatment	"In-vessel" systems are contained and would reduce access from birds and small mammals Windrows open to air – could be picked through by birds and small mammals might choose to live in a windrow for warmth	Old material buried by incoming material – but incoming material may be continuously picked through by gulls
Disposal of treated product	Applied to land and "tilled-in"	Landfill "filled-in" and capped

Table 9.2 A credit system (representing log-reductions) for the barriers for composting and landfilling

Process (Barrier)	Landfill – credits	Compost credits
Source separation	0.0 (i.e. all meat is disposed of to land-fill)	0.0 (all meat is disposed of by composting)
Effectiveness of Process	2.0 + 1.0 (i.e. assumes 1% of raw meat is taken out of bin-liners by birds, and that 90% of that meat is eaten by birds)	4.6 ("Meat" and "Non-meat" fractions, Figure 8.1)
Bird-scarer at landfill	2.0	0.0
Disposal to other places than agricultural land	0.3 (i.e. 50% of all meat discarded by birds falls on agricultural land)	0.0 (i.e. all is put on agricultural land)
Dilution in soil	0.0 (none, i.e. piece of meat is dropped on top of soil)	2.2 (150-fold dilution) – only applicable if pathogens leach in during 2-month no grazing ban
Decay on soil due to grazing ban	0.0 (no grazing ban enforceable)	2.0 (depending on pathogen during 2 month grazing ban)
Total	3.3 (5.3 with bird scarer)	8.8

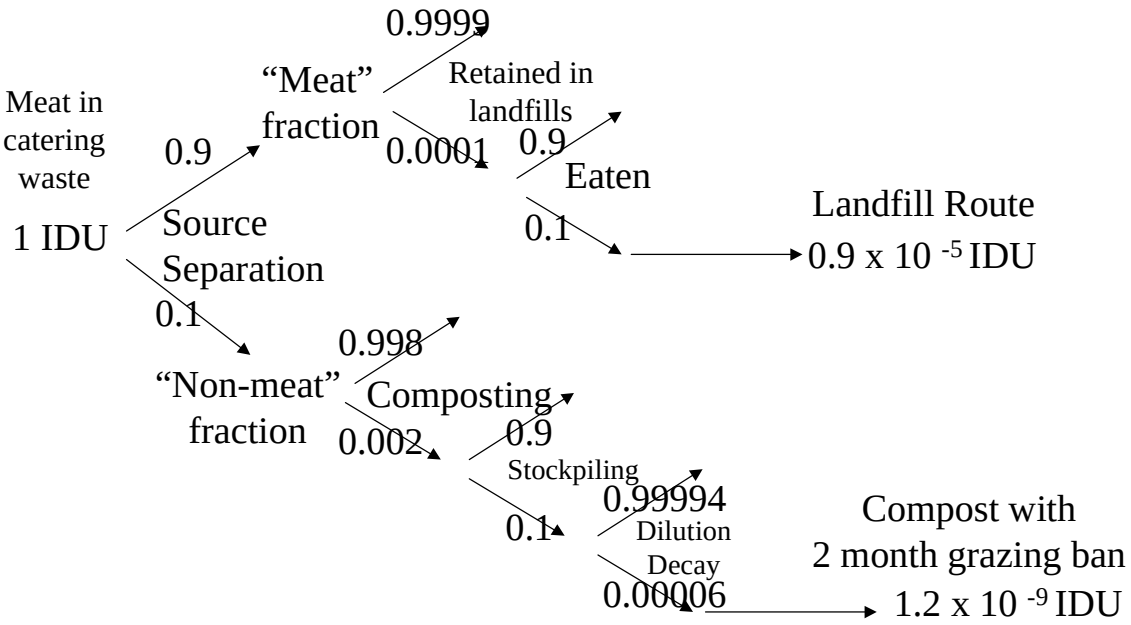

Figure 9.1 Prototype event tree for comparison of exposures to animals through landfill disposal and composting with a 2 month grazing ban. Note model assumes source separation, such that 90% of the meat goes to landfill.

9.5 Conclusions

No quantitative comparison can be made, because there are no data on the amount of raw meat removed from landfills. However, the composting route for catering waste *potentially* presents lower risks to grazing animals than disposal through land-fill. This is because composting offers extra control points. First, the raw catering waste will be delivered to enclosed receptions where birds and animals cannot gain access. Second, a no-grazing period can be enforced after application of the compost to land.

10. Consideration Of Modes Of By-Pass For Composted Catering Waste To Land

10.1 Implication of "by-pass"

The composting multiple barriers schemes set out in **Figure 8.1** will take out some 4.62-logs of pathogens and the biogas process multiple barriers will take out 4.42-logs (**Figure 8.4**).

However, these multiple barriers processes will be "in vain" if raw material were to "by-pass" the composting process. Thus 1% by-pass of the 4.62-log multiple barrier process set up in **Figure 8.1** reduces the net removal from 4.62-logs to just 2.0-logs (**Table 10.1**), i.e. the relative risks are increased over 400-fold.

Table 10.1 Effect of "within-batch" and "between-batch" variation (e.g. from short-circuiting and dead spaces in a digester) on the net destruction of pathogens.

Treatment conditions	% by-passing treatment and receiving 0-log destruction	Arithmetic Mean Survival	Net log destruction
0-log destruction (100%)	0%	1	0.00
2-log destruction (100%)	0%	0.01	2.00
4.62-log destruction (100%)	0%	2.4×10^{-5}	4.62
4.62-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

The multiple barriers process of source separation, composting catering waste, together with a no grazing ban (allowing dilution and decay) will reduce the risks to grazing animals by almost 9-logs (**Figure 10.1**).

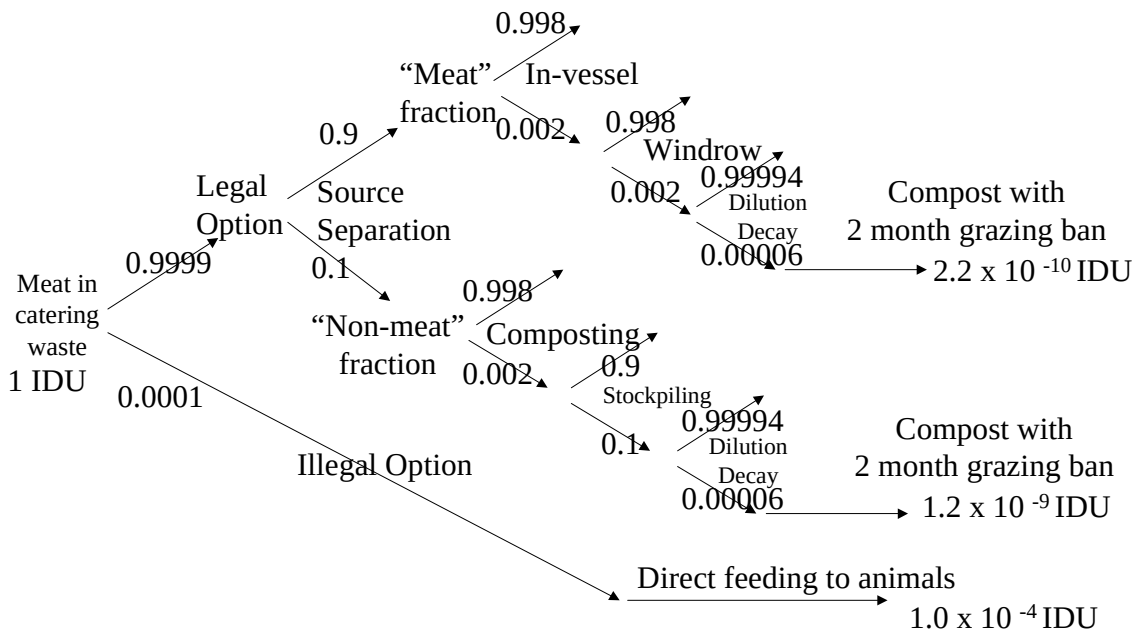

Figure 10.1 The effect of "by-pass"

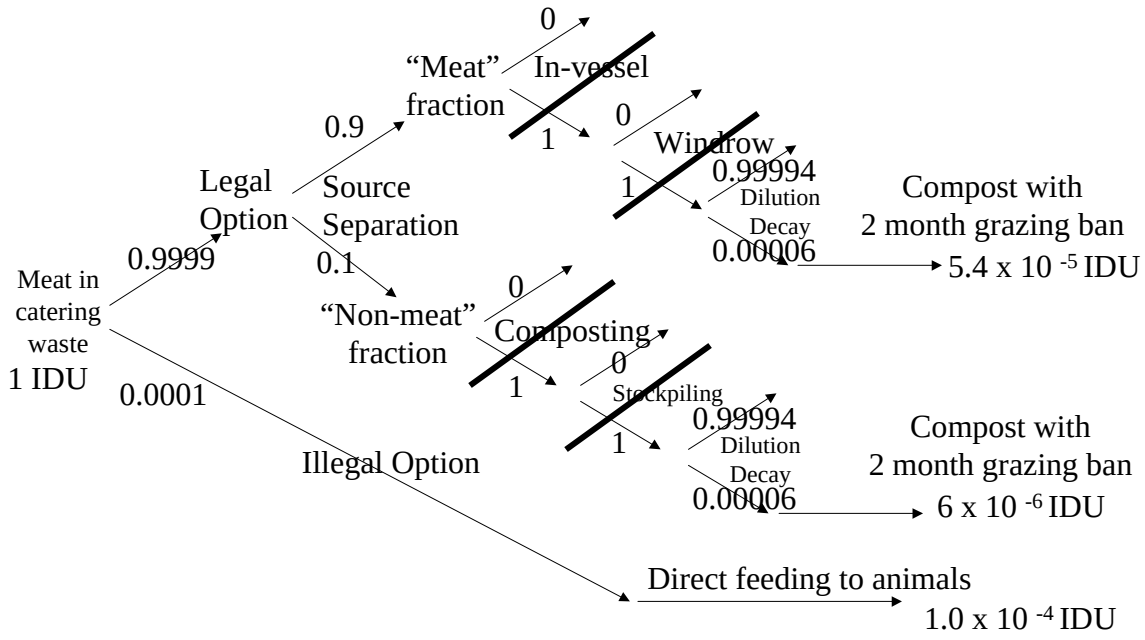

Figure 10.2 Applying 0.01% of the raw meat in catering waste directly to land with no grazing ban would "cancel out" the benefits of the composting processes. This is analogous to the illegal action of feeding catering waste directly to pigs.

A farmer breaking the law and feeding raw catering waste to pigs would by-pass the 9-log process (set out in **Figure 10.1**) comprised of the multiple barriers of source

separation/composting and dilution/decay in the soil (from the 2 month no grazing period). If just 0.01% of the meat in catering waste in the UK were applied direct to land (with no 2 month grazing ban), the risks of an FMD or CSF case would increase by some 100,000-fold. This represents feeding catering waste direct to pigs and would in effect remove the benefits of applying the composting and biogas processes set out in **Figure 8.1** and **Figure 8.4**, respectively. This is shown in **Figure 10.1**. The net reduction in risks across the UK by implementing a multiple barriers composting process with a grazing ban would no longer be 8.85-logs but just 3.79-logs. The "take home" message is that, if just a small proportion of catering waste were illegally fed directly to pigs, then there would be no point in the rest of UK composting the waste. Simply tilling all catering waste produced in the UK into the land and applying the 2 month grazing ban would not present a higher risk. This is apparent in **Figure 10.2**.

10.2 By-pass of the composting process

- Within-batch and between-batch variation of the composting process (see **Section 6.5**);

- A vector (small mammal, bird) moves a piece of raw meat from the raw waste to the adjacent pig field – or to the treated material. Raw material must not be accessible to rats and birds; and

- Cross-contamination by tools, loading vehicles and transport lorries, depends on management of the processes. A potential mechanism would be a lorry delivering a load of catering waste from which juices had leaked. The same lorry, after delivering the raw material, is used to transport "final-product" compost off site.

10.3 Direct feeding of catering waste to animals as swill - complete by-pass of the whole process

This process is banned in the UK. However, a survey of catering outlets by WRc-NSF identified one restaurant from which a farmer used to take the waste food directly from the premises. Although the proprietor confirmed that this practice had ceased, it cannot be ruled as completely eliminated in the UK.

Composting of catering waste should not be practised on livestock farms. An example of complete by-pass would be through pigs gaining access to the raw material.

10.4 Direct ingestion of compost by animals – by-pass of decay and dilution on soil

If compost were delivered to a farm premises for use, then accidental ingestion of the compost by pigs would represent a by-pass of the dilution and decay barriers in the soil.

11. A Summary Of Key Assumptions For The Risk Assessment

Model considers England and Wales as one big field comprising some 9.5 million ha of land (**Table 4.2**) to which compost could be applied. In total 500,000 tonnes of compost are applied to 50,000 ha of this land each year (rate of application = 10 tds ha^{-1}). Assuming pigs, cattle, and sheep graze or are housed randomly over this 9.5 million ha, then 0.52% of the total animals in England and Wales would be exposed to land to which compost had been applied.

11.1 Barriers

The main barriers are:-

- Cooking/Consumption of food
- Exclusion of Meat at Source
- Composting
- Decay in soil over 2 months
- Dilution in soil

11.1.1 BSE and Scrapie

Dorsal root ganglia (DRG) are the main source of (any) BSE agent potentially in catering waste. The model assumes 90% of DRG (and hence any BSE agent) in food enter the catering waste, with no destruction in compost and no decay on soil.

Model assumes 100% of spinal cord in lamb chops, and 1% of lamb/mutton goes to catering waste. Model assumes that composting does not destroy any scrapie prion infectivity, and that there is no decay in the soil.

Models assume no source separation of meat.

11.1.2 Exotic viruses, trichonellas, protozoa and vegetative bacterial pathogens

Source Term focuses on illegal importation on infected pork for exotic viruses and trichenellas. Model assumes 1% of meat is uncooked and discarded.

The model allows for a credit system such that at least 4.7-logs (i.e. a 50,000-fold) reduction occurs through Source Separation and Composting. This is set out in **Table 11.1**.

Bendixen and Ammendrup (1992) consider that in Denmark, not more than 95% of the material will be properly separated in most municipalities. In some places the separation schemes may be even less effectively implemented. The model therefore allows for a 1-

log reduction by source separation, i.e. 90% of the meat is removed by the process (**Table 11.1**).

Table 11.1 A credit system for the barriers

Process (Barrier)	Credits (log-reduction)
Source separation	1.0
Composting process (Table 6.16)	2.7
Stock-piling	1.0
Total	4.7

11.2 Policies

These policies are designed to impact on the potential risks from the exotic viruses (and in particular FMD and CSF)

1. Compost containing catering waste is ploughed in to a depth of 10 cm.

2. A time interval of 2 months is required between application of composted catering waste to land and the grazing or housing of farm animals.

3. Composted catering waste should not be stored on premises where farm animals are kept.

11.3 Modelling these policies in the risk assessment

Linear extrapolation of decay data for FMDV and CSFV in pig slurry (4ºC) collected over an 84 day period would suggest a massive 17.7–log decay over a one year period. For ASFV the extrapolated decay rate would be 10.8-logs. However, it would be inappropriately optimistic to allow such destruction rates in the model even over a period of one year. This is because there appears to be a more resistant sub-population of virus (Prof. A. Donaldson, pers. comm.). Haug (1993) discusses this further in relation to aggregate size of microorganisms. Indeed, Plowright and Parker (1967) demonstrated a rapid 5-log die-off of ASFV followed by a period of slower decline in temperature inactivation experiments (see **Section 6.3.4**).

Thus, the risk assessment model assumes a time-dependent 5-log decay of FMDV, CSFV and ASFV on land, after which the model assumes no further decay. This is illustrated for FMDV in **Figure 11.1**. In the case of SVDV, no decay on land is allowed for.

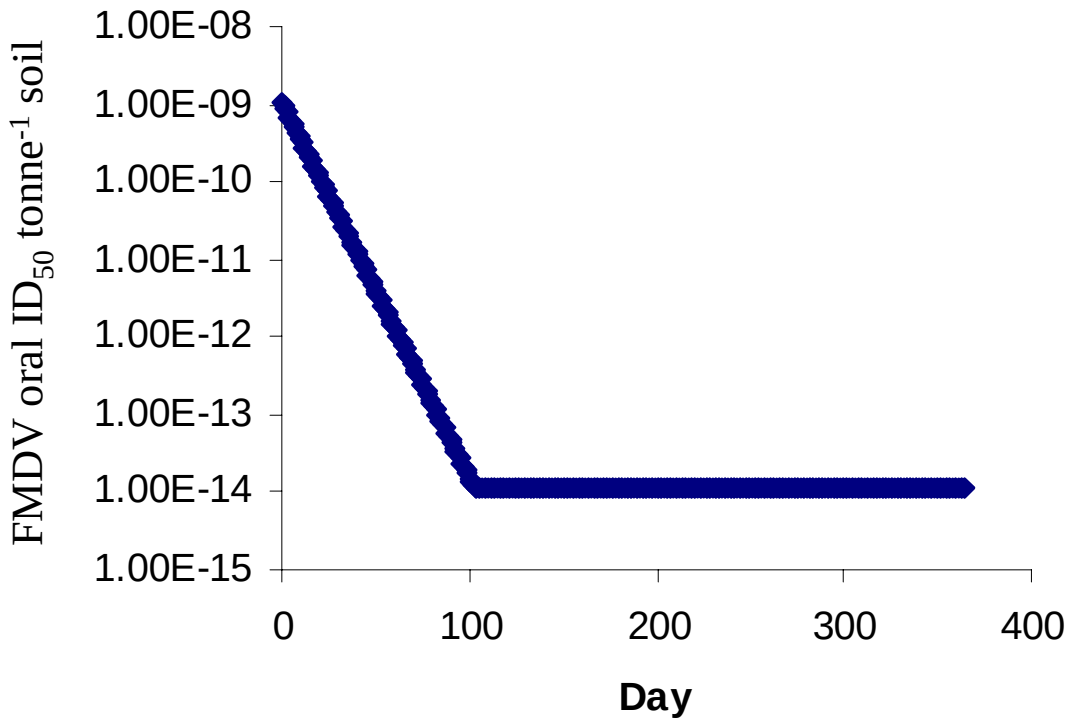

Figure 11.1 Decay of FMDV in soil with time according to Equation 8. Concentrations based on a single FMDV-infected pig carcass entering the food chain.

11.3.1 Modelling accidental grazing of animals on land to which compost has been applied

Laws will undoubtedly be broken albeit unintentionally and by accident in most cases.

In this risk assessment, accidental grazing is only quantified for scenarios where a 1 year (as opposed to 2 month) grazing ban is implemented. This is done by assuming that during the one year no grazing period, virus decays according to **Figure 11.1** and that 1% of the 0.52% of grazing animals in the vicinity of compost-amended fields stray onto the land for a period of 7 days in that first year.

Accidental grazing is not considered in the context of the two month ban.

11.3.2 Modelling accidental and direct ingestion of composted catering waste.

This is not formally covered in the risk assessment.

11.4 Dose-response – estimating the risk of infection from the exposure

Dose response curves are available for some human pathogens (such as salmonellas, campylobacters and VTEC). The predicted arithmetic mean exposure is therefore used directly in those dose-response curves to give a risk of infection.

For many animal viruses, full dose response curves are not available and exposures are calculated in terms of ID_{50} units (typically sub-fractions of an ID_{50} unit). This is the case for TSEs and the exotic pig viruses.

Sutmoller and Vose (1997) consider pathogen doses required to initiate infection, with FMDV as an example. Using a mathematical approach they demonstrate that the risk from exposure to a sub-fraction (x) of an ID_{50} may be calculated as $0.69x$. This was demonstrated by Gale (1998) for BSE agent and is the approach used by Gale and Stanfield (2001) to model the risks of BSE infection through sewage sludge applied to land. It is also the approach adopted here. In effect this approach assumes that the negative exponential (single hit) model is applicable to describe the dose-response relationship

Equation 7 $$p = 1 - e^{-rN}$$

i.e. that pathogens act independently and there is a single and constant parameter r describing the infectivity of the particular pathogen in a specific host. In fact, r is the risk from a dose N of one pathogen (Gale 2001).

12. Bovine Spongiform Encepathalopathy (BSE)

This risk assessment included the risks from BSE in UK beef products and from beef products imported from abroad.

12.1 Source Term

12.1.1 Incidence of BSE infection

The BSE epidemic in the UK is declining in line with forecasts, with confirmed cases in 2001 being 40.4% lower than in 2000. To April 2002, there were 17 BSE cases in the UK born in 1996, of which just one had been born after the August 1996 feed ban.

UK cattle under 30 months of age

Professor Anderson of Imperial College reported to SEAC on 28 November 2000 (www.defra.gov.uk/animalh/bse/bse-science/seac/seac1100.html) that the estimate of the number of animal entering the food chain in the UK within 12 months of developing clinical disease was 0.8 animals in 2000 and 0.5 in 2001. This assumed a 10% maternal transmission, and a 60% reduction in maternal transmission cases due to the Offspring Cull.

BSE in sheep in UK

Kao *et al*. (2001) have recently undertaken a risk assessment to model the number of BSE cases in sheep. Their analysis indicates that at the peak of the BSE epidemic in 1990, the number of cases of BSE-infected sheep would have ranged from fewer than 10 to about 1,500. The model predicts that in 2001 there would have been fewer than 20 clinical cases of BSE in sheep if maternal transmission occurred at a rate of 10%.

BSE epidemiology in other countries

The numbers of BSE cases reported in EU member state countries such as France and Portugal have risen sharply, although are still less than in the UK. The number of cases in EU member state countries are presented in **Table 12.1**.

Table 12.1 BSE in EU countries. Data for 2001 from DEFRA

Member state	Suspects	Surveillance	Total
Belgium	9	37	46
Denmark	1	5	6
Germany	7	118	125
Spain	9	74	83
France	91	186	277
Ireland	123	123	246
Italy	0	50	50
Luxemburg	0	0	0
Netherlands	3	17	20
Portugal	62	51	113
Finland	0	1	1
UK	781	376	1,157

12.2 Pathways and barriers

12.2.1 Barriers at UK abattoirs (cattle under 30 months slaughtered for human food)

An event tree for partitioning of central nervous system material in cattle (under 30 months) slaughtered at UK abattoirs into catering waste is presented in **Figure 12.1**. The model considers brain, spinal cord and the dorsal root ganglia. For the purpose of **Figure 12.1** (and **Figure 12.2**) it is assumed that the brain weigh 500 g, the spinal cord 200 g and the DRG weigh 30 g in total.

Spinal cord

Specific Risk Material (SRM) removal rules safeguard public health. Thus all spinal cord is removed from all cattle carcasses slaughtered for human consumption. All carcasses are inspected by the Meat Hygiene Service. One case of a carcass with spinal cord remaining has been reported. The model assumes that spinal cord remains in 1 in 1,000,000 carcasses (**Figure 12.1**).

Dorsal Root Ganglia

The dorsal root ganglia (DRG) are not removed with the spinal cord but remain attached to the vertebral column. Most DRG come out with the bone (vertebral column) which since August 1997 has been treated as SBM (or SRM). Thus, “T-bone” steaks, and “Rib of beef” purchased with bone-in would contain DRG.

There are 60 DRG, one on each side of the 30 vertebrae. The question to address is what proportion of the meat cuts along the length of the vertebral column are sold to butchers, catering outlets and households with “bone-in”. DNV (1997) report that 52% of fore-ribs go to retail butchers and catering outlets with the bone-in. Similarly, 13% of sirloins from cattle slaughtered are “bone-in” and sold as T-bone steaks (**Table 12.2**). Bone is removed from the neck, chump and rump.

Table 12.2 Fate of DRG in cattle carcasses (Data taken for Figure 3.1 of DNV (1997).

Type of cut of Meat	Percentage of vertebral column	Percentage sold with bone-in to caterers and butchers	Percentage of vertebral column sold with bone-in
Neck/chump/rump Fore-ribs Sirloin	57% 13% 30%	0% 52% 13%	0% 6.8% 3.9%
Total			10.7%

In total therefore, 10.7% of the DRG from cattle enter the food chain. It is most unlikely that the DRG would be removed from the bone and eaten, either in restaurants (e.g., a carvery) on in a household waste (DNV 1997). It is assumed here therefore that 90% of the bone remains of "bone-in" beef products containing the DRG are discarded to the "catering waste" bin (**Figure 12.1**).

The 60 DRG per carcass weigh 30 g in total (DNV 1997).

Brain material

For **Figure 12.1** (and **Figure 12.2**), it is assumed that the bovine brain weighs 500g of which 499 g go to SRM and the remaining 1g of brain in contaminates 0.01% of cattle carcasses. In the other 99.99% of carcasses this 1 g goes to SRM along with the other 499 g of brain.

Total CNS loading in food chain

The total annual loading in the food chain from the 2.43 million cattle under 30 months slaughtered for human consumption is calculated in **Table 12.3**.

In total some 7.8 tonnes of CNS material form cattle under 30 months old enter the human food chain annually. This is 0.42% of the total brain/spinal cord and comprises almost entirely the DRG.

Table 12.3 Fate of CNS in under 30 month cattle slaughtered at UK abattoirs

Tissue	Not removed: Annual Frequency	Weight per carcass	Total loading in food chain
Spinal cord	10^{-6}	200g	$2.43 \times 10^{6} \times 10^{-6} \times 200$ g = 486 g
DRG	0.107	30 g	$2.43 \times 10^{6} \times 0.107 \times 30$ g = 7.8 tonnes
Brain	10^{-4}	1 g	$2.43 \times 10^{6} \times 10^{-4} \times 1$ g = 243 g
Total			7.8 tonnes

An event tree for partitioning into catering waste of bovine CNS material from UK cattle under 30 months slaughtered at UK abattoirs is set out in **Figure 12.1**. The model assumes that 90% of DRG are not eaten and go to the catering waste. The remaining 10% are either made into stock or eaten by pets. In addition, 20% of any brain material "contaminating" the carcass and 50% of any spinal cord is not eaten and goes to the catering waste bin.

In total 7.02 tonnes of CNS (predominantly DRG) enter the catering waste bin per year.

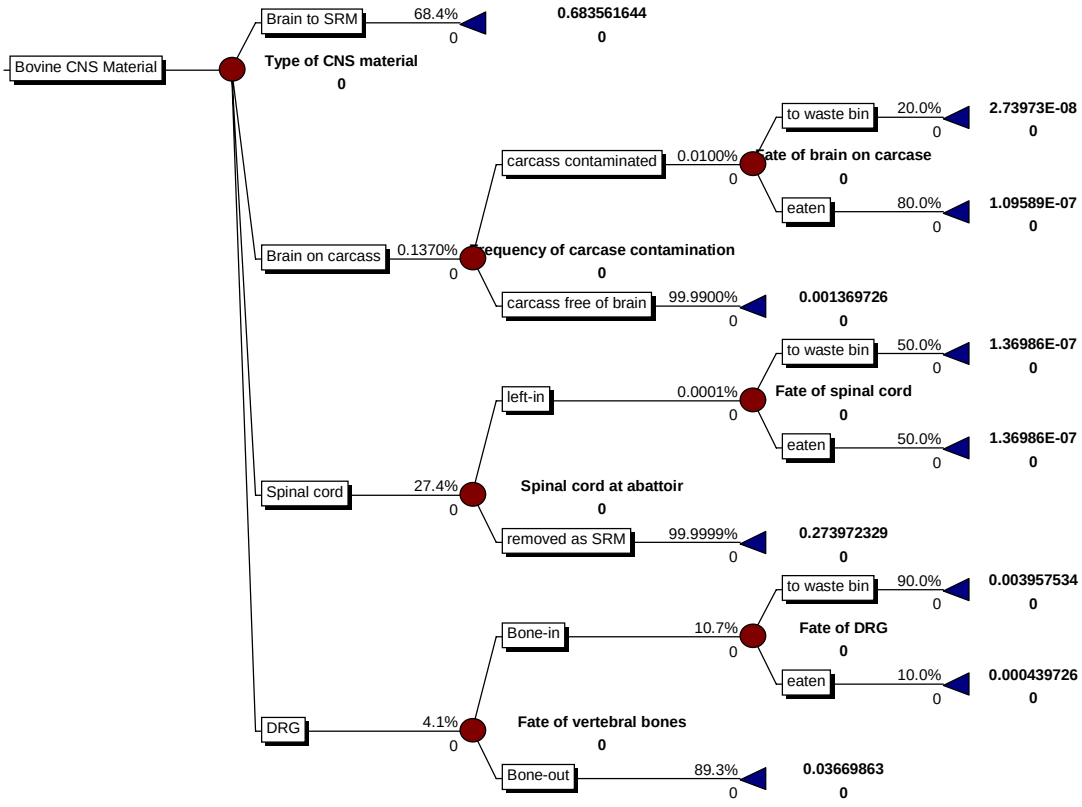

Figure 12.1 Event tree for partitioning of central nervous system material from cattle (under 30 months of age) slaughtered at UK abattoirs into catering waste.

12.2.2 Barriers for imported meats

Imported carcasses

According to MLC figures, 202,000 tonnes of beef carcasses were imported into the UK in 2000. On the basis that carcass meat from a single bovine weighs 0.3026 tonnes (calculated from MLC figures), then 202,000 tonnes of carcasses is equivalent to 667,600 head of cattle. An event tree is presented in **Figure 12.2**.

The OTMS rules apply to imported carcasses. In EU countries the SRM controls apply. Member states check that the SRM rules applied. For imported carcasses, the MHS check out the cutting plants. One or two carcasses in a shipment may fail. However, the

amount of spinal cord is small, and usually restricted to a few grams in a bit of the neck. Thus >90% of the spinal cord is taken out from all carcasses. The model therefore assumes that 20 g of spinal cord from around the neck remains in 0.1% of carcasses (**Figure 12.2**). In addition, the model assumes that the whole spinal cord is not removed in 0.0001% of carcasses (as for the UK). This is a worst-case assumption. However, it does not significantly affect the outcome. Indeed, the model demonstrates that the main component of central nervous system tissue is from DRG (**Table 12.4**). The model assumes that 90% of DRG, 20% of brain and 50% of any spinal cord in the food go to the catering waste bin. This is 1.94 tonnes of CNS material per year.

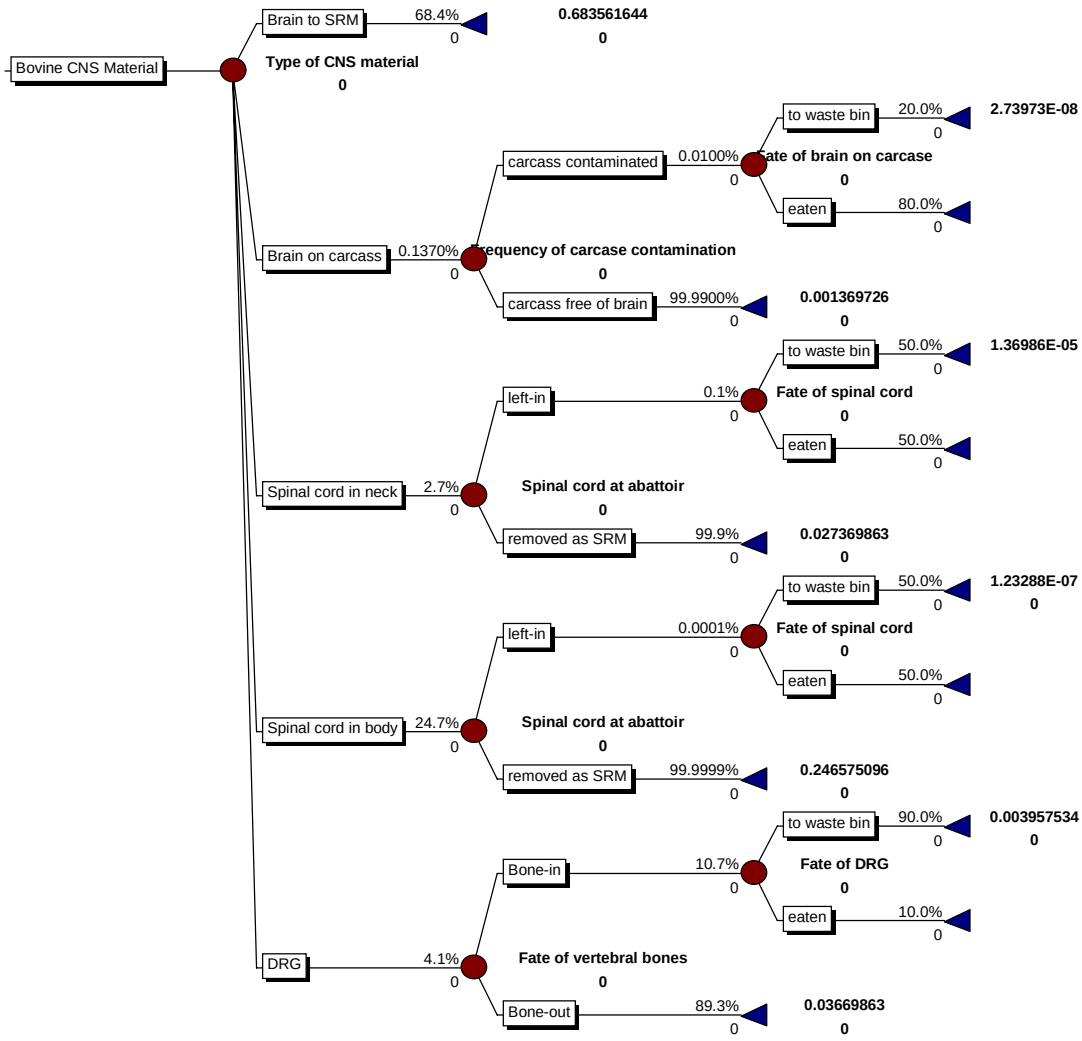

Figure 12.2 Event tree for partitioning of central nervous system material from cattle slaughtered at foreign abattoirs and imported carcasses

Table 12.4 Fate of CNS in carcasses of under 30 month cattle slaughtered abroad and imported into the UK

Tissue	Not removed: Annual Frequency	Weight per carcass	Total loading in food chain
Spinal cord in neck	10^{-3}	20g	$6.68 \times 10^{5} \times 10^{-3} \times 20$ g = 13.3 kg
Whole spinal cord	10^{-6}	200 g	$6.68 \times 10^{5} \times 10^{-6} \times 200$ g = 134 g
DRG	0.107	30 g	$6.68 \times 10^{5} \times 0.107 \times 30$ g = 2.15 tonnes
Brain	10^{-4}	1 g	$6.68 \times 10^{5} \times 10^{-4} \times 1$ g = 67 g
Total			2.15 tonnes

Processed meat products from abroad

The OTMS rules do not apply to processed meats imported from abroad. Furthermore, the head meat can be included in processed products in Europe, where just the brain, eyes and skull parts of the head are included as SRM. There is no measure of the level of contamination in products such as pies and sausages.

An event tree is set out in **Figure 12.3** for contamination of processed meat with brain material.

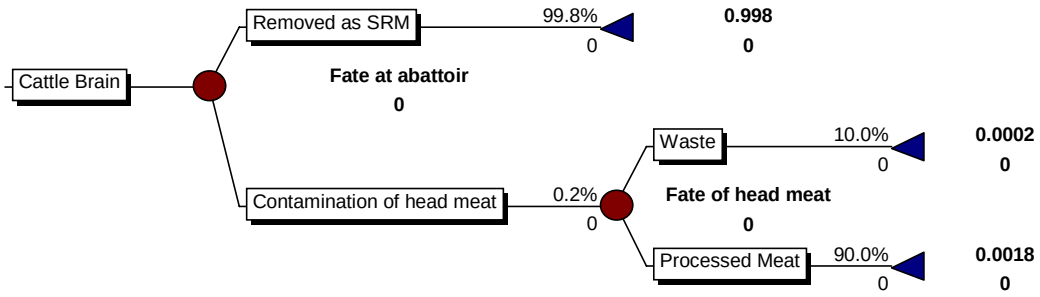

Figure 12.3 Event tree for contamination of processed meat with brain material in foreign abattoirs

A total of 105,000 tonnes of processed meat was imported into the UK in 2000 (data from MLC). If this were carcass meat (on the basis that each a carcass weighs 0.3026 tonnes), then this quantity would be equivalent to 347,000 carcasses. The model assumes for the sake of argument that 0.2% (i.e. 1 g) of bovine brain contaminates the head meat on each and every head and enters the processed food. This is clearly a worst case assumption. Assuming 10% of processed meat (**Figure 12.3**) is discarded to catering waste (either cooked or uncooked), then 0.02% of the brain (i.e. 0.1 g) from each carcass is present in the processed meat. This would be equivalent to 34,700 g

(i.e. 0.0347 tonnes) for 347,000 carcasses and is small (1.6%) in relation to the 2.15 tonnes of DRG in carcasses imported into the UK (**Table 12.4**). For this reason a quantitative risk assessment is not undertaken.

Without definite information on how many bovine carcasses contribute head meat to the 105,000 tonnes of processed meat which is imported, and how much contamination of head meat with brain occurs in foreign abattoirs, it is impossible to estimate with accuracy the potential contamination with brain.

12.3 Risk Assessment

12.3.1 Exposure to cattle grazing on pasture to which composted catering waste residue has been applied.

It is assumed that 1 in 2.43 million cattle under 30 months of age is within 12 months of developing BSE. The probability therefore that any CNS material in the food chain (mainly DRG) is from that one animal is 1 in 2.43 million, i.e. 0.00004%. Of the 7.02 tonnes of DRG in the catering waste bin each year from under 30 month cattle (**Section 12.2.1**), just 2.88 g of CNS would be expected from that one infected animal. (This can also be calculated on the basis that 10.7% (**Table 12.2**) of the 30 g of DRG (i.e. 0.107 x 30 g = 3.21 g) in that one infected cow enter the food chain. Of this 90% (i.e. 0.9 x 3.21 g= 2.88 g) is discarded to catering waste. In this respect the number of cattle slaughtered annually is irrelevant). Assuming the bovine oral ID_{50} is 0.1 g of BSE-infected bovine CNS material (DNV 1997; Gale and Stanfield 2001), 2.88 g of bovine CNS would be equivalent to 28.8 bovine oral ID_{50} units. On the basis that 500,000 tonnes of catering waste is composted each year, the concentration would be 5.8×10^{-5} bovine oral ID_{50} $tonne^{-1}$.

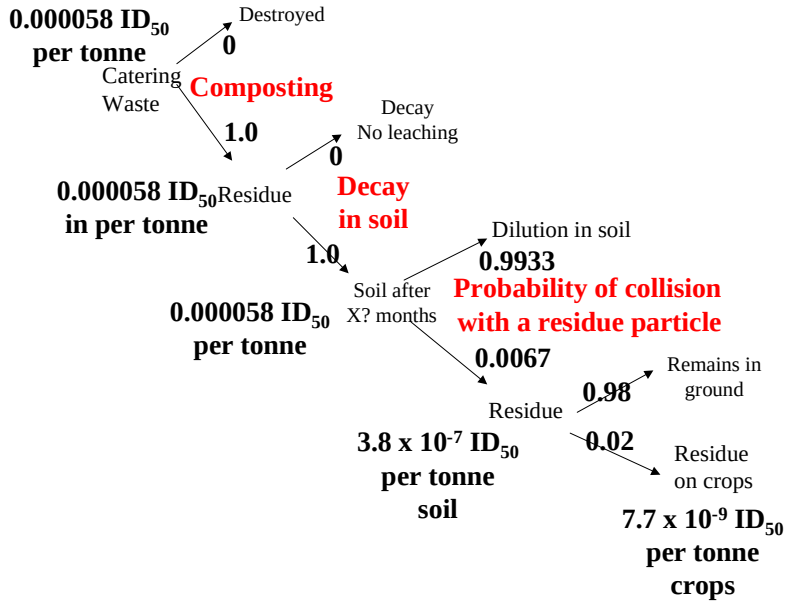

Figure 12.4 Event tree for transmission of BSE infectivity (bovine oral ID_{50} units) to root crops through application of composted catering waste residues to agricultural land.

Figure 12.4 outlines an event tree to model concentration of BSE in soil and the concentration on root crops at point of harvest. It is assumed that composting has no effect on the BSE agent and that there is no decay in the soil (as indeed set out by Gale and Stanfield 2001). After 150-fold dilution in the soil (**Figure 4.2**), the model predicts an arithmetic mean concentration of 3.8×10^{-7} bovine oral ID_{50} units $tonne^{-1}$ soil. Cattle ingesting 0.41 kg cow^{-1} day^{-1} are therefore exposed to 5.76×10^{-8} bovine oral ID_{50} cow^{-1} $year^{-1}$. Assuming a linear dose-response relationship (Gale 1998) this exposure translates into a risk of 4.0×10^{-8} cow^{-1} $year^{-1}$ (**Section 11.4**). This is considerably lower than the risk of 7.1×10^{-5} cow^{-1} $year^{-1}$ predicted for cattle grazing on farm land to which treated sewage sludge had been applied (Gale and Stanfield 2001).

The total tillage and grass area in England and Wales is 9.5 million hectares (Anon 1997). Since in the model 500,000 composted catering waste is applied to 50,000 ha (10 tonnes ha^{-1}), only 0.52% of the total tillage and grass land is affected. Assuming cattle graze at random over the 9.5 million ha of tillage and grass land, then only 0.52% of the England/Wales cattle herd will be exposed. There are 8.17 million cattle in England and Wales, of which 0.52% is 42,800 head (**Table 4.3**). Therefore the expected number of BSE cases on exposing 42,800 cows to a risk of 4.0×10^{-8} cow^{-1} $year^{-1}$ is 0.0017 cases $year^{-1}$.

Since it is assumed that one BSE infected carcass is also imported (**Section 12.2.2**), the risks through compost are the same from imported carcasses as from the UK cattle (**Table 12.5**).

Table 12.5 Summary of risks of BSE transmission to cattle in England/Wales from application of composted catering waste.

Source	Individual Risk (cow^{-1} $year^{-1}$)	Number of BSE cases in UK per year
UK under 30 month cattle	4.0×10^{-8}	0.0017
Imported carcasses	4.0×10^{-8}	0.0017
Imported processed meats	Not estimated	Not estimated
Total		0.0034

12.3.2 Exposure to humans ingesting root crops

Humans ingesting 0.384 kg root crops $person^{-1}$ day^{-1} (EUSES 1997) of which 0.02% (w/w)) is soil ingest a total of 2.8 kg soil $person^{-1}$ $year^{-1}$. Assuming the human oral ID_{50} is 1g of BSE-infected bovine brain, then there are 3.8×10^{-8} human oral ID_{50} $tonne^{-1}$ soil (**Figure 12.4**). The arithmetic mean exposure to a human through ingestion of 2.8 kg soil is 1.1×10^{-10} human oral ID_{50} $person^{-1}$ $year^{-1}$. This translates into a risk of 0.7×10^{-10} $person^{-1}$ $year^{-1}$. This is remote and would clearly be reduced still further by washing the soil off the crops prior to consumption.

12.4 Conclusion

It is concluded that the risks both to grazing cattle and to humans consuming crops grown on soil to which compost has been applied are remote.

A tonne of compost (according to the model) contains 5.8×10^{-6} human oral ID_{50}s. A gardener ingesting a gram of compost would therefore ingest 5.8×10^{-12} human oral ID_{50}s and be exposed to a risk of 4.0×10^{-12} $person^{-1}$ g^{-1}.

13. Scrapie

The risk assessment for scrapie is fundamentally different from that for BSE (**Section 12**) for several reasons:-

- scrapie infectivity occurs in a wide variety of tissues, many of which are consumed by humans. In the case of BSE, infectivity is localised to the brain and spinal cord and DRG (Anon 1994), the majority of which is not consumed by humans.
- Scrapie can be transmitted horizontally from affected to unaffected sheep with no inoculation of infected tissue. There is evidence that transmission can occur through contaminated environments.
- Scrapie has so far not been shown to affect humans, unlike BSE.

13.1 Source Term

Much of the data used here for the Source Term are those presented by DNV (2002).

13.1.1 Tissue distribution

Titres of scrapie infectivity for various sheep tissues are presented in **Table 13.1** as reported in Anon (1994). The titres presented in Anon (1994) are those obtained by intra-cerebral challenge.

Table 13.1 Infectivity titres (bioassay in mice) in tissues from up to 9 Suffolk sheep (34-57 months old). Data from Anon (1994).

Tissue	Log_{10} mouse intracerebral ID_{50} g^{-1} tissue	Oral ID_{50} g^{-1} tissue - assumes 1 intragastric ID_{50} = 10^5 intracerebral ID_{50} (Anon 1994)
Brain	5.6	4.0
Spinal cord	5.4	2.5
Lymph nodes	4.2	0.16
Spleen	4.5	0.32
Tonsil	4.2	0.16
Stomach	2	0.001
Liver	2	0.001
Thymus	2	0.001
Heart	1	0.0001
Kidney	1	0.0001
Intestine	4.7	0.5

Through the oral route, scrapie infected tissues are about 100,000-fold less infectious (Anon 1994). On this basis, oral ID_{50}s per g of tissues are calculated in **Table 13.1**. For example, there are $10^{5.6}$ intracerebral (i.c.) ID_{50} g^{-1} of brain, which is equivalent to 4.0 oral ID_{50} g^{-1}. In effect, the oral ID_{50} for sheep is about 0.25 g scrapie-infected bovine

brain. This is similar to the 0.1 g of BSE-infected bovine brain assumed as the oral ID50 for cattle in the sewage sludge risk assessment (Gale and Stanfield 2001). It should be noted that the ileum has high levels of scrapie infectivity, with about 0.5 ovine oral ID_{50} g^{-1} (**Table 13.1**).

13.1.2 Distribution with age of affected animals.

During the progression of the disease, scrapie infectivity accumulates in different ovine tissues at different rates. An estimation of the percentage build-up of infectivity in infected animals of different ages is presented in **Table 13.2**. Multiplying these proportions by the titres (i.c.) in tissues of sheep with clinical symptoms (**Table 13.1**) gives the arithmetic titres of scrapie in tissues from sheep of different ages (**Table 13.3**).

Table 13.2 Distribution of scrapie infectivity in ovine tissues with age (taken from DNV, 2002).

Tissue	Lambs (< 6 months)	Lambs (>6 months)	Hogetts (1 – 2 years)	Cull ewes
Brain	0.01%	0.10%	10.00%	100.00%
Spinal cord	0.01%	0.10%	10.00%	100.00%
Lymph nodes	1.00%	10.00%	50.00%	100.00%
Spleen	1.00%	10.00%	50.00%	100.00%
Tonsil	1.00%	10.00%	50.00%	100.00%
Stomach	1.00%	10.00%	50.00%	100.00%
Liver	1.00%	10.00%	50.00%	100.00%
Thymus	1.00%	10.00%	50.00%	100.00%
Heart	1.00%	10.00%	50.00%	100.00%
Kidney	1.00%	10.00%	50.00%	100.00%
Intestine	1.00%	50.00%	50.00%	100.00%

Table 13.3 Arithmetic mean scrapie infectivity titres (intracerebral ID_{50} g^{-1}) estimated for ovine tissues with age of animal

Tissue	Lambs (< 6 months)	Lambs (>6 months)	Hogetts (1 – 2 years)	Cull ewes
Brain	39.8	398.1	39,810.7	398,107.2
Spinal cord	25.1	251.2	25,118.9	251,188.6
Lymph nodes	158.5	1584.9	7,924.5	15,848.9
Spleen	316.2	3162.3	15,811.4	31,622.8
Tonsil	158.5	1584.9	7,924.5	15,848.9
Stomach	1.0	10.0	50.0	100.0
Liver	1.0	10.0	50.0	100.0
Thymus	1.0	10.0	50.0	100.0
heart	0.1	1.0	5.0	10.0
kidney	0.1	1.0	5.0	10.0
Intestine	501.2	25059.4	25,059.4	50,118.7

13.1.3 Distribution of scrapie in ovine tissue used in food.

Masses of tissues in lamb are presented in **Table 13.4** together with the utilisation of these tissues in food. From this, the average weight of tissue per animal used in food may be calculated. Brain and spinal cord represent a relatively small proportion. The intestine appears to make the major contribution (**Table 13.4**). The major use of the intestine is in the preparation of natural casings for sausages. The small intestine is cleaned mechanically, removing the "Patches of Peyer". DNV (2002) in their risk assessment for the Food Standards Agency assumes that cleaning reduced the infectivity by a factor of 100-fold.

Table 13.4 Weights and utilisation of ovine tissues in food.

Tissue	Weight of tissues (g) for lamb	Utilisation in food (taken from DNV 2002).		Average weight per animal used in food (g)	
		Lamb	Mutton	Lamb	Mutton*
Brain	100	5%	0%	5	0
Spinal cord	40	20%	0%	8	0
Lymph nodes	40	100%	100%	40	64
Spleen	100	0%	0%	0	0
Tonsil	100	0%	0%	0	0
Stomach	1000	10%	10%	100	160
Liver	650	100%	100%	650	1,040
Thymus	50	100%	100%	50	80
Heart	200	50%	50%	100	160
Kidney	100	100%	100%	100	160
Intestine	1200	90%	90%	1080	1,728

*assumes weights of tissues in mutton are 1.6-fold greater than in lamb (MLC, pers comm).

Ovine oral ID_{50}s in lamb and mutton for food per infected animal are calculated in **Table 13.5**. According to the model, it is the lymph nodes and intestines which contribute the highest levels of scrapie infectivity to food.

13.1.4 Scrapie incidence in the National flock.

Several questionnaire surveys have been conducted in Britain. Between 17% and 34% of sheep farmers reported having seen at least one case of scrapie in their flock at some time, showing a considerable number of sheep flocks are affected. The within-flock incidence has been reported as 2% in Britain.

There are 500 to 600 cases reported in the breeding flock. It is estimated that only 13% of cases are reported. This would suggest approximately 4,500 cases in total in the breeding flock. This is 0.1% of the breeding flock.

Table 13.5 Predicted ovine oral ID_{50} in food per infected animal.

Tissue	Lamb		Mutton	
	<6-month	>6-month	1 – 2 year	Cull ewes
Brain	0.002	0.020	0	0
Spinal cord	0.002	0.020	0	0
Lymph nodes	0.063	0.63	5.1	10.14
Spleen	0	0	0	0
Tonsil	0	0	0	0
Stomach	0.001	0.01	0.08	0.16
Liver	0.0065	0.065	0.52	1.05
Thymus	0.0005	0.005	0.04	0.08
Heart	0.0001	0.001	0.008	0.016
Kidney	0.0001	0.001	0.008	0.016
Intestine*	0.054*	2.71*	4.33*	8.66*
Total	0.130	3.46	10.1	20.1

*assumes 100-fold reduction in infectivity during cleaning process (DNV 2002).

In the UK in 2000, some 2.42 million ewes and rams were slaughtered in addition to 15.96 million lambs. On the basis that 0.1% are infected, 2,420 ewes and 15,960 lambs would be infected with scrapie. Assuming all the lambs are in the >6 mth – 1 year age range (**Table 13.5**), the total infectivity in the food chain from lambs is 15,960 x 3.46 = 54,928 ovine oral ID_{50}s. For ewes this is calculated as 2,424 x 20.1 = 48,761 ovine oral ID_{50}s. The total scrapie infectivity in food is therefore 104,010 ovine oral ID_{50}s per year.

13.2 Pathways

The source term is 104,010 ovine oral ID_{50}s in food per year.

13.2.1 Fate in the kitchen

Unlike the DRG of cattle, most of the tissues (see **Table 13.1**) in lamb and mutton which contain scrapie agent are in the edible portion and would not tend to be selectively discarded to the catering waste bin. Cooking (e.g. by roasting) is likely to kill a considerable portion of the scrapie agent, although there are no data for this. Indeed, rendering destroying some 2.8-logs of scrapie agent (Taylor *et al.* 1997). The model assumes that 1% of the lamb/mutton sold for food in domestic kitchens and catering outlets is discarded into the bin uncooked.

This is equivalent to 1,040 ovine oral ID_{50} units $year^{-1}$.

Model allows for 100% of spinal cord in lamb chops to go to catering waste

It could be argued that spinal cord fragments in lamb chop portions are more likely to be discarded to the catering waste bin than other tissues, which are eaten. Infectivity in spinal cord in lambs <6 months and >6months accounts for 1.55% and 0.6%, respectively, of the total infectivity (**Table 13.5**). Assuming 100% of spinal cord in lamb chops is discarded to the catering waste bin, then the total loading would increase from 1,040 ovine oral ID_{50} units per year to 1,357 ovine oral ID_{50} units $year^{-1}$. This is the figure used in the risk assessment.

13.2.2 Effect of composting

It is assumed that composting has little destructive effect. Dilution of 1,357 ovine oral ID50 units into 500,000 tonnes of compost gives an arithmetic mean concentration is 0.0027 ovine oral ID_{50} $tonne^{-1}$ of composted catering waste.

13.2.3 Fate in the environment

As discussed in Gale & Stanfield (2001) it is not clear whether the 2 to 3-log decrease in scrapie infectivity in soil observed after 3 yr. by Brown & Gajdusek (1991) was due to decay or adsorption to the soil particles. For the purpose of risk assessment, therefore, the worst-case assumption of no decay on the land is applied. Dilution in the soil gives an arithmetic mean concentration of 1.8×10^{-5} ovine oral ID_{50} units $tonne^{-1}$ soil. The Brown and Gajdusek (1991) data demonstrate no leaching of scrapie agent into lower layers of soil.

13.3 Predicted risks

For the purposes of risk assessment, bovines are reported to ingest 0.41 kg soil cow^{-1} day^{-1} (EUSES 1997). It is assumed here that sheep and lambs ingest 0.2 kg soil $animal^{-1}$ day^{-1}. The annual exposure is therefore $0.0002 \times 365 \times 1.8 \times 10^{-5} = 1.3 \times 10^{-6}$ ovine oral ID_{50} animal $year^{-1}$. This translates into a risk of 0.9×10^{-6} $animal^{-1}$ $year^{-1}$ (Gale 1998). This risk is two orders of magnitude lower than that predicted for BSE in cattle grazing on land to which sewage sludge has been applied (Gale and Stanfield 2001).

The total number of sheep in England and Wales is 29.96 million (Anon 1997). Assuming that 0.52% of sheep flock graze of land to which composted catering waste has been applied, then 157,000 sheep are exposed to the risk of 0.9×10^{-6} $animal^{-1}$ $year^{-1}$. The model predicts that 0.14 sheep would be infected in England and Wales per year with scrapie from application of composted catering waste to land.

Sensitivity analysis – effect of intestine washing

Intestines contribute the highest loadings of scrapie infectivity to food (**Table 13.5**). The model assumes that 99% of scrapie infectivity is removed by washing the intestines. If this process is less effective and only removes 90%, then the model predicts 0.75 cases of scrapie in sheep in England and Wales through compost, i.e. a factor of 5-fold more than if washing had removed 99% of the infectivity. The risk of scrapie to sheep grazing on soil to which composted catering waste had been applied would be 4.8×10^{-6} $sheep^{-1}$ $year^{-1}$.

13.3.2 Summary of main assumptions for risk assessment

- 1% of lamb/mutton is discarded to catering waste;
- 100% of spinal cord in lamb chop is discarded to catering waste;
- cooking does not inactivate any scrapie agent; and

- composting has no effect.
- No decay on soil

13.3.3 Allowing for decay of scrapie agent in the soil

Brown and Gajdusek (1991) presented data to show that between 98.3% and 99.7% of scrapie agent was not recovered from soil after a period of 3-years' interment. Using the 98.3% (1.77-logs) decay in 3-years, this would equate to 0.59-log decay $year^{-1}$. In effect the scrapie loading on the soil would decrease by almost 4-fold each year.

On this basis allowing for a one year time interval prior to allowing sheep to graze on land to which compost had been applied would reduce the excess number of scrapie cases from application of compost to land to just 0.037 $year^{-1}$. The risks to individual sheep grazing on the compost-treated soil would be 2.3×10^{-7} sheep-1 $year^{-1}$.

13.4 Imported lamb

The world-wide distribution of scrapie is difficult to determine. Pathological examination of brain tissue or experimental transmission studies provide the only means of confirming infection. The stigma associated with the occurrence of disease means that that some farmers may be reluctant to report cases. Thus disease may remain undetected in some countries unless comprehensive surveillance systems are in operation.

Scrapie is known to be endemic in many European countries, such as Iceland, as it is in India and the USA. Australia and New Zealand did not import the disease with their original European breeding stock, and use vigorous importation and culling policies to remain free of the disease.

The annual within-flock incidence in Iceland is 3-5% and 1-10% in India. In some flocks in Iceland a within-flock incidence of 20% to 30% has been reported.

Data from the MLC for 2000 suggest that the proportion of imported sheep meat is about 25% of the total sheep meat used in the UK; there being 169,000 head of live imports and 123,000 tonnes of imported meat. Assuming each carcass weighs 20 kg, this is equivalent to 6.15 million head of lamb.

To undertake a quantitative risk assessment from imported sheep products would require accurate information on incidence of scrapie in the countries of origin. On the basis of the very low risks estimated for UK lambs and ewes it is felt that imported lamb would pose little risk.

14. Foot And Mouth Disease Virus

14.1 Epidemiology

Many outbreaks of FMD have been traced to waste food being fed to pigs. The 1967 UK outbreak was attributed to the importation of virus in bone marrow of sheep carcasses from South America.

A review of epidemics of FMD highlighted the important role which raw (untreated) milk can play in the spread of the disease in a country whose cattle are not vaccinated. The greatest risk is in the early stages of an outbreak, before the disease control measures have been implemented. Donaldson (1997) estimated that processed milk from an infected premises could have an FMDV titre of $10^{1.9}$ – $10^{2.9}$ ID_{50} per litre. Skimmed milk delivered by a single bulk milk tanker caused three outbreaks, all in pig farms (Henderson, 1969). Three possible mechanisms by which animals could be infected by contaminated milk have suggested (Sellers, 1971). These are:-

- Drinking the milk;
- Inhalation of infective droplets or aerosols of milk; and
- Contamination of people with milk, who then handle the animals.

When developing risk assessments, epidemiology should be drawn on for identifying new and potential routes (Gale 2001). In terms of composted catering waste, analogous routes would be:-

- Eating the compost (either directly or after application to land);
- Inhalation of compost residues
- Contamination of people with compost, who then handle animals

The latter two routes are deemed to be remote, and for the purpose of the risk assessment here only the first route is considered. Milk itself in catering waste is not considered as n issue because most is treated.

14.2 Source Term

14.2.1 Survival of the virus in animal tissues

In the carcasses of animals infected with FMD, the virus is rapidly inactivated in skeletal and heart muscle tissue as a result of the drop in pH that accompanies rigor mortis. The virus may persist for long periods in blood clots, bone marrow, lymph nodes and offals (kidney and liver) because these tissues are protected from the pH changes that accompany rigor mortis.

Inactivation in beef, offals, bone marrow and lymph nodes

Henderson and Brooksby (1948) studied the survival of FMDV in beef and beef offals after storage at temperatures employed in the imported-meat trade. The acidity of rigor mortis of muscular tissue rapidly causes inactivation (**Figure 14.1**). Quick –freezing of beef suspends acid formation and active virus was demonstrated for so long as the meat was kept frozen. Thawing of quick-frozen meat initiates the suspended lactic acid formation at an accelerated rate and rapidly produces a medium unsuitable for virus survival.

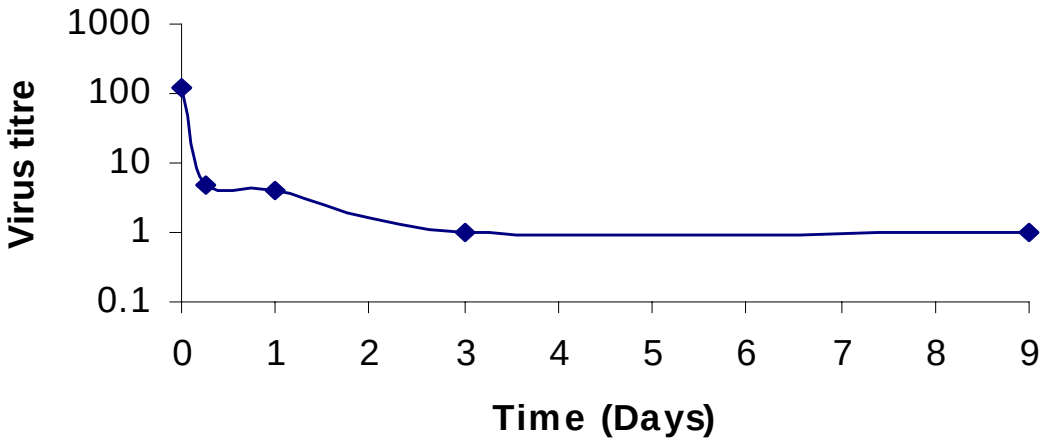

Figure 14.1 Rate of inactivation of FMDV in beef stored at 4°C. Data from Table 3 of Henderson and Brooksby (1948).

Liver, kidney, rumen, lymph node and blood from diseased cattle have all been shown to be highly infective and to remain so if stored frozen. Acid formation in these tissues and in blood is not on the same scale as in muscle, and prolonged survival of virus is more likely even with delay in freezing and after thawing. This remains true of lymph node and of residual blood in vessels of a carcass in which the development of rigor mortis is complete.

Table 14.1 pH values of imported meat and offal determined in a London cold-storage warehouse (from Henderson & Brooksby, 1948)

Tissue	pH
Beef (forequarter)	5.6
Lymph node	6.5
Beef (hindquarter)	5.4
Liver	5.9
Kidney	6.3
Tripe	6.3

The persistence of active FMDV in liver and kidney is favoured by the lack of acid production on a scale equal to that in muscle. Thus, when liver or kidney is stored

frozen, as in the imported-meat trade, it may be shown to have a high degree of infectivity at 4 or more months and the virus remains active for at least 24 h after thawing.

Rigor mortis causes drop in pH which gives inactivation within 48h at 4 °C. However in clotted blood, marrow and lymph nodes virus remains active for 4 months at 4 °C.

Data from Cottral (1969) for survival of FMDV in bone marrow and lymph nodes from infected cattle are presented in **Figure 14.2**. Survival is greater in the bone marrow, than in the lymph nodes. Indeed for bone marrow it takes about 6 months for a 2-log (99%) reduction.

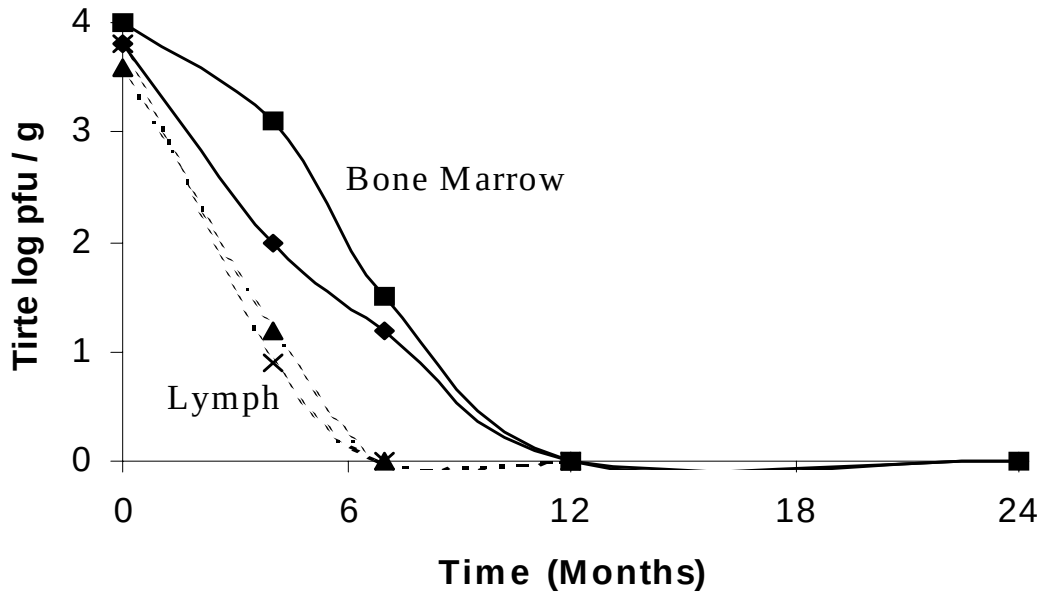

Figure 14.2 Survival of FMDV in tissues of infected cattle during storage at 1 to 4°C. Data from Cottral (1969).

The durations of survival for FMDV in various pork products listed below are taken directly from Farez and Morley (1997). Although there is no indication of the rate of decay (as for example in **Figure 14.2**), the data are of use in a qualitative sense.

- 30 days in different chilled organs such as lungs, stomach, tongue, intestine
- 24 hours in chilled spleen, liver and kidney
- 210 days in frozen lungs, intestine, stomach, tongue, kidney, spleen and liver
- 170 days in Parma hams
- 182 days in white Serrano hams
- 168 days in Iberian ham

- 112 in Iberian shoulder hams
- 42 days in Iberian loins
- 190 days in salted bacon and 183 days in ham fat
- 56 days in sausages
- 250 days in processed intestinal casings
- 7 days in salami
- 10 days in tongue and 1 day in muscle.

pH 6 gives 90% kill per minute and pH 5 90% every second.

14.2.2 Tissue loadings in infected animals

FMDV is distributed throughout the body of the infected animal and can be found in different concentrations for varying periods in the tissues. In pigs the greatest quantities of virus occur in the blood, epithelium, and liver.

The virus is excreted one to ten days before clinical signs appear, and continues for four to ten days.

Very high titres in heart ($10^{10.0}$ $TCID_{50}$ g^{-1}), lymph nodes ($10^{8.2}$ $TCID_{50}$ g^{-1}) and glands (10^{8} $TCID_{50}$ g^{-1}) have been reported (cited in MacDairmid 1991).

Sellers (1971) found high titres of $10^{7.2}$ pfu ml^{-1} blood, $10^{6.6}$ pfu g^{-1} in bone marrow and $10^{5.6}$ pfu g^{-1} in liver of infected pigs. High virus titres of $>10^{5}$ pfu/ml were detected by extraction from both fat and muscle tissue of infected pigs (Panina *et al* 1989). In contrast, Farez and Morley (1997) cited much lower levels in 62 pigs two days after intravenous inoculation (**Table 14.2**).

Table 14.2 Foot and mouth disease viral titres in tissues of 62 pigs two days after experimental infection. Data taken from Farez and Morley (1997).

Tissue	Titre (plaque forming units per ml or g)	
	Mean	Standard deviation
Blood	$10^{3.5}$	$10^{1.5}$
Lymph node	$10^{3.4}$	$10^{1.7}$
Bone Marrow	$10^{1.9}$	$10^{1.5}$
Fat	$10^{0.5}$	$10^{0.8}$
Muscle	$10^{0.03}$	$10^{0.2}$

Afzal and Barya (1968) reported $10^{6.6}$ mouse LD_{50} g^{-1} of tongue from experimentally-infected buffalo calves at 1 day post infection. This was the highest titre. Note that these are mouse LD_{50} and not $TCID_{50}$ or pfu.

Cooked meats are not a risk as long as heat treatment has been used (80-100 °C for 2-3 min or 60-70 °C for 25min). Cured hams and bacon need additional safeguards and cannot be regarded as being safe. Frozen beef must have reached pH 6 before freezing.

Milk

FMDV may be excreted in the milk of animals before clinical signs of the disease are apparent. Indeed, there are several examples of outbreaks in the UK attributed to the movement of infected milk:-

- The “Crewe episode” during the 1951-1952 epidemic in the UK. Feeding of infective milk in calves led to 101 new outbreaks.
- 22 outbreaks resulting from the collection of milk from 25 infective premises during the 1967-1968 outbreak in the UK.

The milk from cattle incubating FMD may contain virus for up to four days before vesicular signs of the disease become evident. Milk may contain up to $10^{6.6}$ $TCID_{50}$ per ml. During the Isle of Wight outbreak in 1981, the amount of virus in the bulk milk tank on the farm was $10^{2.2}$ $TCID_{50}$ per ml.

Survival of FMDV in milk is dependent on the temperature, bacterial content and pH.

14.2.3 FMD loadings in infected animals

The weights of the different pig by-products are presented in **Table 3.3**. FMD loadings in an infected pig are presented in **Table 14.3**. Values of $TCID_{50}$ are calculated on the assumption that 1 pfu equals 10 $TCID_{50}$ (Alex Donaldson, pers. comm.). Therefore in **Table 14.3**, $TCID_{50}$ values are calculated by multiplying the reported pfu counts by a factor of 10.

Heart

For muscle and heart, there will be a rapid increase of pyruvic acid and lactic acid resulting in a pH drop. Thus for the purpose of risk assessment it is assumed (using **Table 14.2**) that there are $10^{0.03}$ pfu g^{-1} (i.e. $10^{1.03}$ $TCID_{50}$ g^{-1}) of skeletal muscle and heart (**Table 14.3**).

Blood

Blood drained from the carcass contains the highest loading of infectivity (**Table 14.3**). For the purpose of risk assessment it is assumed that 5% of the blood with titres of $10^{8.2}$ $TCID_{50}$ ml^{-1} is retained in the carcass (e.g. in blood clots). It is assumed that blood within the muscle tissue has much lower loadings according to **Table 14.2** and that 10% of the carcass weight is blood in muscle.

Table 14.3 FMD loadings in an infected pig

Tissue	Weight (kg)	$TCID_{50}$ / g or / ml	Total loading in pig
Flare fat	1.00	$*10^{6.0}$	1.0×10^{9}
Kidneys	0.26	$*10^{6.6}$	1.0×10^{9}
Feet	2.00		0
Head, tongue	5.00		0
Gut contents	8.40		0
Intestinal fat	0.84	$*10^{6.0}$	8.4×10^{8}
Caul fat	0.11	$*10^{6.0}$	1.1×10^{8}
Intestines	2.70		0
Stomach (maw)	0.55		0
Heart	0.26	$*10^{1.03}$	2,780
Lungs	0.90		0
Trachea	0.04		0
Heart, lungs, trachea	1.20		0
Liver, gall bladder	1.50	$*10^{6.6}$	5.97×10^{9}
Pancreas	0.06		0
Spleen	0.11		0
Blood drained from carcass	3.40	$*10^{8.2}$	[d] 5.4×10^{11}
Cerebro-spinal fluid			0
Skirt	0.35		0
Hair scrapings & hooves	0.84		0
Bladder	0.04		0
Reproductive organs	0.15		0
Lymph nodes	0.04[c]	$10^{8.0}$	4.0×10^{9}
Waste	0.75		0
Bone marrow	5.464[a]	$*10^{7.6}$	2.1×10^{11}
Skeletal muscle	43.712[b]	$*10^{1.03}$	4.7×10^{5}
Blood in muscle	5.464[a]	$*10^{4.5}$	1.7×10^{8}
Total (bone –in)	(62.0)		[d] 2.6×10^{11}
Total (bone-out)	(56.6)		[d] 4.0×10^{10}

[a] assumes 10% of carcass weight (54.64 kg)
[b] assumes 80% of carcass weight (54.64 kg)
[c] value for sheep
[d] model assumes only 5% of high titre blood remains (i.e. 2.7×10^{10} $TCID_{50}$ $carcass^{-1}$) in the carcass (e.g. in blood clots)
*assumes 1 pfu = 10 $TCID_{50}$.

Bone marrow

For the purpose of risk assessment, it is assumed that bone marrow comprises 10% of the carcass weight (**Table 14.3**).

The model assumes that bone marrow comprises 10% of the weight of the carcass, and that skeletal muscle comprises 80% of the carcass weight. According to the model, blood and bone marrow comprise the major loadings of FMDV. In addition, the model assumes that 10% of the carcass weight is “blood in muscle”. The model assumes that

blood in muscle has $10^{4.5}$ $TCID_{50}$ ml^{-1} ($10^{3.5}$ pfu ml^{-1} in **Table 14.2**) on the basis that the low pH from lactic acid will reduce the loading considerably. This is lower that the $10^{7.2}$ pfu ml^{-1} (i.e. $10^{8.2}$ $TCID_{50}$ ml^{-1}) reported by Sellers (1971) which is used in the model (**Table 14.3**) for blood outside the skeletal muscle and which is presumably drained from the pig.

According to the calculation in **Table 14.3** there are 2.6 x 10^{11} $TCID_{50}$ units per porcine carcass with "bone-in". Of this almost 85% is in the bone marrow. Indeed with "bone-out" there are 4.0 x 10^{10} $TCID_{50}$ units per porcine carcass. This is mainly the 5% of clotted "high titre" blood.

14.3 Infectious Dose

14.3.1 Cattle

Donaldson (1997) reports that calves require a dose of $10^{6.0}$ $TCID_{50}$ by ingestion to initiate infection. Inhaling very small amounts of FMDV may infect cattle. 50% of cattle exposed experimentally to $10^{1.1}$ to $10^{2.6}$ $TCID_{50}$ of air-borne virus were infected.

14.3.2 Pigs

Pigs are approximately 1,000-fold less susceptible than cattle through the inhalation route (see Donaldson, 1997).

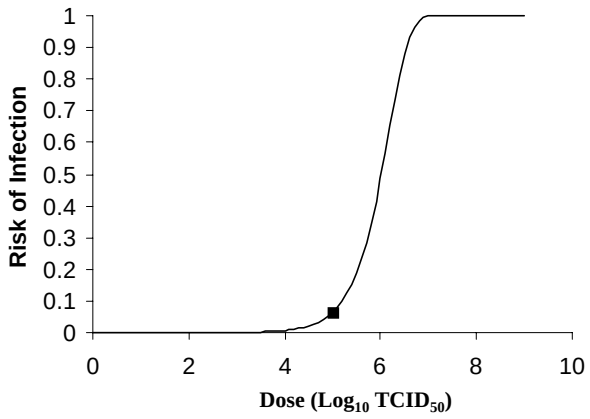

Figure 14.3 Dose-response curve for ingestion of FMDV by pigs. The oral ID_{50} is about $10^{6.0}$ $TCID_{50}$.

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. This point is plotted in **Figure 14.3**. Putting a negative exponential dose-response curve through this one point (which is acceptable) suggests the oral ID_{50} for pigs is in the order of $10^{6.0}$ $TCID_{50}$.

During early acute phase of clinical disease, scraps of meat from infected animals are sufficient to infect pigs orally. As immunity develops (7-10days) the titres in edible tissue will be very low.

14.3.3 Sheep

There are few if any data on the infectious dose for sheep. However, Prof. Alex Donaldson (pers comm.) suggests that sheep would be closer to cattle than to pigs

Table 14.4 Summary of ID_{50}s for FMD.

Animal	Oral	Inhalation
Cattle	$10^{5.8}$ $TCID_{50}$	$10^{1.1}$ – $10^{2.6}$ $TCID_{50}$
Pigs	$10^{6.0}$ $TCID_{50}$	$10^{4.1}$ – $10^{5.6}$ $TCID_{50}$
Sheep	$10^{5.8}$ $TCID_{50}$	$10^{1.1}$ – $10^{2.6}$ $TCID_{50}$

14.4 Effect of Cooking

In blood inactivation occurs at 55 °C for 20 min and 60 °C for 2 min. In minced beef need at least 68 – 79 °C is required for inactivation. If heart or milk present need core temp or 93 °C. In summary need 80-100 °C for 2-3 min or 70 °C for 25 min (MacDiarmid 1991). It is assumed for the purpose of risk assessment that cooking inactivates FMDV.

14.5 Decay on soil

Haas *et al.* (1995) reported data for the survival of FMDV in cattle slurry at two temperatures. These are plotted in **Figure 14.4**.

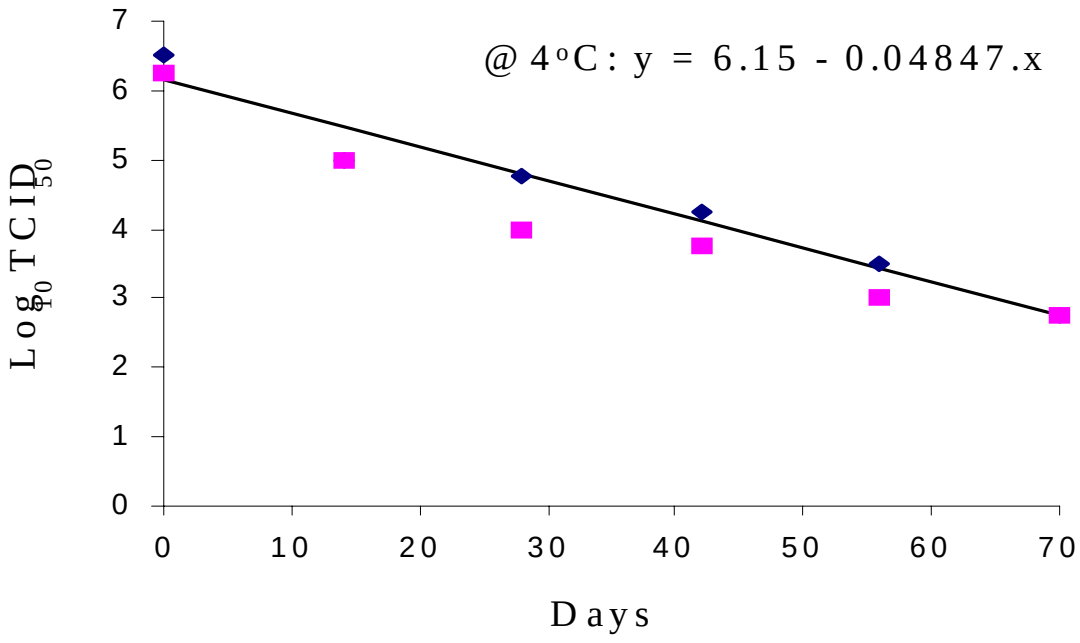

Figure 14.4 Decay of FMDV in cattle slurry at 4°C and 17°C. Data from Haas *et al.* (1995).

In absence of specific data for soil, it is assumed that decay on the soil is similar to that in **Figure 14.4**. The log decay appears is linear for at least 70 days. The decay constant is 0.04847.$\log_{10}$ day^{-1}. For the purpose of risk assessment it is assumed that this log decay continues as a linear process for 103 days giving a 5 log decay. Cattle ingesting 0.41 kg soil day^{-1} (EUSES, 1997) are therefore exposed to smaller and smaller risks over this period.

However, the model does not allow more than 5-log decay on the soil. Thus for the final 262 days of the year, the soil loading remains constant at $10^{-5}.N_0$. For the purpose of the risk assessment, the cumulative annual exposure is therefore expressed mathematically as:-

Equation 8

$$\sum_{t=1}^{t=103} 0.41.N_o 10^{-0.04847.t} + \sum_{104}^{365} 0.41.N_0 10^{-5}$$

The decay with time is plotted in **Figure 14.5**.

14.6 Quantitative risk assessment for FMDV

14.6.1 Source Term – a single FMD-infected porcine virus

An infected porcine carcass with "bone-in" and blood drained out contains 2.6 x 10^{11} $TCID_{50}$ units. One bovine oral ID_{50} comprises $10^{5.8}$ $TCID_{50}$. Therefore the "bone-in" carcass contains about 410,000 bovine oral ID_{50}.

A "bone-out" carcass contains about 63,000 bovine oral ID_{50}.

14.6.2 Pathways

The model assumes that the 1% of meat is discarded uncooked to the catering waste bin (**Section 2.2.2**). Composting removes 4.7 logs, the decay on land is 0.04847-log day^{-1} (**Equation 8**) for the first 103 days, after which there is no decay (**Figure 14.5**).

14.6.3 Exposures and risks - A single FMD infected porcine carcass "bone-in" enters the food chain – model assumes no time interval between application of compost and grazing.

Consider a single FMD-infected pig's carcass (mass 62 kg) with "bone-in" entering the food chain each year. The total loading is 408,000 bovine/sheep oral ID_{50} units. 1% enters the catering waste bin as uncooked meat. Meat Exclusion/Composting removes 4.7-logs (**Table 7.1**), leaving 0.08 bovine/sheep oral ID_{50} units in 500,000 tonnes composted waste. The concentration of infectivity in the compost residues is therefore 1.6 x 10^{-7} bovine/sheep oral ID_{50} units tonne^{-1}. Applying compost residues at a rate of 10 tds ha^{-1} gives a concentration of 1.1 x 10^{-9} bovine oral ID_{50} tonne^{-1} soil at time t = 0 (**Figure 14.5**). The cumulative annual exposure to a bovine ingesting 0.41 kg soil day^{-1} with an FMD decay rate of 0.04847-log day^{-1} for the first 103 days (**Equation 8**) is 4.2 x 10^{-12} oral ID_{50} cow^{-1} year^{-1}. This translates into a risk of 2.9 x 10^{-12} cow^{-1} year^{-1} (assuming a linear dose-response relationship (**Section 11.4**)) for cattle grazing on land

to which the composted catering waste residues have been applied. The risk for sheep eating 0.2 kg soil day^{-1} is estimated at 1.5 x 10^{-12} sheep^{-1} year^{-1}.

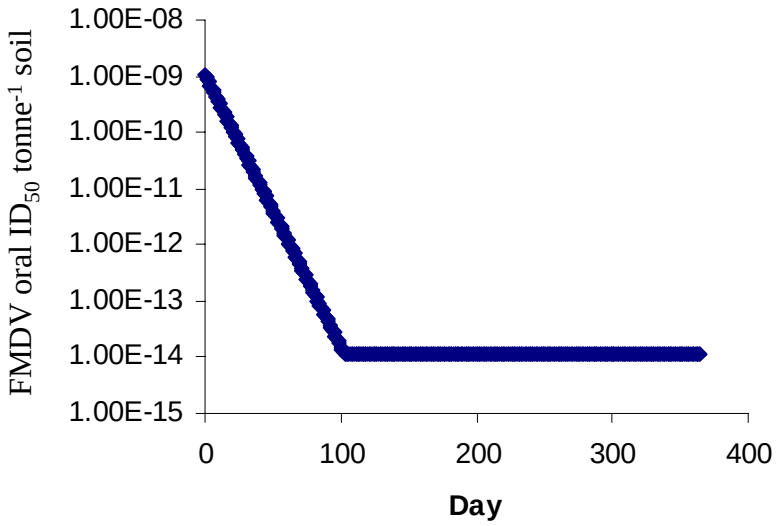

Figure 14.5 Decay of FMDV in soil with time according to Equation 8. Concentrations based on a single FMDV-infected pig carcass entering the food chain.

On the basis that 42,800 head of cattle and 157,000 sheep (i.e. 0.52% of the herd) could be exposed to composted catering waste, the expected number of FMD infections would be 1.2 x 10^{-7} cattle and 2.3 x 10^{-7} sheep in the UK per year.

It is concluded that a single FMD-infected pig's carcass entering the food chain presents a remote risk to grazing cattle and sheep through application of composted catering waste to land – even if no time interval is allowed for between application of compost and grazing. Indeed, one case of FMD would be expected every 2.8 million years.

14.7 Risks from imported material into the UK

FMD is not endemic in the UK. Britain has imported boneless beef from FMD-endemic countries (e.g. Argentina) for many years, without introducing FMD. The safe importation of beef is therefore possible. Eight out of 34 primary outbreaks in the EC between 1977 and 1987 were due to imports of meat that had not been deboned. There is no evidence that boneless beef has ever been the origin of an FMD outbreak.

14.7.1 Illegal imports – assumes 10,000 infected "bone-in" pig's carcasses imported per year.

Corso (1997) developed a risk assessment of the likelihood of exposing domestic swine in the USA to selected exotic disease agents by feeding uncooked swill. The hazard was assumed to originate from contraband food items entering the USA and subsequently being discarded in household waste. Four exotic diseases were studied,

including classical swine fever (CSF), foot and mouth disease (FMD), swine vesicular disease (SVD) and African swine fever (ASF). The study showed that, of these four viral agents, the probability of exposure was highest for CSF virus. The median annual likelihood of one or more contaminated loads of swill being fed to swine in the continental USA, according to Corso (1997) are summarised in **Table 14.5**.

Table 14.5 Likelihood of exposure of domestic swine to exotic disease agents through uncooked swill in the USA.

Virus	Probability of one or more infected loads being fed to swine in USA
CSF	0.063
FMD	0.043
SVD	0.005
ASF	0.005

Contraband may enter the UK through a variety of methods. Corso (1997) focused specifically on contraband introduced by travellers through established ports of entry and on items sent through the mail. Corso (1997) assumed that contraband in the USA would be exclusively in waste from households, and not other facilities such as restaurants. Corso reasoned that restaurants are likely to contain food items that were acquired legally from a common source, and are unlikely to include individual pieces of contraband food.

There are no data on the amount of pork illegally imported into the UK. The model therefore assumes that 10,000 FMD-infected "bone-in" porcine carcasses are imported into the UK each year. This represents 620 tonnes of infected carcass. On the basis that 1% of illegally imported pigs' meat is infected this would represent a total illegal importation of 62,000 tonnes of pig's meat into the UK per year. In the absence of any concrete data, I have therefore constructed this worst-case scenario on the basis that it is unrealistic.

Source Term

Assuming no time interval between application of compost and grazing.

The individual risks to cattle and sheep grazing on land to which the composted catering waste has been applied are presented in **Table 14.6**. Assuming 0.52% of the UK herd grazes on this land then according to the model there is one FMD infection in cattle and sheep every 283 years from 10,000 FMD-infected "bone-in" porcine carcasses.

Table 14.6 Summary of predicted FMD risks from composting of catering waste from the illegal importation of 10,000 FMD-infected "bone-in" porcine carcasses – assumes no time interval period.

Animal	Cumulative Annual Risk ($animal^{-1}$ $year^{-1}$)	Number of FMD infections ($year^{-1}$)
Cattle	2.9×10^{-8}	0.0012

Sheep	1.45×10^{-8}	0.0023
Total		0.0035

Allowing for a one year time interval between application of compost and grazing (Figure 14.5).

Assuming a 5-log reduction of FMDV in the soil after one year (**Figure 14.5**), then the arithmetic mean soil loading is 1.1×10^{-10} FMDV ID_{50} $tonne^{-1}$ soil. The annual risk to cattle ingesting 0.41 kg soil cow^{-1} day^{-1} is remote at 1.11×10^{-11} cow^{-1} $year^{-1}$. Similarly for a sheep ingesting 0.2 kg soil $sheep^{-1}$ day^{-1} the risk is remote at 5.7×10^{-12} $sheep^{-1}$ $year^{-1}$. Assuming that 0.52% of the cattle and sheep then graze on this land after one year, the numbers of FMD infections would be remote at 1.39×10^{-6} cases in England/Wales per year (**Table 14.7**).

Accidental grazing during the 1 year no grazing period

Of those 0.52% of the cattle and sheep grazing in the vicinity of the land to which compost has been applied, it is assumed that 1% accidentally graze on the land during the 1 year ban for a period of seven days. The average FMDV exposure from ingesting soil calculated over the one year period assuming decay as in **Figure 14.5** is 1.1×10^{-10} ID_{50} cow^{-1} day^{-1}. Over the 7 day period of accidental grazing, this is 8.0×10^{-10} ID_{50} cow^{-1}, which translates into a risk of 5.6×10^{-10} cow^{-1} (by multiplying by 0.69 (**Section 11.4**)). Assuming 1% of 0.52% of the England/Wales herd (i.e. 428 cows) accidentally graze for 7 days, then there would be 2.4×10^{-7} FMD cases $year^{-1}$ in cattle in England and Wales (**Table 14.7)**.

The total number of FMD-infected cattle and sheep allowing for a 1 year ban but with some accidental grazing is 2.1×10^{-6} $year^{-1}$ (**Table 14.7**). This compares to 0.0035 cases without the 1 year ban. Thus, the one year ban reduces the risks by 1,700-fold even with accidental breach of the ban.

Table 14.7 Summary of predicted numbers of FMD cases from composted catering waste assuming the illegal importation of 10,000 FMD-infected "bone-in" porcine carcasses. Numbers based on 0.52% of UK herd grazing on land to which compost has been applied – assumes 1 year ban with 1% of animals spending 7 days accidentally grazing on that land.

Animal	Number of FMD infections in England/Wales ($year^{-1}$)			
	No grazing ban	1 yr. grazing ban in place	Accidental Grazing during 1 year ban	Total for 1 yr. ban but with accidental grazing
Cattle	0.0012	4.9×10^{-7}	2.4×10^{-7}	7.3×10^{-7}
Sheep	0.0023	9.0×10^{-7}	4.4×10^{-7}	1.3×10^{-6}
Total	0.0035	1.39×10^{-6}	6.8×10^{-7}	2.1×10^{-6}

14.7.2 Legal imports – boneless meat.

A total of 105,000 tonnes of processed beef was imported into the UK in 2000, together with 123,000 tonnes of imported lamb. The total "bone-out" imported beef and sheep

meat is therefore 228,000 tonnes or 2.28 x 10^8 kg. According to **Table 14.3** a "bone-out" pig contains 4.0 x 10^{10} $TCID_{50}$ and weighs 56.6 kg. Assuming the tissue loadings of FMD in cattle and sheep tissues are the same as for pigs, then the average FMD loading in meat from infected animals is 4.0 x 10^{10} / 56.6 = 7.1 x 10^8 $TCID_{50}$ / kg. Assuming 1% (2.28 x 10^6 kg) of the imported "boneless" meat is from FMD-infected animals, then 2.28 x 10^6 kg x 7.1 x 10^8 $TCID_{50}$ / kg = 1.6 x 10^{15} $TCID_{50}$ are imported into the UK annually. Assuming 1% of this is discarded on uncooked meat into catering wastes, then the risk to grazing cattle and sheep from application of composted catering wastes are very low (**Table 14.8**). Indeed, on the basis of meat exclusion/composting removing 4.7-logs (**Table 7.1**) of FMD, then one case of FMD in sheep or beef is predicted every 451 years if there is not a one year ban. Enforcing a one year ban and allowing for accidental grazing, reduces this to one case every 780,000 years.

Table 14.8 Summary of predicted number of FMD cases from composted catering waste from the importation of 2.28 x 10^5 tonnes of boneless beef/sheep of which 1% is from FMD-infected carcasses.

Animal	Number of FMD infections ($year^{-1}$)	
	No grazing ban	1 yr. ban but with accidental grazing
Cattle	0.0007	4.6 x 10^{-7}
Sheep	0.0014	8.4 x 10^{-7}
Total	0.0021	1.3 x 10^{-6}

14.8 Risks during an FMD outbreak in the UK

The virus is excreted one to ten days before clinical signs appear (Gibbens *et al*. 2001). Therefore during an FMD epidemic in the UK, large quantities of FMD-infected tissues could enter the human food chain particularly in the early stages. This raises the question of whether the application of composted catering waste residues to agricultural land could promote the outbreak.

Table 14.9 Total animals slaughtered in infected premises in UK 2001 outbreak (to 24 Feb 2002).

Cattle	301,448
Sheep	960,313
Pigs	20,308

Gibbens *et al.* (2001) describes the epidemiology of the 2001 foot-and-mouth disease epidemic in Great Britain. Outbreaks such as that in the UK in 2001 may prolong; indeed to mid-July 2001, 1849 cases had been detected. This raises the question of how many FMD-infected carcasses could enter the food chain prior to intervention during the period of one year. To 24 February 2002, the total cattle, sheep and pigs slaughtered in infected premises are listed in **Table 14.9**. Pigs accounted for only a small proportion. Sheep accounted for the main proportion.

The model assumes that the FMD loadings in sheep are similar to those in pigs. Assuming that 960,313 sheep "bone-in" sheep carcasses went into the human food chain in 2001, the model predicts one extra FMD-infected sheep or cow every 3 years across England and Wales from the application of composted catering waste to land (**Table 14.10**). This is unacceptable. However, the enforcement of the one year "no grazing" interval is sufficient to reduce the risk to an acceptable level even allowing of accidental grazing during that period.

Table 14.10 Summary of predicted FMD risks from composting of catering waste from 960,313 FMD-infected "bone-in" sheep carcasses during an FMD outbreak.

Animal	Number of FMD infections ($year^{-1}$)	
	No grazing ban	1 yr. ban but with accidental grazing
Cattle	0.119	0.7×10^{-4}
Sheep	0.219	1.3×10^{-4}
Total	0.338	2.0×10^{-4}

Thus even during an outbreak of FMD, application of composted catering waste to land will not promote the outbreak providing the 1 year no-grazing interval is enforced.

14.9 Effect of the time period of the no grazing ban

From the decay curve for FMDV presented in **Figure 14.5**, the effect of the length of the no grazing ban was investigated. Annual exposures were calculated using an approach similar to **Equation 8** but starting at t = 31 for 1 month, t = 61 for 2 month and t = 91 for 3 month and summing for 365 days. The expected numbers of infected cattle and sheep (assuming 0.52% of the UK animals graze on such land) are presented in **Table 14.11**. A 1 month ban reduces the predicted number of cases by 31-fold. An additional one month ban (i.e. 2 months) reduced the predicted number of cases by a further 22-fold, giving a 693-fold reduction. After this the "law of diminishing returns" applies with a further 1 month extension (i.e. to 3 months in total) only reducing the risks by 3.5-fold compared to the 2 month ban. Going from 2 months to 1 year only reduces the number of cases by 3.7-fold (**Table 14.11**). Thus, even during an outbreak, a two month ban would be sufficient.

Table 14.11 Summary of predicted numbers of FMD cases in cattle and sheep from composted catering waste allowing for different no grazing time intervals (assuming soil decay according to Figure 14.5).

Assumes 10,000 FMD-infected "bone-in" pig carcasses imported

	No grazing ban	**1 month**	**2 month**	**3 month**	**1 year**
	Predicted number of FMD infections in England/Wales (animals $year^{-1}$)				
Cows	0.0012	3.9×10^{-5}	1.8×10^{-6}	5.1×10^{-7}	4.9×10^{-7}

Sheep	0.0023	7.2×10^{-5}	3.3×10^{-6}	9.3×10^{-7}	8.9×10^{-7}

14.10 Sensitivity analysis – the number of $TCID_{50}$s comprising a pfu - A

There is no fixed relationship between $TCID_{50}$ and pfu end points even in the same cell system. The pfu endpoint could be 1 to 4 logs less than those obtained in a $TCID_{50}$ assay, with a factor of 5 to 10-fold (i.e. 1-log) being a "rough" estimate (Prof. Alex Donaldson, pers. comm.). The model therefore assumes that 1 pfu is comprised of 10 $TCID_{50}$s.

Setting the number of $TCID_{50}$ units in one pfu to 10,000 (i.e. 4-log, the absolute upper value) predicts 2.5×10^{14} $TCID_{50}$ units in an infected pig (as opposed to just 2.6×10^{11}; see **Table 14.3**). The predicted number of FMD cases (on the basis of 10,000 infected pigs entering the food chain annually) increases by a factor of 1,000-fold relative to those in **Table 14.1**1. Thus with a 2 month no grazing period, there will be 0.0018 cows infected and 0.0032 sheep infected each year, i.e. 1 case every 200 years. This is quite acceptable. However, a grazing ban of 2 months becomes vitally important.

15. Classical Swine Fever Virus (Hog Fever)

Classical swine fever (also known as hog cholera or swine plague) is caused by a virus of the genus Pestivirus, of the family Flaviviridae. Under natural circumstances the pig is the only animal to become infected. The virion envelope contains lipids; thus the virus is inactivated by organic solvents. Classical swine fever does not affect humans. Pigs are the only animals clinically affected, although the virus can replicate transiently in sheep and cattle. Transmission is by ingestion, contact with the conjunctivae and mucous membranes, contact with skin abrasions, insemination and percutaneous blood transfer. Spread is by direct contact with infected pigs or products from infected pigs (e.g. from feeding insufficiently-cooked waste food).

15.1 Source Term

The pig is the only natural reservoir of CSFV. Blood, tissues, secretions and excretions from an infected animal contain CSFV. The virus initially infects the epithelial cells of the tonsillar crypts and subsequently spreads to the surrounding lymphoreticular tissue. Replication occurs in the regional lymph nodes associated with the tonsils from where it reaches the peripheral blood and high titres are attained in the spleen, bone marrow and visceral lymph nodes and the lymphoid structures lining the small intestine. The level of viraemia is high and persistence of this state depends on the virulence of the particular CSFV strain.

Pigs in the prodromal period of infection could pass through an abattoir without being detected as abnormal.

CSFV is stable in pH range 5-10 but inactivated at pH 3 or below and above pH 10. No destruction would occur at the pH levels due to rigor mortis in the muscle.

Farez and Morley (1997) reported mean vital titres (as pfu) in **Table 15.1**. To convert to $TCID_{50}$ units, a factor of 251 is applied. This was calculated from the fact that 4-5 days after infection muscle had 10 pfu g^{-1} (**Table 15.1**), while on day 7 in another experiment $10^{3.4}$ $TCID_{50}$ g^{-1} of CSF virus was reported in quadriceps (Farez and Morley 1997).

Table 15.1 Classical swine fever viral titres in tissues of 64 pigs four or five days after experimental infection. Data from Farez and Morley (1997).

Tissue	Mean Titre (per ml or g)	
	PFU	$TCID_{50}$ corrected by multiplying pfu by 251
Blood	$10^{3.8}$	1.6×10^{6}
Lymph node	$10^{3.9}$	2.0×10^{6}
Bone Marrow	$10^{5.2}$	4.0×10^{7}
Fat	$10^{0.8}$	1,584
Muscle	$10^{1.0}$	7.9×10^{5}

CSF virus titres in muscle ranged from $10^{3.4}$ to $10^{4.9}$ $TCID_{50}$ per g and titres in lymph nodes ranged from $10^{5.0}$ to $10^{7.5}$ $TCID_{50}$ per g with similar titres in other tissues. A value of $10^{4.9}$ $TCID_{50}$ g^{-1} is used for muscle and $10^{7.5}$ $TCID_{50}$ g^{-1} for lymph in **Table 15.2**. For blood a value of $10^{3.8}$ pfu / ml (**Table 15.1**) is used (= 1.6×10^{6} $TCID_{50}$ per ml).

Table 15.2 Classical Swine Fever Virus 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}
Kidneys	0.26		
Feet	2.00		0
Head, tongue	5.00		0
Gut contents	8.40		0
Intestinal fat	0.84	*1,584	1.3×10^{6}
Caul fat	0.11	*1,584	1.7×10^{5}
Intestines	2.70		0
Stomach (maw)	0.55		0
Heart	0.26	$10^{4.9}$	2.1×10^{7}
Lungs	0.90		0
Trachea	0.04		0
Heart, lungs, trachea	1.20		0
Liver, gall bladder	1.50		
Pancreas	0.06		0
Spleen	0.11		0
Blood drained from carcass	3.40	$*1.6 \times 10^{6}$	[d] 5.3×10^{9}
Cerebro-spinal fluid			0
Skirt	0.35		0
Hair scrapings & hooves	0.84		0
Bladder	0.04		0
Reproductive organs	0.15		0
Lymph nodes	0.04[c]	$10^{7.5}$	1.3×10^{9}
Waste	0.75		0
Bone marrow	5.464[a]	$*4.0 \times 10^{7}$	2.2×10^{11}
Skeletal muscle	43.712[b]	$10^{4.9}$	3.5×10^{9}
Blood in muscle	5.464[a]	$*1.6 \times 10^{6}$	8.6×10^{9}
Total (bone –in)	(62.0)		[d] 2.31×10^{11}
Total (bone-out)	(56.6)		[d] 1.37×10^{10}

[a] assumes 10% of carcass weight (54.64 kg)
[b] assumes 80% of carcass weight (54.64 kg)
[c] value for sheep
[d] model assumes only 5% of high titre blood remains in the carcass (e.g. in blood clots)
*assumes 1 pfu = 251 $TCID_{50}$.

According to **Table 15.2**, some 94.6% of the CSFV loading in infected pigs is in the bone marrow. Each infected “bone-in” pig carcass contributes 2.3×10^{11} CSFV $TCID_{50}$s.

15.2 Sensitivity analysis – uncertainty over viral titres in blood

Viral titres as high as $10^{5.0}$ to $10^{6.9}$ pfu/ml plasma have been reported (Farez and Morley 1997). This is higher than the $10^{3.8}$ pfu / ml used in **Table 15.2**. Setting the blood titre to $10^{6.9}$ pfu / ml plasma increases to total loading in "bone-in" pig from 2.3 x 10^{11} CSFV $TCID_{50}$s (**Table 15.2**) to 5.7 x 10^{11} CSFV $TCID_{50}$s, i.e. roughly doubles the risk.

15.3 Oral ID_{50} for pigs

CSFV virus is highly infections to pigs through the oral route. Thus, the minimal infectious dose resulting in infectious disease was <10 $TCID_{50}$. Assuming the porcine oral ID_{50} is 100 $TCID_{50}$ (i.e. ID_{50} = 10 x minimum infectious dose, which is acceptable when considering the dose response curve for FMD in pigs, **Figure 14.3**) then each infected "bone-in" pig carcass contributes 2.3 x 10^{9} CSFV porcine oral ID_{50}s.

15.4 Survival in food

CSFV can survive in pork and pork products. Thus Farez and Morley (1997) report a 2 month survival in the bone marrow of salt-cured pork. Different forms of curing have varying effects giving survival times between 17 and 188 days.

The virus is very resistant to temperatures below 0°C but is more sensitive to warm temperatures and is readily killed by pasteurisation or cooking. Data for inactivation gives values of:

- 65°C for 30 minutes
- 71°C for 1 minute (2 cm^3 cubes)
- 66°C for 60 minutes, 68 °C for 45 minutes and 69 °C for 30 minutes (blood contaminated with 10^5 $TCID_{50}$ per ml)
- 50°C for 3 days
- 37°C for 7 to 15 days.
- -70°C for many years

The virus was not inactivated after 30 minutes at 62 °C.

The inactivation of ASFV depends on the physical nature of the medium. The virus is more resistant to heat in animal tissues compared with cell culture medium.

For the purpose of risk assessment, it is assumed that there is no decay of CSFV in food.

15.4.1 CSF Viral titres reported in pig meat

According to the calculations in **Table 15.2** there are 1.2 x 10^{10} $TCID_{50}$ units in a porcine "bone-out" carcass. On the basis that the carcass weight is 62 kg, this there are "on

average" 2.2 x 10^5 $TCID_{50}$ units g^{-1} of porcine meat. This is a about a 10-fold higher than the titre of 1.9 x 10^4 $TCID_{50}$ g^{-1} estimated for pig meat samples prepared from CSF-infected pigs at slaughter (**Table 15.3**). This demonstrates the worst-case nature of the risk assessment developed here for CSF.

Table 15.3 CSFV titres in meat samples from four pigs infected with ASF. Data from McKercher *et al.* (1978).

Product	Days after slaughter	pfu g^{-1}	*Estimated $TCID_{50}$ g^{-1}
Whole meat from pig killed 5 day post infection	0	$10^{1.87}$	1.9 x 10^4
Salami sausage	22	$10^{1.3}$	5,000
Pepperoni sausage	22	$10^{1.5}$	7,900
Salami sausage	104	<10	<2,510

*assume 1 pfu = 251 $TCID_{50}$

15.5 Decay in soil

Decay data for CSFV in pig slurry are presented in **Figure 15.1**.

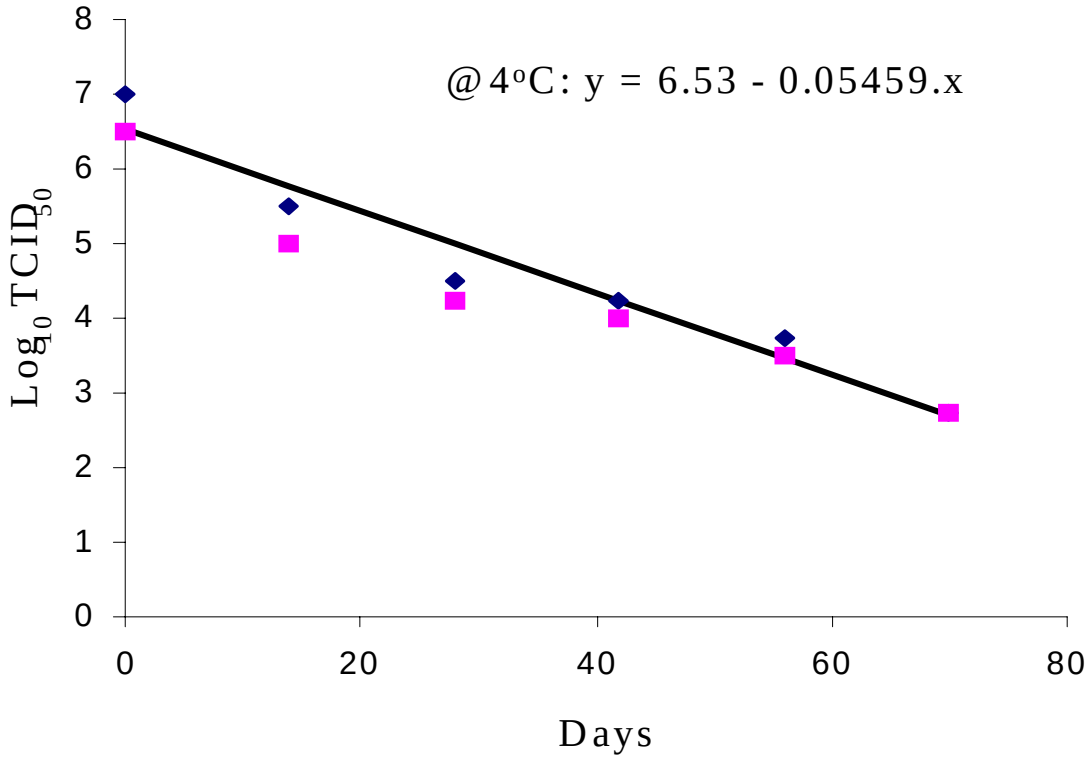

Figure 15.1 Decay of Classical Swine Fever in pig slurry at 4°C and 17°C. Data from Haas *et al.* (1995).

The risk assessment model does not allow more than 5-log decay on the soil. The decay rate of 0.05459 log_{10} day^{-1} gives a 5-log decay in 92 days. Thus for the final 273 days of the year, the soil loading remains constant at $10^{-5}.N_0$. The cumulative annual exposure to a pig eating 0.41 kg soil pig^{-1} day^{-1} to CSFV is therefore expressed mathematically as:-

Equation 9

$$\sum_{t=1}^{t=93} 0.41.N_o 10^{-0.05459.t} + \sum_{94}^{365} 0.41.N_0 10^{-5}$$

where N_0 is the concentration of CSFV (ID_{50} kg^{-1}) in the soil at t = 0.

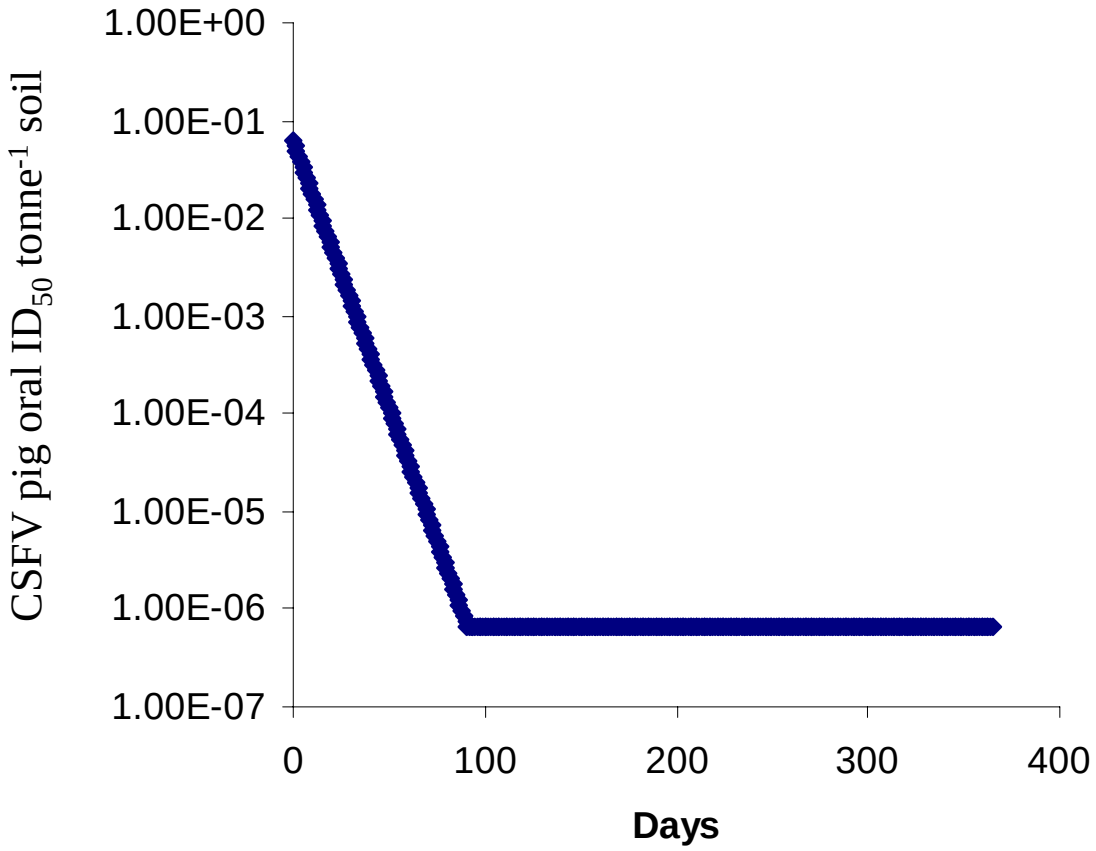

Figure 15.2 Decay of CSFV in soil with time according to Equation 9. Concentrations based on a 10,000 CSFV-infected pig carcasses entering the food chain per year.

In contaminated hay exposed to air but protected from sunlight and rain, viability was retained for 7 days but not present after 14 days. Survival in water varied from 6-24 days at 20 °C.

15.6 Risks of CSF from imported material into the UK

CSF is not endemic in the UK. The risks through catering waste are therefore directly related to the amount of CSF-infected pork imported into the UK.

15.6.1 Illegal imports – assumes up to 10,000 infected "bone-in" pig's carcasses imported per year.

There are no data on the amount of pork illegally imported into the UK. The model therefore assumes that (as for the FMD risk assessment) that 10,000 CSF-infected "bone-in" porcine carcasses are imported into the UK each year. This represents 620 tonnes of infected carcass. On the basis that 1% of illegally imported pigs' meat is infected this would represent a total illegal importation of 62,000 tonnes of pig's meat into the UK per year.

Source Term

10,000 CSF-infected porcine cascasses contribute 2.30×10^{15} $TCID_{50}$ (**Table 15.2**) per year. Assuming 1 oral ID_{50} = 100 $TCID_{50}$ (see **Section 15.3**), and that 1% of the porcine material goes to catering waste uncooked, then the total loading in catering waste is 2.3×10^{11} oral ID_{50} $year^{-1}$. Composting destroys 4.7-logs, leaving 4.6 million oral ID_{50} in the 500,000 tonnes of compost. The concentration in compost is therefore 9.2 oral ID_{50} $tonne^{-1}$. A 150-fold dilution in the soil gives N_0 = 0.06 oral ID_{50} $tonne^{-1}$ soil at t = 0 (**Figure 15.2**).

Assuming no time interval between application of compost and grazing.

Using **Equation 9**, the cumulative annual exposure for pigs grazing from t = 0 on land to which compost has been tilled in is 2.1×10^{-4} oral ID_{50} $year^{-1}$. Multiplying by 0.69 (see Gale & Stanfield 2001), this translates into an annual risk of 1.5×10^{-4} pig^{-1} $year^{-1}$ (**Table 15.4**). The individual risks to pigs grazing on land to which the composted catering waste has been applied are compared for different numbers of infected carcasses being imported in **Table 15.4**. Assuming 0.52% of the pigs are housed on this land then according to the model there are 5 CSF cases in pigs $year^{-1}$ from 10,000 illegally-imported CSF-infected "bone-in" porcine carcasses. This is clearly unacceptable. If only 100 CSF-infected carcasses were illegally imported each year, then the models predicts one case of CSF in pigs every 20 years.

Table 15.4 Summary of predicted number of CSF infections from composted catering waste from the illegal importation of 10,000 CSF-infected "bone-in" porcine carcasses – assumes no time interval period between application of compost and grazing.

Animal	Cumulative Annual Risk (pig^{-1} $year^{-1}$)	Number of CSF infections (pigs $year^{-1}$)
10,000	1.5×10^{-4}	4.9
1,000	1.5×10^{-5}	0.5
100	1.5×10^{-6}	0.05

Allowing for a one year time interval between application of compost and grazing (Figure 14.5).

Assuming a 5-log reduction of CSFV in the soil after one year (**Figure 15.2**), then the arithmetic mean soil loading is 6.6 x 10^{-7} CSFV ID_{50} $tonne^{-1}$ soil. The daily exposure to pigs ingesting 0.41 kg soil pig^{-1} day^{-1} is therefore 2.7 x 10^{-10} ID_{50} day^{-1}, which is equivalent to 9.9 x 10^{-8} ID_{50} pig^{-1} $year^{-1}$. This translates into a remote risk of 6.8 x 10^{-8} pig^{-1} $year^{-1}$ (**Table 15.5**). Assuming that 0.52% of the 6.37 million pigs (i.e. 33,400 pigs; **Table 4.3**) are housed on this land over one year, the numbers of CSF infections would be low at 0.0023 pigs $year^{-1}$ in England/Wales per year (**Table 15.6**).

Accidental grazing during the 1 year no grazing period

Of those 0.52% of the pigs in England/Wales house in the vicinity of the land to which compost has been applied, it is assumed that 1% accidentally gain entry to the land during the 1 yr. ban for a period of seven days. The average CSFV concentration in soil over the one year period is 1.4 x 10^{-3} oral ID_{50} $tonne^{-1}$ (**Figure 15.2**). Exposure from ingesting soil for seven days over the one year period assuming decay as in **Figure 15.2** is 4.1 x 10^{-6} oral ID_{50} pig^{-1}, which translates into a risk of 2.8 x 10^{-6} cow^{-1} (by multiplying by 0.69 (see Gale and Stanfield 2001)). Assuming 1% of 0.52% of the England/Wales herd (i.e. 334 pigs) accidentally graze for 7 days, then there would be 9.0 x 10^{-4} CSF cases $year^{-1}$ in pigs in England and Wales (**Table 14.7**).

The total number of CSF-infected pigs allowing for a 1 year ban but with some accidental grazing is 0.0032 $year^{-1}$ (**Table 15.6**). This compares to 4.9 cases without the 1 year ban. Thus, the one year ban reduces the risks by 1,500-fold.

It is concluded that the one year "grazing" ban is an important factor for CSF if it is believed that 10,000 CSF-infected "bone-in" pig carcasses enter the UK each year.

Table 15.5 Summary of predicted risks of CSF cases in pigs from composted catering waste assuming the illegal importation of 10,000 CSF-infected "bone-in" porcine carcasses.

Number of CSF-infected bone-in carcasses imported	Risk of infection (pig^{-1} $year^{-1}$)	
	No grazing ban	1 yr. grazing ban in place
10,000	1.5 x 10^{-4}	6.8 x 10^{-8}
1,000	1.5 x 10^{-5}	6.8 x 10^{-9}
100	1.5 x 10^{-6}	6.8 x 10^{-10}

The main assumptions of the model are:-

- 1% of pigs carcass enters the catering waste uncooked
- no decay of CSFV in the meat
- composting removes 4.7-logs
- pigs ingest 0.41 kg soil d^{-1}
- 0.52% of pigs in England/Wales are housed on land to which composted catering waste has been applied.

Table 15.6 Summary of predicted numbers of CSF cases in pigs from composted catering waste. Numbers based on 0.52% of UK pigs housed on land to which compost has been applied – assumes 1 year ban with 1% of animals spending 7 days accidentally gaining entry to that land.

Number of CSF-infected bone-in carcasses imported	Number of CSF infections in England/Wales (pigs year^{-1})			
	No grazing ban	1 yr. grazing ban in place	Accidental Grazing during 1 year ban	Total for 1 yr. ban but with accidental grazing
74,793	36.6	0.017	0.007	0.024
10,000	4.9	0.0023	9.0×10^{-4}	0.0032
1,000	0.5	0.00023	9.0×10^{-5}	0.00032
100	0.05	0.000023	9.0×10^{-6}	0.000032

15.7 Risks during a CSF outbreak in the UK

In the 2000 outbreak of CSF in the some 74,793 pigs were slaughtered. Although completely unrealistic, a worst-case assumption would be to assume that as many infected animals went into the food chain. Allowing for a 1 year no grazing policy, and also accidental grazing during the first year, this would result in 0.024 pigs being infected in England and Wales through composted catering waste (**Table 15.6**). This is in effect one case every 41 years, and could be interpreted to mean that there is one additional case through compost every 41 outbreaks. However, it should be noted that considerably greater decay could occur during the one year ban allowed for here (**Figure 15.2**) such that the risks are muck lower.

15.8 Effect of the time period of the no grazing ban

From the decay curve for CSFV presented in **Figure 15.2**, the effect of the length of the no grazing ban was investigated. Annual exposures were calculated using an approach similar to **Equation 9** but starting at t = 31 for 1 month, t = 61 for 2 month and t = 91 for 3 month and summing for 365 days. The expected numbers of infected pigs (assuming 0.52% of the UK pigs are housed on such land) are presented in **Table 15.7**. A 1 month ban reduces the predicted number of cases by 48-fold. An additional months ban (i.e. 2 months) reduced the predicted number of cases by a further 23-fold, giving a 1,150-fold reduction. After this the “law of diminishing returns” applies with a further 1 month extension (i.e. to 3 months in total) only halving the risks compared to the 2 month ban.

Table 15.7 Summary of predicted numbers of CSF cases in pigs from composted catering waste allowing for different no grazing time intervals (assuming soil decay according to Figure 15.2).

Number of CSF-infected "bone-in" carcasses imported	No grazing ban	1 month	2 month	3 month	1 year
	Predicted number of CSF infections in England/Wales (pigs year^{-1})				
74,793	36.6	0.76	0.032	0.017	0.017
10,000	4.9	0.1	0.004	0.002	0.002
1,000	0.5	0.01	0.0004	0.0002	0.0002

16. Swine Vesicular Disease

Swine vesicular disease (SVD) is indistinguishable in the field from FMD. Pigs and humans are the only species affected by SVD.

16.1 Source Term

All tissues are infected and can act as vehicles for transmission of the disease. Tissues contain high titres before clinical signs apparent. Therefore the time of slaughter is critical; titres are highest 2-3 days after inoculation, but drop off rapidly. Titres in pork products are low unless prepared from a herd in early stages of infection. Apparently healthy pigs can be slaughtered and infected pork and pork products passed into the food chain. Most virus is produced during the first week of infection and rather less during the second week. Indeed titres in the blood peak at about 10^5 pfu g^{-1} at about 3 days (Hedger and Mann 1989).

Burrows *et al.* (1974) provides a most comprehensive table of SVDV titres in pig tissues. In general data for a pig slaughtered 4 days after inoculation are used. Titres of infectivity in lymph nodes of pigs at 2 – 4 days after infection were $10^{5.8}$ pfu g^{-1}. (Mann and Hutchings 1980). Burrows *et al*. (1974) reported serum loadings of up to $10^{6.0}$ ml^{-1} at 2 days after inoculation.

The major loading appears to be from blood. Indeed, unlike FMD, ASF and CSF, there loading in bone marrow is relatively low, accounting for only 0.3% of the total loading in the carcass.

In bone-in carcasses there are an estimated 7.8×10^9 SVDV pfu (**Table 16.1**).

16.2 Survival in meat

SVDV is unaffected by the pH change of rigor mortis and persists indefinitely in refrigerated pork.

SVDV is relatively stable over a pH range of 2-12. Data for survival gives values of:

- 300 days in Parma hams
- 200 days in dry salami sausage, dry pepperoni sausage and intestinal casings
- 400 days in dried pepperoni and salami sausage
- 780 days in processed intestinal casings
- 40 days in salami and pepperoni sausages
- 509 days in unprocessed intestinal casings
- 28 days in Iberian loins

- 112 days in Iberian shoulder hams
- 560 days in Iberian hams
- 539 days in white Serrano hams.

It is assumed that there is no decay in meat.

Table 16.1 Swine Vesicular Disease Virus loadings in an infected pig (Data from Burrows *at al.* (1974) and Mann and Hutchings, 1980).

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}
Feet	2.00		0
Head, tongue	5.00	$10^{5.0}$	5.0×10^{8}
Gut contents	8.40		0
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}
Stomach (maw)	0.55		0
Heart	0.26	$*10^{4.5}$	8.2×10^{6}
Lungs	0.90		0
Trachea	0.04		0
Heart, lungs, trachea	1.20		0
Liver, gall bladder	1.50	$10^{5.0}$	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 drained from carcass	3.40	$10^{6.0}$	[d] 3.4×10^{9}
Cerebro-spinal fluid			0
Skirt	0.35		0
Hair scrapings & hooves	0.84	$10^{4.2}$	1.3×10^{7}
Bladder	0.04		0
Reproductive organs	0.15		0
Lymph nodes	0.04[c]	$10^{6.2}$	6.3×10^{7}
Waste	0.75		0
Bone marrow	5.464[a]	$10^{3.6}$	2.2×10^{7}
Skeletal muscle	43.712[b]	$*10^{4.5}$	1.4×10^{9}
Blood in muscle	5.464[a]	$10^{6.0}$	5.5×10^{9}
Total (bone –in)	(62.0)		[d] 7.8×10^{9}
Total (bone-out)	(56.6)		[d] 7.7×10^{9}

[a] assumes 10% of carcass weight (54.64 kg)
[b] assumes 80% of carcass weight (54.64 kg)
[c] value for sheep
[d] model assumes only 5% of high titre blood remains in the carcass (e.g. in blood clots)
*Data for Farez and Morley (1997)

16.3 Dose-response of SVDV in pigs

Damaged skin is the most susceptible tissue. Indeed Hedger and Mann (1989) write that, "When exposed to small amounts of virus, e.g. in unprocessed waste food, pigs probably become infected through damaged skin". Indeed, relatively large amounts of virus are required to produce clinical disease through oral and nasal routes.

16.3.1 Oral route

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 16.1**).

Thus each infected pig only contains 1,235 oral ID_{50} units. This is considerably lower than the 2.3×10^{9} CSFV oral ID50s per pig's carcass (**Section 15.3**).

16.3.2 Skin route

For skin, however, Mann and Hutchings (1980) found that although $10^{3.0}$ pfu did not produce disease, a dose of $10^{3.6}$ pfu infected 75% (3/4) of pigs. This suggests that the ID_{50} for SVDV is somewhere between $10^{3.0}$ and $10^{3.6}$ pfu for the skin route. According to the negative exponential dose-response curve fitted to the data in **Figure 16.1** the probability (r) of infection from a single pfu on the skin is 0.00035.

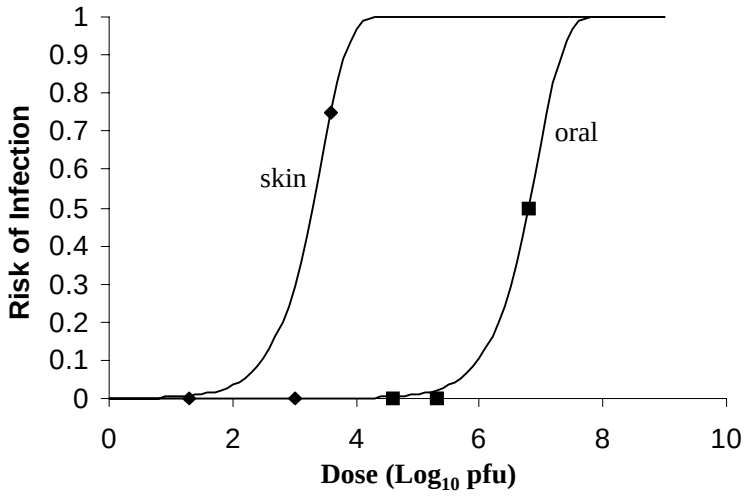

Figure 16.1 Negative exponential dose response curves fitted to data for oral (r = 00000011) and skin (r = 0.00035) challenge of SVDV to pigs. Data from Mann and Hutchings (1980).

16.4 Assume no decay in the soil

The epizootiology of SVD is related to the extraordinary stability of the virus outside the host. It is resistant to environmental factors over a wide pH range and is stable at normal temperatures. Virus has been isolated from crevices in farm buildings 11 weeks after slaughter of the herd even after vigorous cleansing and disinfection. Thus, recrudescence of disease may occur when susceptible pigs are introduced into contaminated buildings. In the UK, many outbreaks have been attributed to the contamination by infected pigs of hauliers' vehicles.

The risk assessment therefore assumes no decay of the SVDV in soil.

16.5 Risks of SVD from imported pig meat into the UK

SVD is not endemic in the UK. The last outbreak of SVD in the UK was in 1982. The risks through catering waste are therefore directly related to the amount of CSF-infected pork imported into the UK. Corso (1997) predicted annual risks of SVDV-contaminated swill being fed to pigs in the USA about an order of magnitude lower than for FMDV and CSF (**Table 14.5**). For FMD and CSF it was assumed, for the purpose of risk assessment, that 10,000 infected "bone-in" porcine carcasses were imported annually into the UK. On the basis of the probability estimates for Corso (1997), a ten-fold lower import challenge is assumed for SVD.

16.5.1 Illegal imports – assumes up to 10,000 infected "bone-in" pig's carcasses imported per year.

There are no data on the amount of pork illegally imported into the UK. The model therefore assumes that 1,000 SVDV-infected "bone-in" porcine carcasses are imported into the UK each year. This represents 62 tonnes of infected carcass. On the basis that 0.1% of illegally imported pigs' meat is infected this would represent a total illegal importation of 62,000 tonnes of pig's meat into the UK per year. The results of the risk assessment are summarised in **Table 14.6**.

Table 16.2 Summary of predicted SVD risks to pigs from composting of catering waste from the illegal importation of SVD-infected "bone-in" porcine carcasses. Risk calculated through oral challenge.

Number of CSF-infected carcasses imported illegally $year^{-1}$	Cumulative Annual Risk (pig^{-1} $year^{-1}$)	Number of CSF infections (pigs $year^{-1}$)
1,000	3.4×10^{-10}	1.13×10^{-5}
100	3.4×10^{-11}	1.13×10^{-6}
10	3.4×10^{-12}	1.13×10^{-7}

The main assumptions of the model are:-

- 1% of pigs carcass enters the catering waste uncooked
- no decay of SVDV in the meat
- composting removes 4.7-logs
- pigs ingest 0.41 kg soil d^{-1}
- no decay of SVDV in soil
- pigs randomly graze on land across the UK to which composted catering waste has been applied.

On the basis of 1,000 SVD-infected porcine carcasses being imported illegally into the UK, the model predicts 1 cases of SVD in pigs in the UK every 90,000 years from exposure to composted catering waste applied to land. This assessment is based on challenge through the oral route.

It should be noted that SVDV is about 1,000 more infectious to pigs through broken skin (**Figure 16.1**). The model assumes each pig ingests 0.41 kg soil day^{-1}. It is difficult to estimate the exposure to pigs wallowing in mud through broken skin. However, even if pigs manage to adsorb all the SVDV from 0.41 kg soil day^{-1}, the risk in **Table 16.2**, although 1,000-fold higher would still be remote.

17. African Swine Fever

ASF only affects pigs. Mortalities in outbreaks of ASF may be extremely high; up to 100%.

Table 17.1 African Swine Fever Virus loadings in an infected pig. Tissue HAD_{50} 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}
Kidneys	0.26		
Feet	2.00		0
Head, tongue	5.00		0
Gut contents	8.40		0
Intestinal fat	0.84	$10^{5.4}$	2.1×10^{8}
Caul fat	0.11	$10^{5.4}$	2.7×10^{7}
Intestines	2.70		0
Stomach (maw)	0.55		0
Heart	0.26	$10^{6.6}$	1.0×10^{9}
Lungs	0.90		0
Trachea	0.04		0
Heart, lungs, trachea	1.20		0
Liver, gall bladder	1.50		
Pancreas	0.06		0
Spleen	0.11		0
Blood drained from carcass	3.40	$10^{7.9}$	[d] 2.7×10^{11}
Cerebro-spinal fluid			0
Skirt	0.35		0
Hair scrapings & hooves	0.84		0
Bladder	0.04		0
Reproductive organs	0.15		0
Lymph nodes	0.04[c]	$10^{8.5}$	1.3×10^{10}
Waste	0.75		0
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] model assumes only 5% of high titre blood remains in the carcass (e.g. in blood clots).

The spread of ASF has been invariably linked to the feeding to pigs of waste food containing scraps of uncooked pigmeat originating in countries where ASF is endemic. ASFV spreads among pigs by direct contact or by bites of infected ticks. In addition, airborne transmission of virus was demonstrated by the production of ASF in pigs kept on a platform 2.3 m above infected pigs (see Wilkinson and Donaldson 1997).

ASF is enzootic in parts of Africa, Spain, Portugal and Sardinia. The disease has never been reported in Asia, North America or in Australasia.

17.1 Source Term

African Swine Fever virus titres in tissues of 65 pigs five days after experimental infection are presented in **Table 17.1**. The total loadings for each of the porcine tissues are calculated and the total infectivity in a carcass is estimated at 1.8×10^{13} HAD_{50} units. Assuming bone marrow accounts for 10% of the carcass weight, then the infectivity in bone marrow accounts for 96% of the total carcass infectivity. A "deboned" carcass thus contains 6.4×10^{11} HAD_{50} units (according to the model).

17.1.1 ASF Viral titres in meat

According to the calculations in **Table 17.1** there are 6.4×10^{11} HAd_{50} units in a porcine "bone-out" carcass. On the basis that the carcass weight is 62 kg, this there are "on average" 1.2×10^{7} HAd_{50} units g^{-1} of porcine meat. This is about a 1,000-fold higher than the titres reported in "food meat" samples prepared from ASF-infected pigs (**Table 17.2**). This demonstrates the worst-case nature of the risk assessment developed here for ASF.

Table 17.2 ASFV titres in meat samples from four pigs infected with ASF. Data from McKercher *et al*. (1978).

Product	Days after slaughter	Titre (heamadsorbing units 50% (HAd_{50}) per g (lower value	upper values)
Whole meat	2	$10^{3.25}$	$10^{3.75}$
Ground meat	2	$10^{3.25}$	$10^{3.75}$
Salami	3	$10^{2.0}$	$10^{2.5}$
Salami sausage	9	10^{-1}	
Pepperoni	3	$10^{3.0}$	$10^{3.25}$
Brined ham	2	$10^{2.5}$	$10^{3.75}$

17.2 Oral ID_{50} for pigs

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. This dose response curve for infectivity for pigs through the oral route based on data from McVivar (1984) is plotted in **Figure 17.1**.

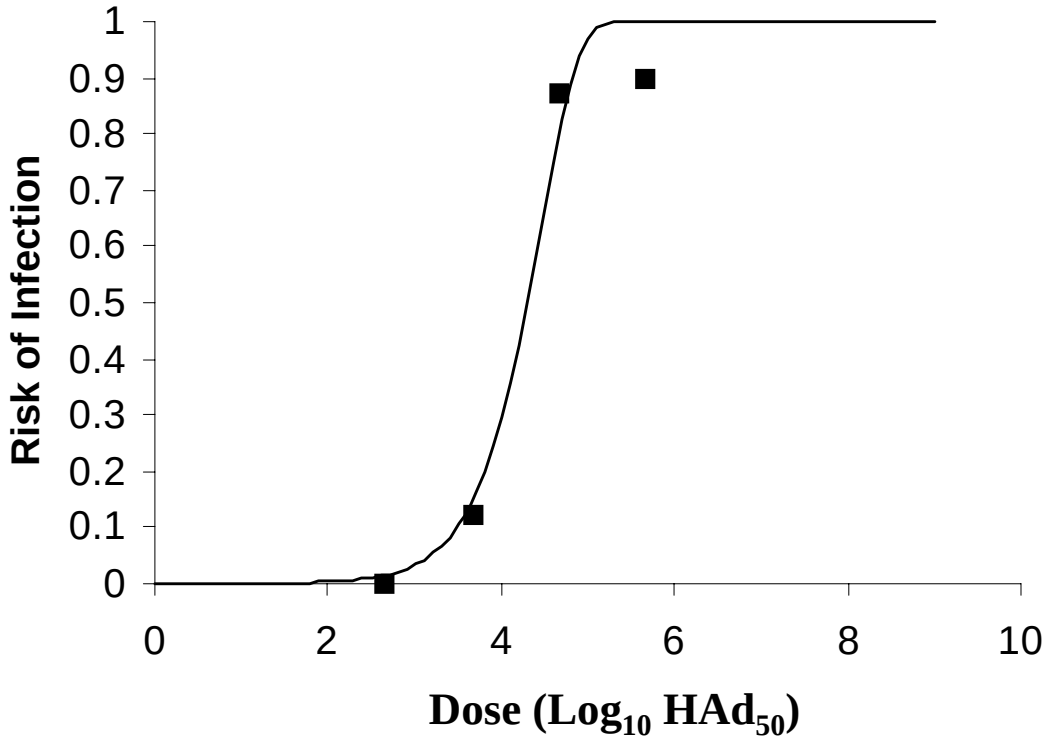

Figure 17.1 Negative exponential dose-response curve (r = 0.000035) for ingestion of ASFV by pigs. The porcine oral ID_{50} is about $10^{4.3}$ HAd_{50}. Data from McVicar (1984).

17.3 Survival in foods

Unlike FMDV, ASFV is resistant to the pH changes that accompany rigor mortis. ASFV is not inactivated by freezing and thawing and survives for many months in raw unprocessed frozen meat. ASFV has been recovered after 150 days from infected meat kept at 4ºC and after 188 days from bone marrow stored at –4ºC.

Furthermore, no significant decline in titre was observed in blood over a period of 75 weeks at 4ºC (Plowright and Parker 1967).

Survival

The virus is quite stable and will survive over a wide range of pH. In the absence of organic matter it is inactivated at pH values at or below 3.9 and at or above 11.5. In the presence of organic matter survival times are:

- 7 days at pH 13.4 in 25% serum
- 15 weeks in putrefied blood
- 11 days in faeces held at room temperature

- 18 months in pigs blood at 4°C
- 150 days in boned meat at 39 °F (sic)
- 140 days in salted dried hams.

The model allows for no decay in the meat.

17.4 Decay in soil

The decay of African Swine Fever virus in pig slurry at two temperatures is shown in **Figure 17.2**. Data are from Haas *et al.* (1995). The daily decay rate (0.029-$\log_{10}$ units d^{-1}) is half that of FMDV (**Figure 14.4**) and CSFV (**Figure 15.1**).

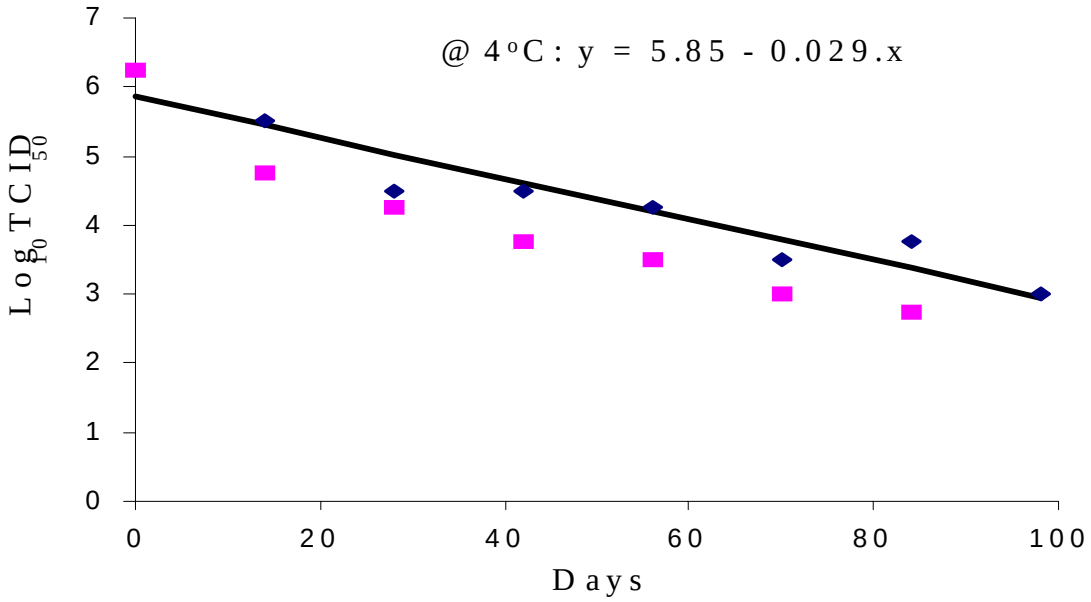

Figure 17.2 Decay of African Swine Fever Virus in pig slurry at 4°C and 17°C. Data from Haas *et al.* (1995).

The model does not allow more than 5-log decay on the soil (**Figure 17.3**). The decay rate of 0.029 $\log_{10}$ day^{-1} gives a 5-log decay in 169 days. Thus for the final 196 days of the year, the soil loading remains constant at $10^{-5}.N_0$. The cumulative annual exposure is therefore expressed mathematically as:-

Equation 10

$$\sum_{t=1}^{t=169} 0.41.N_o 10^{-0.029.t} + \sum_{170}^{365} 0.41.N_0 10^{-5}$$

where N_0 is the concentration of ASFV (ID_{50} kg^{-1}) in the soil at t = 0.

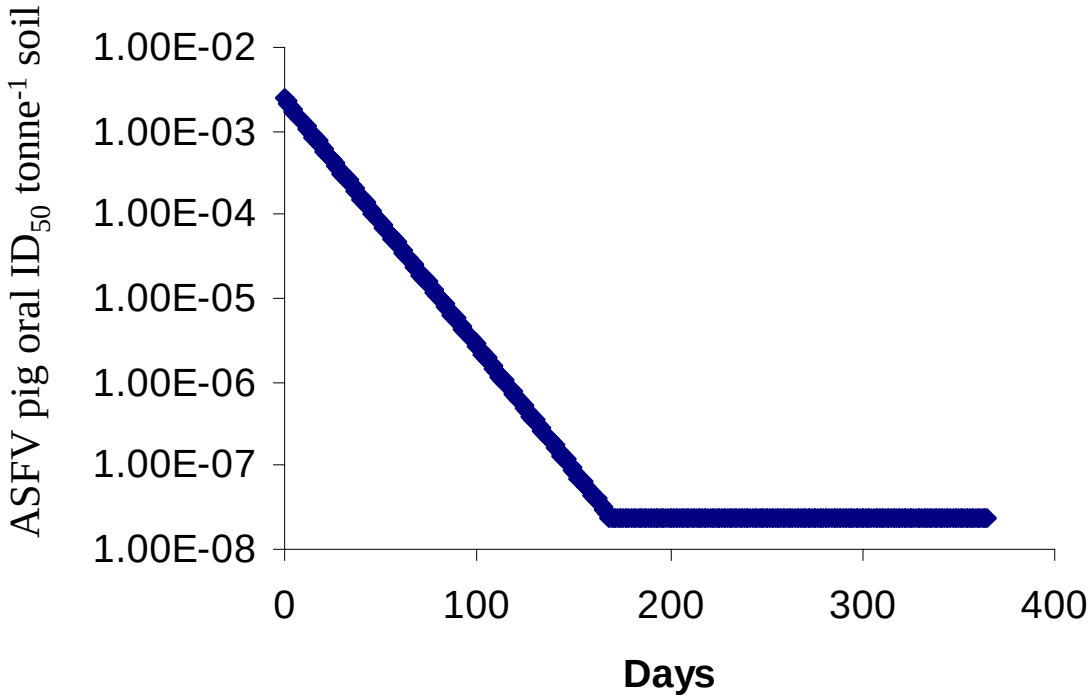

Figure 17.3 Decay of ASFV in soil with time according to Equation 10. Concentrations based on a 1,000 ASFV-infected pig carcasses entering the food chain per year.

17.5 Risks of ASF from imported material into the UK

There has never been an outbreak of African Swine Fever virus in the UK. Corso (1997) predicted annual risks of ASF-contaminated swill being fed to pigs in the USA about an order of magnitude lower than for FMDV and CSF (**Table 14.5**). For FMD and CSF it was assumed, for the purpose of risk assessment, that 10,000 infected "bone-in" porcine carcasses were imported annually into the UK. On the basis of the probability estimates for Corso (1997), a ten-fold lower import challenge is assumed for ASF, i.e. an upper limit of 1,000 ASF-infected pig carcasses.

17.5.1 Illegal imports – assumes up to 1,000 infected "bone-in" pig's carcasses imported per year.

The model assumes that 1,000 ASF-infected "bone-in" porcine carcasses are imported into the UK each year. This represents 62 tonnes of infected carcass. On the basis that 0.1% of illegally imported pigs' meat is infected with ASF this would represent a total illegal importation of 62,000 tonnes of pig's meat into the UK per year.

Source Term

1,000 CSF-infected porcine cascasses contribute 1.8 x 10^{16} HAD_{50} (**Table 17.1**) per year. Assuming 1 oral ID_{50} = $10^{4.3}$ HAD_{50} (see **Section 17.2**), and that 1% of the porcine material goes to catering waste uncooked, then the total loading in catering waste is 9.0 x 10^{9} oral ID_{50} $year^{-1}$. Composting destroys 4.7-logs, leaving 180,000 oral ID_{50} in the 500,000 tonnes of compost. The concentration in compost is therefore 0.36 oral ID_{50} $tonne^{-1}$. A 150-fold dilution in the soil gives N_0 = 0.0024 oral ID_{50} $tonne^{-1}$ soil at t = 0 (**Figure 17.3**).

Assuming no time interval between application of compost and grazing.

Using **Equation 10**, the cumulative annual exposure for pigs grazing from t = 0 on land to which compost has been tilled in is 1.5 x 10^{-5} oral ID_{50} $year^{-1}$. Multiplying by 0.69 (see Gale & Stanfield 2001), this translates into an annual risk of 1.0 x 10^{-5} pig^{-1} $year^{-1}$ (**Table 17.3**). The individual risks to pigs grazing on land to which the composted catering waste has been applied are compared for different numbers of infected carcasses being imported in **Table 17.3**. Assuming 0.52% of the pigs are housed on this land then according to the model there are 0.34 ASF cases in pigs $year^{-1}$ from 1,000 illegally-imported ASF-infected "bone-in" porcine carcasses. This is clearly unacceptable. If only 10 ASF-infected carcasses were illegally imported each year, then the models predicts one case of ASF in pigs every 290 years.

Table 17.3 Summary of predicted number of ASF infections from composted catering waste from the illegal importation of 1,000 ASF-infected "bone-in" porcine carcasses – assumes no time interval period between application of compost and grazing.

Animal	Cumulative Annual Risk (pig^{-1} $year^{-1}$)	Number of CSF infections (pigs $year^{-1}$)
1,000	1.0 x 10^{-5}	0.34
100	1.0 x 10^{-6}	0.034
10	1.0 x 10^{-7}	0.0034

Allowing for a one year time interval between application of compost and grazing (Figure 14.5).

Assuming a 5-log reduction of ASFV in the soil after one year (**Figure 17.3**), then the arithmetic mean soil loading is 2.4 x 10^{-8} ASFV ID_{50} $tonne^{-1}$ soil. The daily exposure to pigs ingesting 0.41 kg soil pig^{-1} day^{-1} is therefore 9.9 x 10^{-12} ID_{50} day^{-1}, which is equivalent to 3.6 x 10^{-9} ID_{50} pig^{-1} $year^{-1}$. This translates into a remote risk of 2.5 x 10^{-9} pig^{-1} $year^{-1}$ (**Table 17.4**). Assuming that 0.52% of the 6.37 million pigs (i.e. 33,400 pigs; **Table 4.3**) are housed on this land over one year, the numbers of ASF infections would be low at 0.00008 pigs $year^{-1}$ in England/Wales per year (**Table 17.5**).

Table 17.4 Summary of predicted risks of ASF infection in pigs from composted catering waste assuming the illegal importation of 1,000 ASF-infected "bone-in" porcine carcasses.

Number of ASF-infected bone-in carcasses imported	Risk of infection (pig^{-1} year^{-1})	
	No grazing ban	1 yr. Grazing ban in place
1,000	1.0 x 10^{-5}	2.5 x 10^{-9}
100	1.0 x 10^{-6}	2.5 x 10^{-10}
10	1.0 x 10^{-7}	2.5 x 10^{-11}

Accidental grazing during the 1 year no grazing period

Of those 0.52% of the pigs in England/Wales house in the vicinity of the land to which compost has been applied, it is assumed that 1% accidentally gain entry to the land during the 1 yr. ban for a period of seven days. The average ASFV concentration in soil over the one year period is 9.9 x 10^{-5} oral ID_{50} tonne^{-1} (**Figure 17.3**). Exposure from ingesting soil for seven days over the one year period assuming decay as in **Figure 17.3** is 2.8 x 10^{-7} oral ID_{50} pig^{-1}, which translates into a risk of 1.9 x 10^{-7} cow^{-1} (by multiplying by 0.69 (see Gale and Stanfield 2001)). Assuming 1% of 0.52% of the England/Wales herd (i.e. 334 pigs) accidentally graze for 7 days, then there would be 6.6 x 10^{-5} ASF cases year^{-1} in pigs in England and Wales (**Table 17.5**).

The total number of ASF-infected pigs allowing for a 1 year ban but with some accidental grazing is 0.00015 year^{-1} (**Table 17.5**). This compares to 0.34 cases without the 1 year ban. Thus, the one year ban reduces the risks by 2,200-fold.

It is concluded that the "grazing" ban is an important factor for ASF if it is believed that 1,000 ASF-infected "bone-in" pig carcasses enter the UK each year.

Table 17.5 Summary of predicted numbers of ASF cases in pigs from composted catering waste assuming the illegal importation of 10,000 ASF-infected "bone-in" porcine carcasses. Numbers based on 0.52% of UK pigs housed on land to which compost has been applied – assumes 1 year ban with 1% of animals spending 7 days accidentally gaining entry to that land.

Number of ASF-infected bone-in carcasses imported	Number of ASF infections in England/Wales (pigs year^{-1})			
	No grazing ban	1 yr. grazing ban in place	Accidental Grazing during 1 year ban	Total for 1 yr. ban but with accidental grazing
1,000	0.34	8.4 x 10^{-5}	6.6 x 10^{-5}	1.5 x 10^{-4}
100	0.034	8.4 x 10^{-6}	6.6 x 10^{-6}	1.5 x 10^{-5}
10	0.0034	8.4 x 10^{-7}	6.6 x 10^{-7}	1.5 x 10^{-6}

The main assumptions of the model are:-

- 1% of pigs carcass enters the catering waste uncooked
- no decay of ASFV in the meat

- composting removes 4.7-logs
- pigs ingest 0.41 kg soil d^{-1}
- pigs randomly graze on land across the UK to which composted catering waste has been applied.

17.6 Effect of the time period of the grazing ban

From the decay curve for ASFV presented in **Figure 17.3**, the effect of the length of the no grazing ban was investigated.

Table 17.6 Summary of predicted numbers of ASF cases in pigs from composted catering waste allowing for different no grazing time intervals (assuming soil decay according to Figure 17.3).

Number of ASF-infected "bone-in" carcasses imported	No grazing ban	1 month	2 month	3 month	1 year
	Predicted number of ASF infections in England/Wales (pigs $year^{-1}$)				
1,000	0.34	0.042	0.005	7.6×10^{-4}	8.4×10^{-5}
100	0.034	0.0042	0.0005	7.6×10^{-5}	8.4×10^{-6}
10	0.0034	0.00042	0.00005	7.6×10^{-6}	8.4×10^{-7}

Annual exposures were calculated using an approach similar to **Equation 10** but starting at t = 31 for 1 month, t = 61 for 2 month and t = 91 for 3 month and summing for 365 days. The expected numbers of infected pigs (assuming 0.52% of the UK pigs are housed on such land) are presented in **Table 17.6**. A 1 month ban reduces the predicted number of cases by 8-fold. An additional months ban (i.e. 2 months) reduced the predicted number of cases by a further 8-fold, giving a 63-fold reduction. An additional months ban (i.e. 3 months) reduces the predicted number of cases by a further 7-fold, giving a 450-fold reduction in total. After this the "law of diminishing returns" applies with an extension to one year in total only reducing the risks by a further 9-fold compared to the 3 months ban.

17.7 Other routes of infection

ASFV is very infectious to pigs through cutaneous scarification; the ID_{50} being around 50 HAD_{50} (McVicar 1984).

18. Newcastle Disease

Poultry products contaminated with pathogenic strains of Newcastle disease virus are a source of virus transmission to susceptible poultry flocks. Guittet *et al.* (1997) conclude that the probability of contamination varies according to the type of product. Pathogenic viruses can be isolated from the carcasses of chickens, whether vaccinated or not, during a brief period after experimental infection. Eggs laid by hens infected with Newcastle disease virus present a very low risk. Feathers, bones, blood and offal present potential risks if they are incorporated in poultry feed. Also, poultry droppings used as a fertiliser can present a major risk of infection in certain circumstances.

Two-stage composting has been shown to be effective in destroying the viruses of Newcastle disease and infectious bursal disease (cited in Senne *et al.* 1994).

18.1 A quantitative risk assessment

Data for ND are obtained from MAF (New Zealand) (1999), which outlines a risk assessment for imported chicken meat products from the UK. The document quotes data from Dennis Alexander (Veterinary Laboratories Agency), who determined that 4 $\log_{10}$ EID_{50} of the ND virus Herts 33/56 strain, which is highly pathogenic are required to establish infection in 3-week old chickens when given by the oral route. It is assumed therefore that the oral ID_{50} for chicks is 10^4 EID_{50}.

Dennis Alexander also determined the viral titres in a range of tissues and organs from 6-week old experimentally infected chickens. These are summarised in **Table 18.1**. It is assumed that the heart/kidney/spleen (giblets) weigh 30 g and are present in 5% of chickens at retail (**Section 3.4**). Assuming each chicken is "coated" in 1 g of faeces, the total loading on an infected chicken is 3.03×10^7 EID_{50}.

Table 18.1 Newcastle Disease EID_{50} loadings in an infected chicken

Tissue	EID_{50}	weight of organ (g)	Net loading (EID_{50} per infected chicken)
Heart/kidney/spleen	10^6	30 (in 5% of retail chickens)	1.5×10^6
Breast muscle	10^4	-	
Leg muscle	$10^{4.2}$	-	
Muscle average	12,924	2,229	2.9×10^7
Faeces	10^4	1	10^4
Total		2,260	3.03×10^7

In the UK 6.15×10^8 chickens are slaughtered annually. For the purpose of risk assessment, a "What if?" scenario is developed to address the risks through application of composted catering waste, if, for example, 100,000 of those birds were infected with Newcastle Disease. From **Table 18.1**, it may be calculated that the source term is 3.03×10^{12} EID_{50}. Assuming 1% of chicken goes into catering waste uncooked, then the total

loading on the compost process would be 3.03 x 10^{10} EID_{50}. This is equivalent to 3.03 x 10^{6} oral ID_{50}.

Assuming Source Separation/Composting removes 4.7-logs (**Table 7.1**), then just 60 oral ID_{50} would remain in the 500,000 tonnes of compost produced annually in the UK. The concentration of ND is therefore 1.2 x 10^{-4} chicken oral ID_{50} $tonne^{-1}$ of compost. On application of the compost into soil, dilution reduces this further to 8.0 x 10^{-7} oral ID_{50} $tonne^{-1}$ of soil. This is a factor of 12-fold lower than the number of oral ID_{50} units (1.08 x 10^{-5} $tonne^{-1}$) predicted for FMDV in soil (based 10,000 FMD-infected pigs entering the food chain annually). Since chickens will ingest considerably lower volumes of soil compared to cattle, pigs and sheep, it is concluded that the risks of ND to chickens from compost are lower in magnitude than the risks to sheep, cattle and pigs from FMDV.

This risk assessment is therefore no taken any further.

19. Protozoan Parasites Such Toxoplasma

Pathogenic protozoa are commonly transmitted to food in developing countries, but food-borne outbreaks of infection are relatively rare in developed countries. *Cyclospora cayetanensis* has emerged as a food-borne pathogen in foods imported into North America from South America. Nichols (2000) concludes that the measures needed to prevent food-borne protozoa causing disease require clear assessments of the risks of contamination and the effectiveness of processes to inactivate them. The globalisation of food production can allow new routes of transmission. Furthermore advances in diagnostic detection methods and surveillance systems have extended the range of protozoa that may be linked to food.

19.1 Cryptosporidium

Food can become contaminated with *Cryptosporidium* oocysts through irrigation or spraying with non-potable water. Outbreaks have been associated with inadequately pasteurised milk, apple juice, uncooked green onions, and chicken salad. Incidents have also been linked to raw milk, inadequately pasteurised milk, sausage and frozen tripe. *Cryptosporidium* oocysts have been found in 14% of raw vegetables in Peru (Nichols 2000).

19.1.1 Qualitative risk assessment

Application of composted catering waste will pose very low risk to grazing animals and humans because:-

- Oocyst concentrations likely to be low in food.
- Cryptosporidiosis is endemic in cattle and sheep in UK
- Cannot multiply outside host
- Destroyed by composting temperatures (55°C)
- Some decay on soil

19.2 Toxoplamsa gondii

This parasite infects a broad spectrum of vertebrates including birds. Domestic and feral cats are the definitive hosts, but other mammals, including humans, can be infected following the ingestion of undercooked meat, or by ingestion of the oocysts from soil contaminated by cat faeces.

Humans

Among the most important diseases transmitted to man by mutton and goat meat, toxoplasmosis remains the greatest threat, particularly in immuno-compromised people and in pregnant women (Pepin *et al.* 1997). Man is infected through consumption of inadequately cooked meat from infected secondary host species such as agricultural animals. Toxoplasmosis is common within many countries of the world and is usually a sub-clinical condition. In pregnant women, infection can lead to mental retardation and loss of vision in their congenitally infected children (cited in Nichols 2000).

Studies of pregnant women in Norway found that eating raw or uncooked meat was the major risk factor for acute toxoplasma infection. An epidemiological study of risk factors for recent toxoplasma infection in pregnant women in Southern Italy found a strong association with eating cured pork and raw meat (cited in Warnekulasuriya *et al.* 1998).

Warnekulasuriya *et al.* (1998) detected viable *T. gondii* in one out of 67 ready-to-eat cured meat samples indicating a failure of the commercial curing process.

The incidence of Toxoplasma infection appears to vary between regions of the UK. Highest prevalence of infection is found in N. Ireland and Wales where, for example, over 30% adult blood donors have been found to be infected. This compares to southern Scotland and some English regions where a prevalence of 15-20% has been found in comparable groups. National studies have reported an annual incidence of approximately 0.2% in women of childbearing age.

Animals

Infects over 200 species of animals including birds and humans. *T. gondii* is found in the tissues of food animals and is an important cause of abortion and mortality in sheep and goats throughout the world. Outbreaks of infection have been associated with food, milk and environmental contamination with cat faeces.

The definitive host is the cat and reproduction occurs in gut epithelium. Oocysts excreted in cat faeces. These infect secondary hosts by ingestion where organism enters the blood which distributes them to a range of tissues in which they become encysted. The life cycle is complete when cats eat infected tissue. Secondary hosts become infected via encysted bradyzoites in tissues.

19.2.2 Epidemiology

Sheep are frequently maintained in an environment significantly contaminated with oocysts and infection follows ingestion of contaminated food or water, with pasture perhaps being the most common source of infection. Fields treated with manure and bedding from farm animals where cats lives can cause infection and cats defecating in farm feeds, such as hay and stored grain, will pose a risk (cited in Buxton et al. 1998).

Buxton (1998) writes that “cat faeces can create a large, potent, long-lasting source of infection for sheep. Thus, oocyst contamination of farm feeds and bedding, as well as pasture, is a threat to susceptible, pregnant sheep and goats and is closely related to the number and distribution of cats”.

19.2.3 Source Term

Meat

Most species of livestock, including sheep, goats and pigs, are infected with T. gondii. Prevalence rates vary in pigs, but generally exceed 10%-20% in most countries. Infection rates are higher in breeding populations than in market pigs, reflecting that time of exposure is a factor in acquiring toxoplasmosis (Gamble 1997).

May be high incidence in UK already. Paper in early 90s claims 50% of meat in Europe contains *T gondii*. Warnekulasuriya *et al.* (1998) highlight the need for improved methods for detecting toxoplasma contamination of food. This suggests that there is not enough data to undertake quantitative risk assessments for *T. gondii* in meat.

Cat faeces

It has been calculated that less than 1% of all cats are shedding oocysts at one time (cited in Buxton 1998). Buxton (1998) also cites that a single defecation from a cat may contain as many as 10^7 oocysts.

Feral cats can be infected through food i.e. voles, mice and shrews, which are persistently infected with *T. gondii*. In addition, mice can pass the infection on in utero without causing clinical disease. Thus mice present a reservoir of *T. gondii* tissue cyst infection for cats which can exist for a long time within that mouse population.

The contribution of cats to the spread of toxoplasmosis in pigs cannot be over-emphasised (Gamble 1997). In studies of prevalence exposure to *T. gondii* in farm cats, seropositive rates were found to range from 41.9%-70.7% (cited in Gamble 1997). Although cats only shed for one week, 1.8% of cats tested in one study were found to be sheeding oocysts actively (cited in Gamble 1997). Management of cats on farms is a major control measure.

19.2.4 Pathway Term

T. gondii tachyzoites survive for at least 7 d at 4°C in goats' milk (Walsh *et al.* 1999).

Data show that *T. gondii* is killed in 336s at 49°C, in 44s at 55°C, and in 6s at 61°C. The parasite survived microwave cooking (cited in Warnekulasuriya *et al*. 1998), although this is probably due to uneven heating (Gamble 1997). Dubey *et al.* (1990) examined the effect of heat on the infectivity of *T. gondii* tissue cysts.

Tissue cysts remained viable at temperatures slightly below freezing, but parasites were inactivated virtually instantaneously at temperatures of –9.4°C and lower (Gamble 1997).

Relatively few studies have examined the efficiency of the curing process for the inactivation of *T. gondii* (cited in Warnekulasuriya *et al.* 1998). Smoking of meats or salting appears to be destructive,

19.2.5 Receptor Term

The human infective dose for *T. gondii* is not established (cited in Warnekulasuriya *et al*. 1998).

When susceptible pregnant sheep were orally doses with 2,000 *T. gondii* oocysts, less than 18% of lambs were born live and viable (cited in Buxton 1998). Thus, a dose of 2000 oocysts gives an 82% chance of still birth. This is plotted on a dose-response curve in **Figure 19.1** and a negative exponential curve is plotted through the point. The LD_{50} is about 800 oocysts and the risk from a single oocyst, r, is 0.00085. Vaccination increased the chance of live birth from an oral dose of 2,000 oocysts from 18% to 75%.

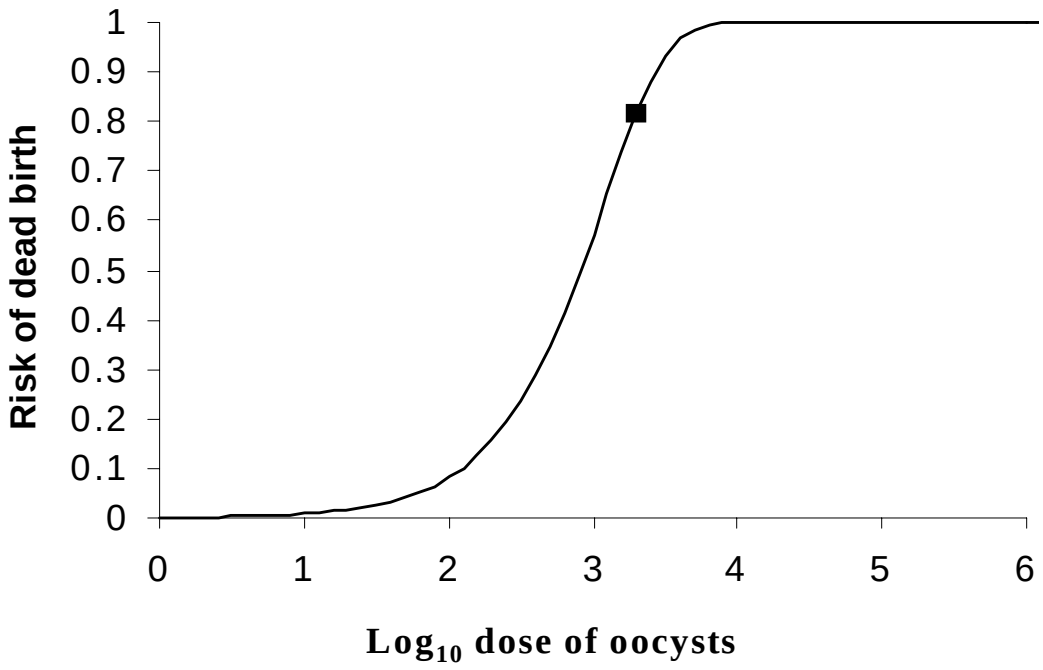

Figure 19.1 Negative exponential dose-response curve (r = 0.00085) for risk of dead birth in sheep from oral exposure to *T. gondii*. Data from Buxton (1998) who cited an experiment in which less than 18% of births were live after an oral dose of 2,000 oocysts. This is plotted as the point.

According to numbers cited by Buxton (1998), an oral infectious dose for sheep is in between 40 and 200 oocysts of *T. gondii*.

19.2.6 A quantitative risk assessment for *T. gondii*

A quantitative risk assessment for domestic cat faeces in MSW is set out in **Table 19.1**. This is based on some broad assumptions.

According to the calculation 5.4 x 10^{12} *Toxoplasma gondii* oocysts are composted per year. On the basis that composting destroys 4.7-logs and 500,000 tonnes of compost is produced per year, the model predicts 218 oocysts $tonne^{-1}$ of compost. Applying 10

tonnes ha^{-1} year^{-1} tilled into a depth of 0.1 m give a soil concentration of 1.5 oocysts tonne^{-1} soil. There are no data for decay of *T. gondii* oocysts on soil. It is assumed that they decay at a similar rate to *Cryptosporidium parvum* oocysts (**Section 4.3.1**) which is 1-log in 12 weeks (0.0119-log d^{-1}) at 4°C. Assuming a sheep eats 0.2 kg soil day^{-1} then the annual cumulative exposure may be calculated as:-

Equation 11 $$\sum_{t=1}^{t=365} 0.20.N_o 10^{-0.0119.t}$$

Table 19.1 A quantitative risk assessment for *Toxoplasma gondii* in domestic cat faeces in composted MSW.

Term	Value
Number of homes in UK	30,000,000
Percentage of homes with cats	25%
Number of domestic cats	7,500,000
Percentage of cats shedding (Buxton 1998)	1%
Number of infected domestics cats	75,000
Percentage of homes using cat litter	20%
Number of infected cats discharging to cat litter	15,000
Number of *Toxoplasma* oocysts discharged per cat per day to cat litter (Buxton 1998)	10,000,000
Number of *Toxoplasma* oocysts discharged per day to cat litter	1.5 x 10^{11}
Number of Toxoplasma oocysts discharged per year to cat litter	5.4 x 10^{13}
Percent of MSW composted	10%
Number of *Toxoplasma* oocysts composted per year	5.4 x 10^{12} per year

where N_0 is the concentration of *T. gondii* (0.0015 oocysts kg^{-1}) in the soil at t = 0. According to **Equation 11**, each sheep grazing on land to which compost has been applied ingest 0.011 oocyst sheep^{-1} year^{-1}. This assumes there is no grazing ban.

Allowing for a 2 month grazing ban

Assuming decay occurs according to **Equation 11** a cummulative annual exposure may be calculated for days 61 to 425. This gives a cumulative exposure of 0.002 oocysts sheep per year.

Risks to grazing sheep

The risk of a pregnant ewe giving a dead birth after ingestion of a single oocyst, according to **Figure 19.1** is given by r = 0.00085. The risk to pregnant ewes giving a dead birth after grazing on land to which composted catering waste has been applied is 9.1 x 10^{-6} sheep^{-1} year^{-1}. This is on the basis of no grazing ban. Assuming 0.52% of the England/Wales sheep herd graze on land to which compost has been applied, then 157,000 sheep are exposed (**Table 4.3**). Of those just 1.5 pregnant sheep would give a dead birth per year because of *T. gondii* in composted MSW waste (according to the

assumptions in **Table 19.1**). This reduces to just 0.27 sheep give a dead birth year^{-1} (individual risk to sheep of 1.7 x 10^{-6} sheep^{-1} year^{-1}) if a two month grazing ban is implemented.

A qualitative assessment of risk

Since farmyard cats appear to be a major source of infection for sheep and goats, it would seem that the risks to grazing sheep and goats from application of composted catering waste would present a relatively lower risk. A qualitative risk assessment is set out in **Table 19.2**.

Table 19.2 Summary – Qualitative risk assessment for *Toxoplasma* in composted catering waste.

	Good news	Bad news
Source – cats	Incidence may be low in domestic cats which would contribute cat faeces to the waste bin (cat litter) Farm yard cats more likely to have higher incidence – and defecate directly on the pasture land. Farm animals not allowed to graze on land to which composted catering waste has been applied. However, cats on farms may defecate in animal feeds, on pasture land, and in pens. Risk from pet faeces in composted MSW may be relatively lower	
Source - meat		No effective inspection procedures for post-slaughter detection of *T. gondii* in meat are available 50% of pork may be infected.
Pathway barriers for composted catering waste	Freezing meat will destroy cysts Composting temperatures will destroy cysts	
Receptor humans – meat and root crops		No ID_{50} data for humans – but likely to be similar to *C. parvum*, i.e. highly infectious
Receptor sheep/goats – grazing on land	Time of exposure is a factor in acquiring toxoplasmosis – only limited exposure likely on land to which composted catering waste has been applied	*T. gondii* causes abortion in sheep

It is concluded that the risks to sheep and goats from *Toxoplasma gondii* from application of composted catering waste to land should be considered low in the light of the risks posed by farm yard cats, which may defecate directly in the feed.

20. Endemic Faecal Bacterial Pathogens – Salmonellas, *e. Coli* o157 and Campylobacters

Poultry meat, beef and lamb products, will undoubtedly in some cases be contaminated with endemic faecal pathogens such as salmonellas, *E. coli* O157 and campylobacters.

In UK poultry responsible for 50% of outbreaks of salmonellosis compared to 2% for beef. Compared to *Salmonella*, chicken is more contaminated with *Campylobacter* (Durfrenne *et al.* 2001).

20.1 Source Term

20.1.1 Salmonellas

Salmonella prevalence and levels are higher in ground poultry than in ground beef (FSIS 1998). Indeed average MPN salmonella levels for both ground chicken and turkey are more than 20 times higher than for ground beef (FSIS 1998). For this reason only poultry products are considered here.

Prevalence

Overall 29% of raw chicken obtained from retail outlets in South Wales was positive for salmonellas (**Table 20.1**). A breakdown of the prevalence according to chicken parts is shown in **Table 20.2**.

Table 20.1 Percentage of Salmonella spp. positive raw chicken obtained from local supermarket chains (n = 175) and butchers' shops (n = 125) during a seven month study in South Wales (Harrison *et al.* 2001).

Supermarket	33%
Butchers' shop	24%
Overall	29%

Table 20.2 Breakdown of the percentage of salmonella spp. positive raw chicken samples by chicken type. Data from Harrison *et al*. (2001) for South Wales. Number of samples in parentheses.

	Whole chicken	Breast	Wings
Supermarkets	69 (55)	35 (55)	17 (65)
Butchers	30 (40)	30 (40)	13 (45)
Overall	53 (95)	33 (95)	15 (110)

Salmonella densities on chicken meat

There are surprisingly few studies reporting concentrations of salmonellas in chicken products.

Before scalding

Kotula and Pandya (1995) performed a quantitative study of *Salmonella* spp. and *campylobacter* spp. on the skin of broiler chickens directly after killing in the processing plant. The mean counts ranged from 5.4 to 6.9 $\log_{10}$ g^{-1} skin for salmonella.

FSIS (1998) report salmonella counts for ground chicken and turkey. From each sample that had a qualitatively positive result for Salmonella, a frozen subsample was quantitatively analysed for MPN. The highest MPN value for the ground poultry products was 2,300 MPN g^{-1}.

A statistical distribution for salmonella counts in ground poultry meat was obtained from (www.). The counts are shown in **Table 20.3**. The total loadings on the 110 samples was 3,041 MPN g^{-1}. The arithmetic mean is therefore 27.6 MPN g^{-1}.

Table 20.3 Frequency distribution for counts on salmonellas on ground poultry meat.

MPN counts g^{-1}	Frequency	Product
0.023	0	0
0.1	1	0.1
0.23	2	0.46
1	53	53
2.3	23	52.9
10	22	220
23	5	115
100	3	300
230	0	0
1000	0	0
2300	1	2300
10000	0	0
23000	0	0
Total	110	3,041

After scalding

Dufrenne *et al.* (2001) report quantitative data on contamination levels with Salmonella and Campylobacter in chicken and chicken products in The Netherlands at retail level. These are presented in **Table 20.4**, together with the arithmetic mean counts.

Table 20.4 Counts of samonellas (MPN cacrass^{-1}) estimated from graphs in Figure 1 from Dufrenne *et al.* (2001).

Sample	Fresh	Frozen
1	50	50
2	50	50
3	50	50
4	50	50
5	50	10
6	50	10
7	50	10
8	50	10
9	10	10
10	10	10
11	10	10
12	10	10
13	10	10
14	10	10
15	10	10
16	10	10
17	10	10
18	10	10
19	10	10
20	10	10
21	10	10
22	10	10
23	10	10
24	10	10
25	10	10
26	10	3
27	10	3
28	10	3
29	10	3
30	10	3
31	10	10
32	10	20
33	10	30
34	10	30
35	10	40
36	2	40
37	3	40
38	3	50
39	3	50
40	10	50
41	10	100
42	20	200
43	50	300
44	60	1100
45	100	
46	1100	
Average	**44.1**	**56.5**

20.1.2 Campylobacters

Campylobacter is a human bacterial pathogen that has been associated with raw poultry. Thus, approximately, 80% of raw chickens sold in the UK are contaminated with thermophilic campylobacters and they can be found on the carcasses at levels as high as several thousands per cm^2 of skin (Corry and Atabay, 2001). A survey carried out by the FSA in chickens on retail sale revealed campylobacters in 50% of chickens (FSA pers comm.). Berrang *et al.* (2001) undertook a study to determine if broiler chicken parts (collected at a commercial plant) without skin are less contaminated with campylobacter than those parts with skin. They found that no campylobacter were recovered from meat collected from the breasts or thighs, and only 2 of 10 drumstick meat samples had detectable levels of campylobacter. However, 9 of 10 breast skin, 10 of 10 thigh skin, and 8 of 10 drumstick skin samples were positive for campylobacter, with between 2 and 3 $\log_{10}$ CFU/g. Similar trends were noted for coliform and *E. coli*. Chicken skin harbours a large number of campylobacters in the early stages of processing. Berrang *et al*. (2000a) quote values as high as 4.5 log CFU/g of campylobacter in breast skin (excluding feathers) before the carcass entered the scald tank (**Table 20.5**). After scalding and picking, Campylobacter can be recovered in high numbers from whole carcass rinses or skin swabs of broilers (Berrang *et al*. (2000b). Indeed Berrang *at al*. (2000b) conclude that a postscald treatment gentle enough not to alter the carcass appearance or meat quality would not effectively lower Campylobacter, *E. coli* or coliform bacteria counts. Contamination of the meat can occur in the process of removing skin with feathers from a carcass (Berrang *et al.* 2001).

Table 20.5 Campylobacter counts ($\log_{10}$) recovered from external and internal organs of prescald broiler carcasses from a commercial processing plant. Data from Berrang *et al.* (2000a).

Visit to plant*	Feathers	Skin	Crop	Ceca	Colon
Weight of organ(g)	1.5	6.5	5.1	7.8	3.1
1	4.6	3.1	4.7	6.9	6.8
2	5.5	3.9	4.5	7.7	7.7
3	6.1	4.5	5.0	7.3	7.2

*six chickens analysed per plant

Harrison *et al*. (2001) demonstrated a high incidence of campylobacter in retail chickens in South Wales (**Table 20.6**).

Table 20.6 Breakdown of the percentage of Campylobacter spp. positive raw chicken samples by chicken type. Data from Harrison *et al*. (2001) for South Wales. Numbers of samples in parentheses.

	Whole chicken	Breast	Wings
Supermarkets	82 (55)	82 (55)	71 (65)
Butchers	70 (40)	58 (40)	53 (45)
Overall	77 (95)	72 (95)	64 (110)

Campylobacter counts in chicken portions purchased at a retail outlet in the USA (Berrang *et al*. 2001) are presented in **Table 20.7**.

Table 20.7 Campylobacter populations recovered from breasts, thighs and drumsticks of broilers purchased at a retail outlet and skinned in the laboratory. Data from Berrang *et al.* (2001).

Sample	Mean weight (g)	Campylobacter $\log_{10}$ CFU/part	no. positive
Breast skin and meat	315	2.8	9/10
Breast skin	35	2.6	9/10
Breast meat	247	2.6	9/10
Thigh skin and meat	194	2.7	8/10
Thigh skin	40	2.5	9/10
Thigh meat	160	2.7	8/10
Drum skin and meat	118	2.1	6/10
Drum meat	113	2.2	6/10

Campylobacter counts recovered from chicken parts purchased, with or without skin, from retail outlets are presented in **Table 20.8**. The differences are probably not of practical significance. Indeed thighs and drumsticks had similar microbial populations regardless of the presence or absence of skin. Berrang *et al.* (2001) concluded that no trends were evident for store-bought skin-on versus skin-off product. Thus, removal of chicken skin prior to disposal to the waste bin does not appear to lower the counts of campylobacters on the meat. It should be noted in the case of drum-sticks that the incidence of campylobacter-positives was roughly halved. Berrang *et al.* (2001) suggest that more research is needed to determine the effects of skin removal.

Table 20.8 Campylobacter populations recovered from breast meat, thigh meat and drumstick meat of broilers purchased at a retail outlet with and without skin. Data from Berrang *et al.* (2001).

Sample	**Mean weight (g)**	**Campylobacter $\log_{10}$ CFU/part**	**no. positive**
Breast skin on	353	2.5	17/20
Breast skin off	209	2.1	16/20
Thigh skin on	157	2.4	17/20
Thigh skin off	131	2.2	20/20
Drum skin on	126	2.2	17/20
Drum skin off	124	2.2	9/20

Total Campylobacter loadings on a chicken

The arithmetic mean campylobacter loadings for breast skin and meat ($10^{2.8}/325 = 2.0$ CFU g^{-1}), thigh skin and meat ($10^{2.7}/194 = 2.6$ CFU g^{-1}) and drum stick skin and meat ($10^{2.1}/118 = 1.1$ CFU g^{-1}) as calculated from **Table 20.7** give a pooled density of 2.0 cfu

g^{-1} of chicken. A carcass weighing 2,260 g would therefore contain 4,534 cfu of Campylobacter.

Dufrenne *et al*. (2001) presented Campylobacter MPN measured in fresh chicken at retail level in The Netherlands. These are plotted as a log-Normal distribution in **Figure 20.1**. It is apparent that many of the data points are censored (i.e. contained counts well above or below the limits of detection). The straight line plotted through the "non-censored" data represents a log-Normal distribution, the arithmetic mean of which is 85,500 MPN $carcass^{-1}$.

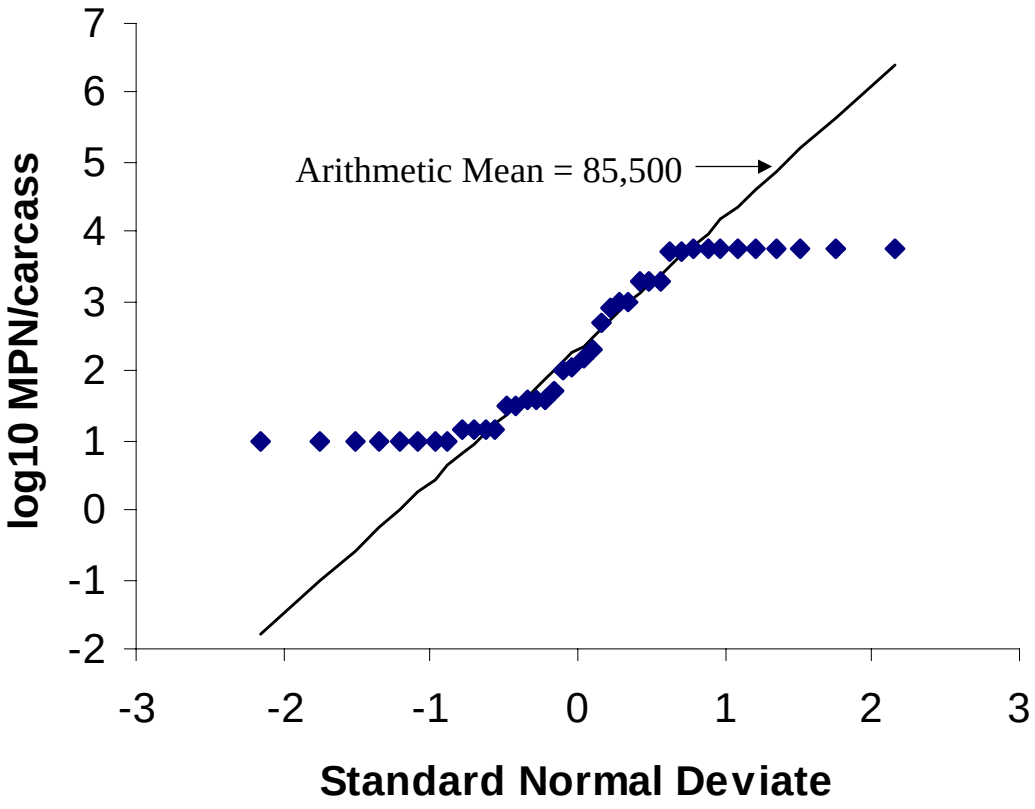

Figure 20.1 Estimation of the arithmetic mean campylobacter loading (MPN/carcass) for fresh chickens in The Netherlands. See text for details. Data from Dufrenne *et al*. (2001).

20.2 Regrowth of bacterial pathogens on discarded food.

20.2.1 Campylobacters

The thermophilic campylobacters require unusual conditions for growth (atmosphere with ~10% CO_2 and 6% O_2, temperature above 30°C and high relative humidity).

Multiplication in food or the food processing environment thus seems unlikely, at least in the UK climate (Corry and Atabay, 2001). For thermophilic campylobacters, the problem for food is how long can they survive, rather than how to prevent them growing. This topic has been reviewed in depth (see Corry and Atabay, 2001). Survival in food is better at lower temperatures (4°C) than higher temperatures (20°C).

20.2.2 *E. coli* O157:H7

Berry and Koohmaraie (2001) studied the influence of various levels of endogenous beef bacterial microflora on the growth and survival of *E. coli* O157:H7 on bovine carcass surface tissue. Regardless of the microflora level, there was no substantial growth of *E. coli* O157:H7 on bovine carcass tissue (BCT) at 4°C under either aerobic conditions or vacuum-packaged conditions. Instead, viable cell numbers at 4°C remained constant, with no reduction in numbers associated with the different beef microflora levels. However, *E.coli* O157:H7 grew on all BCT stored at 12C, regardless of the microflora inoculation treatment, reaching higher populations on aerobic samples than on vacuum-packaged samples in 10 d. The presence of beef microflora did delay the onset of growth or slow the growth of the pathogen.

Table 20.9 Summary of *E. coli* O157:H7 growth on bovine carcass tissue under aerobic conditions. Data from Berry and Koohmaraie (2001).

Temperature	No added microflora	High microflora
4°C	0.2-log	0-log
12°C	5-log	3.0-log

Note, for the purpose of risk assessment a 4-log growth is allowed for.

20.3 Routes of exposure for endemic bacterial pathogens through composted catering waste

It should be noted that the catering waste was intended for human consumption (albeit after cooking, in the case of any raw meat products discarded to the bin uncooked).

There are two pathways by which exposure to humans, through application of composted catering waste to agricultural land, could be increased. These are:-

- Contamination of vegetable crops grown on land
- Increase in the incidence of VTEC in cattle and hence in the level of VTEC contamination on raw meats.

20.4 A quantitative model for *E. coli* O157 in catering waste

Source Term

In a one year study (April 1995 to March 1996) of rectal faeces collected immediately after slaughter, Chapman *et al*. (1997) reported *E. coli* O157 in:-

- 752 (15.7%) of 4,800 cattle;
- 22 (2.2%) of 1,000 sheep;
- 4 (0.4%) of 1,000 pigs; and
- 0 of 1,000 chickens

For the purpose of *E. coli* O157 risk assessment, the model considers contamination of lamb/mutton and beef products.

According to data from the MLC, 708,000 tonnes of beef and 359,000 tonnes of sheep meat were produced (or imported) in the UK for consumption. For the purpose of risk assessment, it is assumed that 0.01% (w/w) of the meat was faeces. In the absence of any data, it is believed that this is a realistic worst-case assumption.

Cattle/beef

Tuttle *et al.* (1999) reported median *E. coli* O157:H7 concentrations of 1.5 g^{-1} of ground beef patty. This number could be used directly in the risk assessment.

Shere *et al.* (1999) reported concentrations of up to 87,000 cfu *E. coli* O157 g^{-1} of faeces from infected heifers. Assuming 15.7% of cattle faeces is infected (Chapman *et al.* 1997) and that 0.01% (w/w) of meat sold in shops is faeces, then the total *E. coli* O157 loading on the 708,000 tonnes of beef products in the UK is calculated as

7.08×10^{11} g x 0.0001 x 87,000 cfu g^{-1} x 0.157 = 9.7×10^{11} *E. coli* O157 per year.

The *E. coli* O157 concentration is therefore $9.7 \times 10^{11} / 7.08 \times 10^{11} = 1.4$ g^{-1} of beef, which is in good agreement with that reported by Tuttle *et al.* (1999).

Sheep/lamb

The arithmetic mean *E. coli* O157 concentration in faeces from sheep and lambs during the New Deer outbreak may be estimated from data of Strachan *et al.* (2001) as 365,500 g^{-1}. This assumes that the count of $>10^6$ g^{-1} recorded in one lamb was 10^7 g^{-1}. Assuming 2.2% of sheep faeces is infected (Chapman *et al*. 1997) and that 0.01% (w/w) of lamb sold in shops is faeces, then the total *E. coli* O157 loading on the sheep meat products in the UK is calculated as:-

3.59×10^{11} x 0.0001 x 365,500 cfu g^{-1} x 0.022 = 2.88×10^{11} *E. coli* O157 per year.

Total E. coli O157 loading on food.

The total loading on food in the UK is therefore 1.2×10^{12} *E. coli* O157 per year.

This may seem a large loading for food. However, Doyle and Schoeni (1987) in as study of retail fresh meats and poultry in Canada concluded that *E. coli* O157:H7 is not a rare contaminant of meats. Indeed, they isolated *E. coli* O157:H7 from 3.7% of beef, 1.5% of pork, 1.5% of poultry and 2% of lamb samples from grocery stores and retail outlets.

Pathway Term

The assumptions describing the pathway are summarised in **Table 20.10**. The calculation assumes that 1% of meat in the UK is discarded uncooked to the catering waste bin (**Section 2.2.2**). The model assumes that *E. coli* O157 multiply 10,000-fold in the meat (**Table 20.9**) and that source separation/composting gives a 4.7-log reduction (**Table 7.1**).

Table 20.10 Estimating *E. coli* O157 loadings in composted catering waste in UK.

		E. coli O157 loading (cfu year^{-1})
Total loading in catering waste		1.2×10^{12}
Uncooked meat discarded to catering waste bin	1%	1.2×10^{10}
Regrowth of *E. coli* O157 on meat in catering waste	10,000-fold	1.2×10^{14}
Destruction by composting	4.7-logs	2.5×10^{9}

Thus after composting, some 2.5×10^{9} *E. coli* O157 will be left in the composted residues (**Table 20.10**). Assuming 500,000 tonnes per year of compost is produced then the *E. coli* O157 concentration in compost is 5,010 tonne^{-1}. A gardener consuming 1 g of compost will therefor be exposed to a dose of 0.005 *E. coli* O157.

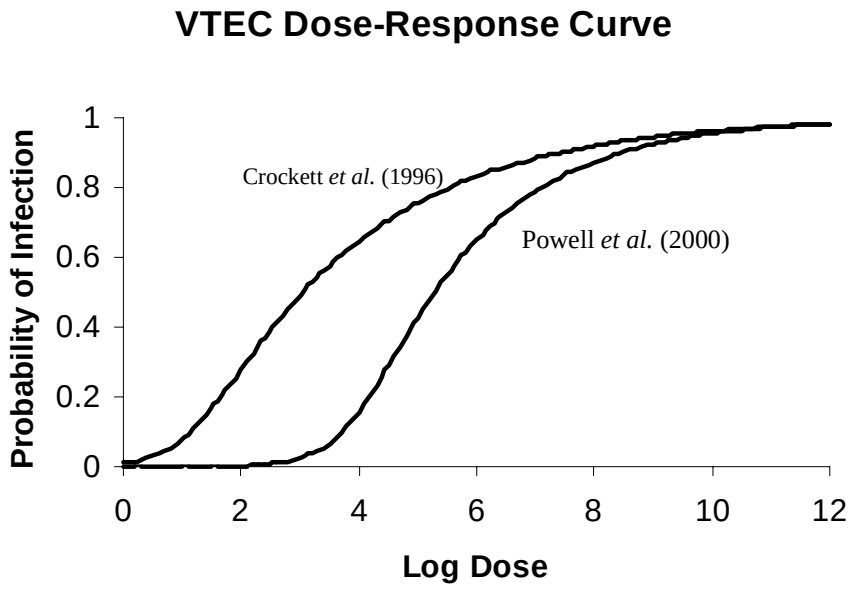

Figure 20.2 Dose-response curve for *E. coli* O157:H7. "Most-likely" Beta-Poisson model fitted using $\alpha = 0.221$; $\beta = 8{,}722.46$ (from Powell *et al*. 2000). Also shown is Beta-Poisson model ($\alpha = 0.49$; $N_{50} = 1{,}130$) proposed by Crockett *et al.* (1996) and supported by data of Strachan *et al*. (2001).

A dose-response curve calibrated by Powell *et al*. (2000) from the 1992-93 Pacific North West hamburger outbreak is presented in **Figure 20.2**. The ID_{50} is estimated at 190,000 cfu. According to the Beta-Poisson model fitted, a dose of 0.005 *E. coli* O157 would present a risk of 1.3 x 10^{-7} g^{-1} of compost ingested. This is the most likely value. There is huge uncertainty in the dose-response curve. Thus the maximum estimate of the dose-response envelope according to Powell *et al.* (2000) would predict a risk of 0.86 x 10^{-4} g^{-1} of compost ingested. Furthermore, a recent analysis of the New Deer (Scotland) *E. coli* O157 outbreak (Strachan *et al*. 2001) supports the dose-response curve proposed by Crockett *et al*. (1996). This is also shown in **Figure 20.2** and predicts much higher risks for low doses of *E. coli* O157. According to this dose-response curve the risk from ingestion of a gram of compost would be 0.5 x 10^{-4} $person^{-1}$ g^{-1}.

Comparison of loadings with manure and sewage sludge

The net *E. coli* O157 loading from application of composted catering wastes to land is compared with those estimated for manure and sewage sludge in **Table 20.11**. These figures do not allow for any decay on the soil. The loading for manure is "optimistic" in that it allows for a 4.5-log destruction during a 90 day storage period on the farm. However, the loading from catering waste is a factor of 5,200-fold lower than from manures and 44-fold lower than for treated-sewage sludge, even allowing for a 10,000-fold regrowth factor of the *E. coli* O157 in the catering waste.

Manure

Some 77% of manure is of bovine origin and likely to contain *E. coli* O157. There are no regulations regarding treatment of manure prior to application to agricultural land, although there may be considerable die-off of *E. coli* O157 during storage of the manures. The levels of *E. coli* O157 on vegetables from application of composted catering waste residue will be remote in comparison with the loadings from application of manures.

Gale (unpublished results for FSA) estimated that raw manure contains 8.2 x 10^{9} *E. coli* O157 $tonne^{-1}$. This is based on the same assumptions used for composted catering waste (**Table 20.10**), i.e. 15.7% of cattle are infected and there are 87,000 cfu g^{-1} of faeces. It is also assumed that manure is 60% cattle faeces. 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. Thus, the concentrations of *E. coli* O157 in stored manure is 2.6 x 10^{5} $tonne^{-1}$. In England and Wales, some 52,300,000 tonnes of manure are applied to land (ADAS data). The total loading of *E. coli* O157 on land from application of "stored" manure is therefore 1.3 x 10^{13} cfu in England and Wales.

Sewage sludge

Gale (unpublished results for UKWIR, EA, DEFRA) calculated the *E. coli* O157 concentration in treated sewage sludge. The main source of *E. coli* O157 in raw sewage is from slaughter of cattle and sheep at abattoirs. The model uses DEFRA data that 3.13 million cattle and 15.86 million sheep are slaughtered annually at abattoirs in England and Wales. It assumes, on the basis of expert advice, that 5% of faecal material in animals slaughtered at abattoirs enters the sewage treatment works, either directly through sewer or by tankering. As for the catering waste model, it is assumed that bovine faeces from an infected animal contains 87,000 cfu *E. coli* O157 g^{-1}. Assuming

15.7% of cattle are infected (Chapman *et al.* 1997) and that each bovine contains 10 kg of faeces at time of slaughter, then 2.1 x 10^{13} *E. coli* O157 $year^{-1}$ enter sewage works across England and Wales from the slaughter of cattle. The arithmetic mean *E. coli* O157 concentration in faeces from sheep and lambs during the New Deer (Scotland) outbreak may be estimated from data of Strachan *et al.* (2001) as 365,500 g^{-1}. This assumes that the count of >10^6 g^{-1} recorded in one lamb was 10^7 g^{-1}. Assuming 2.2% of sheep flocks are infected (Chapman *et al.* 1997) and each sheep has 1 kg of faeces at point of slaughter, then the arithmetic mean *E. coli* O157 loading across sewage works in England and Wales from slaughter of sheep in abattoirs is 6.3 x 10^{12} cfu $year^{-1}$. In England and Wales, 967,000 tonnes dry solids (tds) of sewage sludge are produced annually (Gale and Stanfield 2001). On the basis of the salmonella model (Gale 2001b), 82.9% of *E. coli* O157 in raw sewage would partition into the raw sludge at the works. Thus, the arithmetic mean loading from cattle and sheep combined in raw sewage sludge in England and Wales is 2.4 x 10^7 *E. coli* O157 tds^{-1}.

Conventional sludge treatment (e.g. anaerobic digestion) is required to give a 2-log destruction of *E. coli* and pathogens according to the according to the UK Code of Practice for Agricultural Use of Sewage Sludge (Department of the Environment 1996). The predicted *E. coli* O157 concentration in treated sludge is therefore 2.4 x 10^5 cfu tds^{-1}. In England and Wales, 480,000 tds of sewage sludge are applied annually to agricultural land (WRc 1998). The total *E. coli* O157 loading is therefore 1.1 x 10^{11} in England and Wales.

Table 20.11 Comparison of *predicted E. coli* O157 loadings (cfu) in "stored" manure, conventionally-treated sewage sludge and composted catering waste in England/Wales.

Manure	Sewage sludge	Composted catering waste*
1.3 x 10^{13}	1.1 x 10^{11}	2.5 x 10^9

*UK

Conclusion

Since the *E. coli* O157 loading is so much lower than for treated sewage sludge and manures, it is concluded that application of composted catering waste to land will have little impact with respect to *E. coli* O157.

20.5 Quantitative Risk Assessment for Campylobacter

A total of 6.15 x 10^8 broilers are slaughtered annually in the UK. The arithmetic mean Campylobacter density on chickens at retail level in The Netherlands is estimated at 85,500 cfu $carcass^{-1}$ (**Figure 20.1**). The total *Campylobacter* loading on carcasses at retail would therefore be 6.15 x 10^8 x 85,500 = 5.26 x 10^{13} cfu $year^{-1}$. Assuming 1% of chicken is discarded uncooked to the catering waste bin, then challenge for composting processes would be 5.26 x 10^{11} cfu $year^{-1}$. Assuming no growth of campylobacters in the meat, and allowing for a 4.7-log destruction by composting, the total loading in the treated compost would be 1.05 x 10^7 campylobacters $year^{-1}$. Assuming 500,000 tonnes of compost is produced annually, the concentration of campylobacters in compost would be 20.98 $tonne^{-1}$. On dilution in the soil, this would give 0.14 campylobacters $tonne^{-1}$ of soil.

Public health risks from contact with compost

The concentration of campylobacters in compost is estimated at 20.98 tonne^{-1}. A gardener ingesting 1 g of compost would be exposed to 2.1 x 10^{-5} campylobacters. A Beta-Poisson dose-response curve (α = 0.15; β = 7.9) for *Campylobacter jejuni* has been calibrated by Teunis *et al*. (1999). Using this model an exposure of 2.1 x 10^{-5} campylobacters translates into a risk of 0.4 x 10^{-6} person^{-1} g^{-1} of compost ingested.

Public health risks from consumption of crops grown in fields

The concentration of campylobacters in soil to which composted catering waste has been applied is 0.14 tonne^{-1}. Over a period of 16 days, a 2-log decay was observed (**Section 4.3**). Over 2 months therefore a 7-8 log reduction might be expected. Allowing for just a 2-log decay gives a soil concentration of 0.0014 campylobacters tonne^{-1} soil. Assuming each person ingests 0.384 kg person^{-1} day^{-1} of root crop (of which 2%(w/w) is soil) then the annual exposure to humans is 3.9 x 10^{-6} campylobacters person^{-1} year^{-1}. This assumes the root crops are unwashed and not cooked. Using the Beta-Poisson dose-response curve (α = 0.15; β = 7.9) for *Campylobacter jejuni* calibrated by Teunis *et al*. (1999), this translates into a risk of 7.5 x 10^{-8} person^{-1} year^{-1}.

20.6 Risk assessment for salmonellas

Total salmonella loading in chickens in England and Wales

In 2000, some 1.39 x 10^{12} g (y^{-1}) of chicken (excluding “Dead on Arrivals”) was potentially produced at slaughter houses (**Section 2.5**). On the basis of that the arithmetic mean salmonella concentration in chicken carcasses at retail level is 56.5 MPN carcass^{-1} (scalded; see **Table 20.4**), there would be 3.5 x 10^{10} salmonellas y^{-1} in the food chain through poultry.

Assuming 1% of chicken meat goes uncooked into catering waste, then 3.5 x 10^{8} salmonellas would enter the catering waste.

Growth and survival in food

Salmonellas are resistant to freezing. They do not survive temperatures above 70°C. With exception of *S senftenberg*, salmonellas are destroyed by 56°C for 10 to 20 min. There is better heat tolerance at low water activity and in high fat foods. Salmonellas can survive for years in certain dry foods and survive for several months in 20% salt particularly where there is a high fat content (e.g. sausages).

In food, growth occurs at temperatures between 8 and 45°C with water activity above 0.94 and in pH range 4 – 8. According to the Food MicroModel (FSA), up to 7-log increases can occur through growth on foods. The presence of indigenous bacteria will reduce this somewhat. As a worst cases assumption, the model assumes a 6-log increase in salmonellas through growth on the food in the catering waste.

Estimating salmonella concentrations in compost from catering waste.

Allowing for a 6-log growth of the salmonellas on the chicken meat and a 4.7-log destruction by the composting process, would leave 6.9 x 10^9 salmonellas in the compost y^{-1}. Assuming the annual production of compost is 500,000 tonnes y^{-1}, then the arithmetic mean concentration of salmonellas in compost is 13,860 $tonne^{-1}$.

Concentrations in soil

Applying compost at a rate of 10 tds ha^{-1} y^{-1} tilled into a depth of 10 cm would give an arithmetic mean soil concentration of 92.4 salmonellas $tonne^{-1}$ soil. The salmonella concentration predicted in soil from application of sewage sludge treated by a process which removes 2-logs of salmonellas is 3.52 x 10^5 $tonne^{-1}$ soil (Gale, P. 2002 Report for UK Water Industry, Environment Agency and DEFRA). This is a factor of 3,800–fold greater than from application of compost containing catering waste. Watkins and Sleath (1981) reported salmonella concentrations in soil of 130 / 100g (i.e. 1,300,000 $tonne^{-1}$) of soil immediately after incorporation of raw sewage sludge. The salmonella counts had reduced to <1 / 100 g after six weeks (Watkins and Sleath 1981). Initial estimates for salmonella loadings from application of manures in the UK are higher than for treated sewage sludge (Gale and Stanfield, preliminary risk assessment for FSA).

Health risks from contact with compost

A gardener ingesting 1 g of compost would be exposed to 0.0139 salmonellas. A Beta-Poisson dose-response curve (α = 0.4059; β = 5,308) for salmonella has been calibrated by FAO/WHO (2000). Using this model an exposure of 0.0139 salmonellas translates into a risk of 1.06 x 10^{-6} $person^{-1}$ g^{-1} of compost ingested.

20.6.2 Regrowth of salmonellas in compost

Sidhu *et al*. (2001) studied the role of indigenous microorganisms in suppression of salmonella regrowth in composted biosolids. They concluded that the indigenous microflora is the single most important factor that controls regrowth of salmonella in composted biosolids.

Public health risks from consumption of crops grown in fields

The concentration of salmonellas in soil to which composted catering waste has been applied is 92 $tonne^{-1}$. Over a period of 5 weeks, Watkins and Sleath (1981) reported a 2-log decay of salmonellas on soil (**Section 4.3**). Over 2 months therefore a 4 log reduction might be expected. Allowing for just a 2-log decay gives a soil concentration of 0.92 salmonellas $tonne^{-1}$ soil. Assuming each person ingests 0.384 kg $person^{-1}$ day^{-1} of root crop (of which 2%(w/w) is soil) then the annual exposure to humans is 0.0026 salmonellas $person^{-1}$ $year^{-1}$. This assumes the root crops are unwashed and not cooked. Using the Beta-Poisson dose-response curve (α = 0.4059; β = 5,308) for salmonella calibrated by FAO/WHO (2000), this translates into a risk of 2.0 x 10^{-7} $person^{-1}$ $year^{-1}$.

20.6.3 Conclusions

Assuming a 6-log growth of salmonellas in catering waste:-

1. The risks from salmonella on soil through application of compost containing catering waste are at least 1,000-fold lower than from application of conventionally-treated sewage sludge.

2. The risks to a gardener ingesting a gram of compost are in the order of 10^{-6} person g^{-1} ingested.

3. The risks to consumers from eating raw and unwashed root crops are $<10^{-6}$ person^{-1} year^{-1}.

21. Trichinae (*t. Spiralis*)

A nematode (roundworm) that causes trichinellosis, that can affect all species of carnivores and omnivores. Particularly common in pigs, rats, cats and humans. Recent epidemiological studies indicate that the transmission of *Trichinella spiralis* in agricultural ecosystems may involve a complex interaction among swine, rats and resident wild and feral animals. Indeed, the role of the rat in the on-farm transmission may well be important, since pigs will eat rats. Leiby *et al.* (1990) reported that on one farm in the US over a 25 month period, 42.4% (n = 443) rats were infected. Cannibalism is a major vehicle for spread of *T. spiralis* in pigs (Schad *et al.* 1987). Furthermore rats eat the porcine carcasses.

21.1 Epidemiology

In the US, risk of exposure of pigs to trichinae are greatly reduced by:-

- Banning the feeding of uncooked catering waste products and animal carcasses to pigs;
- Minimising exposure of pigs to live wildlife;
- Maintaining effective rodent control programme; and
- Removing dead pigs immediately to avoid cannibalism.

Stability

Remain viable in rotten meat for up to 4 months, survive salting, drying and smoking. Meat inspection is insensitive method of control and outbreaks have occurred via meat that had been inspected.

Influence of temperature

Freezing kills (-15 C for 64 min) and cooking kills: roasting (77 °C) more than adequate to achieve core temperature of 60 °C.

Recommendations for treating pig swill include 100 °C for 30 minutes. *T. spirallis* killed in 47 minutes at 52 °C and 6 min at 55 °C and <1 min at 60°C (www.aphis.usda.gov/vs/trichinae/docs/fact_sheet.htm). These temperatures are only effective if taken as core temperature and there is even temp distribution. US Code of Federal Regulations for pork products require 2h at 52 °C.

International Commission on Trichenellosis has issued time and temperature regulations for pork muscle. These range from 49.0 °C for 21 h up to 61 °C for I minute. At 58 to >60 °C time and temperature do not need to be monitored if none of the meat is more than 50mm thick and refrigeration does not begin with 5 minutes.

Transmission

Lives in the small intestine produces 1000 larvae per female which penetrate gut wall and via blood to muscle where they lodge as a cyst and remain viable for years.

Infection due to ingestion of contaminated meat or rarely by contact with larvae in faeces. Meat is usual vehicle. Pigs from uncooked meat scraps. Also some inference that rats can transmit to livestock. Mortality in human outbreaks can be 40%.

Prevalence

Uncommon in Western Europe but prevalent in Spain and Eastern Europe and also in South America. Trichinellosis occurs in the US and Canada with human infection associated with eating undercooked game animals.

21.2 Risk assessment

The question to be addressed is how likely is composted catering waste to initiate infection of *T. spiralis* in pigs in the UK. Cannibalism and rodents could then spread the disease to other pigs within the herd. The source, pathway, receptor qualitative approach is summarised in **Table 21.1** and suggests that the risks are relatively low.

Table 21.1 Summary – Qualitative risk assessment for *T. spiralis* in composted catering waste.

	Good news	Bad news
Source – pets	Incidence likely to be low in pets in the UK – larvae unlikely to be present in cat faeces	
Source - meat	Effective inspection procedures for post-slaughter detection of *T. spiralis* in meat are available Prevalence in domestic swine very low in Western Europe – Denmark and Netherlands are free	Curing may not be effective Illegally imported pork is not inspected
Pathway barriers for composted catering waste	**Unlike *Toxoplasmsa*, there does not seem to be an environmental stage in the life cycle – raises questions about how long larvae could survive on soil**.	Freezing – temperatures <10°C required for destruction Long times required for inactivation at composting temperatures (e.g. 2 hours at 52.2°C).
Receptor humans – meat and root crops		*T. spiralis* is highly pathogenic in people
Receptor sheep/goats – grazing on land		Assume ID_{50} is a single larvae

A quantitative risk assessment is now described.

Larval densities as high as 395 g^{-1} tongue muscle have been reported by Schad *et al*. (1987). Arithmetic mean larval densities for infected porcine muscle ranged between 60.3 g^{-1} and 260g^{-1} (Schad *et al*. 1987).

Assuming a pig carcass contains 43.7 kg of skeletal muscle and 0.26 kg heart muscle (**Table 14.3**) then an infected pig carcass with arithmetic mean of 260 larvae g^{-1} would contain 1.14 x 10^7 larvae. Assuming 10,000 porcine carcasses infected with *T. spiralis* enter the UK food chain each year, then total challenge would be 1.14 x 10^{11} larvae in pork.

Allowing for 1% of meat to be discarded uncooked to the catering waste bin, then 1.14 x 10^9 larvae would enter the compost process. The 4.7 log reduction would reduce this to 22,800 larvae per year. Diluting these into 500,000 tonnes of compost would give 0.046 larvae $tonne^{-1}$ of compost.

21.2.1 Risks to gardeners ingesting compost are remote

A “gardener” ingesting a gram of compost would therefore be exposed to 4.6 x 10^{-8} larvae. Assuming the risk of infection from ingestion a single larva is 0.5 (i.e. ID_{50} = 1 larva), the risk would be 2.3 x 10^{-8} g^{-1} of compost ingested.

21.2.2 Risks to pigs from application of compost to soil

Applying 10 tds compost ha^{-1} $year^{-1}$ gives 0.46 larvae ha^{-1}, which assuming the compost is tilled in gives a soil concentration of 0.0003 larvae $tonne^{-1}$ soil (at t = 0).

Allowing for no decay of larvae in the soil, then an animal ingesting 0.41 kg soil $animal^{-1}$ day^{-1} would be exposed to 4.5 x 10^{-5} larvae $animal^{-1}$ $year^{-1}$. Assuming the ID_{50} = 1 larva, then risk is 2.2 x 10^{-5} $animal^{-1}$ $year^{-1}$. Assuming 0.52% of the pig herd is exposed, then this would translate into one case per year in England and Wales. This is based on the “What If?” assumption that 10,000 infected porcine carcasses enter the human food chain each year.

21.2.3 Risks to vegetable consumers

A person consuming 0.384 kg d^{-1} of root crop would ingest 2.8 kg $year^{-1}$ of soil, assuming the root crop is 2% (w/w) soil. This is clearly a worst case assumption. Since soil to which compost has been applied contain 0.0003 larvae $tonne^{-1}$, the annual exposure would be 8.5 x 10^{-7} larvae $person^{-1}$ $year^{-1}$. This translates into a risk of 4.2 x 10^{-7} $person^{-1}$ $year^{-1}$ (assuming the ID_{50} is 1 larva).

22. *Clostridium Botulinum* (Botulism)

22.1 Types of botulism

22.1.1 Infant botulism

Infant botulism is extremely rare but occurs when the baby ingests spores which germinate to produce the bacterial cells that reproduce in the gut and release toxin. In most adults and older children, this would not happen because the natural defenses which have developed in an adult gut would prevent the germination and growth of C. botulinum. In some babies, these defenses have not yet developed, and so this gives the infection a chance to get a foothold and produce the toxin.

22.1.2 Foodborne botulism (Food poisoning)

Foodborne botulism occurs when the spores have germinated and the bacteria have reproduced in an environment outside the body and produced toxin. This environment is usually a foodstuff in an airtight environmnet. However, toxin can form in loose wet mince-meat exposed to the atmosphere, suggesting the potential for bacterial activity in rotting catering waste.

In spite of the wide distribution of *C. botulinum* spores in the soil and on fruits and vegetables, botulism is uncommon. For botulism to result, the organism must multiply and form its toxin in the food before consumption.

Botulinum toxin is one of the most potent substances known and the lethal dose for humans can be less than 1 microgram, depending on the toxin type and route of administration. Individuals vary in their degree of susceptibility to botulinum toxin. There are seven specific types of botulinum toxin, designated by the letters A to G; of these, only types A, B, E and rarely, type F, are known to have caused illness in humans. Types C, D and G are known to cause illness in animals (including cattle). Unlike the spores, botulinum toxin is readily inactivated by heat treatment (85 °C for 5 minutes), although the time and temperature may vary between toxin types. Thus foodborne botulism is usually transmitted by foods that are not cooked, or have not been heated thoroughly before eating.

Certain types of foods, particularly acidic foods such as canned tomatoes, could protect botulinum toxin against heat inactivation. Most botulinum toxins appear to be more resistant to heat inactivation between pH 4.5 – 5.0. For example, at pH 6.2, type A toxin is inactivated by heating at 79°C for 8 minutes whereas, at pH 4.2, it takes 15 minutes at the same temperature. The toxin is rapidly inactivated by standard potable water treatments (chlorination) although chlorine levels in water can vary.

22.1.3 Botulism outbreaks in farm animals

During June 2001, 141 cattle in a herd of 164 dairy cows died (Cobb *et al.* 2002). The epidemic lasted for 21 days. Cattle are usually affected by *C. botulinum* types C or D.

The sources of types C and D toxins are usually putrefied carcasses of birds or small animals which have contaminated the water supply, a feed hopper or silo, pasture or the bedding material.

22.2 Infectious and lethal doses

22.2.1 Lethal doses for the toxin

The lethal dose for botulinum neurotoxin is around 0.5 - 1ηg/kg body weight for mammals.

22.2.2 ID_{50} for spores

The infectious/lethal dose of spores is hard to estimate in humans. In a very small number of human infants the infectious dose is low (perhaps <100 spores) but in the large majority of human infants consumption of this number of organisms would have no effect. In the vast majority of adult mammals (probably >99.9%) consumption of low numbers of spores will have no obvious effect (M. Brett, pers. comm.).

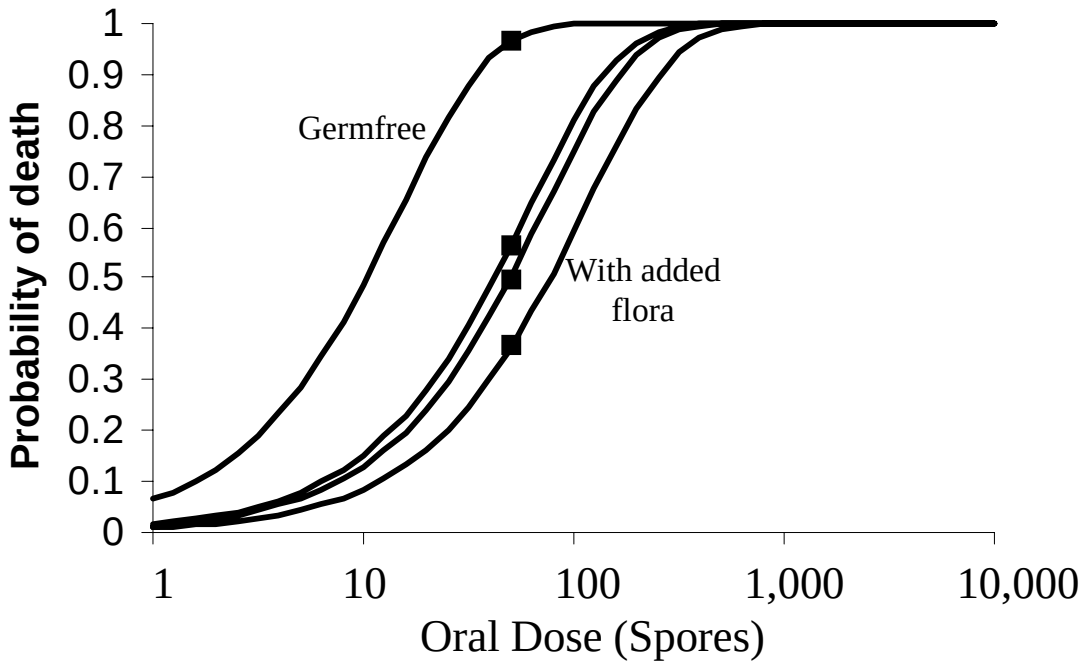

Figure 22.1 Effect of limited intestinal flora on dose-response of spores of *Clostridium botulinum* type A in mice. Negative exponential dose response curves fitted to data of Wells *et al*. (1982) using parameters in Table 22.1.

Rodents have an age-dependent susceptibility to enteric colonisation by *C. botulinum*. The intestinal microflora play an important role in determining the susceptibility of mice to enteric colonisation by *C. botulinum*. Adult mice are resistant to challenges of $>10^5$ spores, but the germ-free counterparts are infected when fed 10 spores (Moberg and Sugiyama 1979). Moreover, the highly susceptible germ-free adult mice become resistant to infection when they acquire the intestinal organisms indigenous to conventional adult mice. The values of *r* for the four dose-response curves in **Figure 22.1** are presented in **Table 22.1**. The dose-response curve used is the negative exponential model:-

Equation 12 $p = 1 - e^{-rN}$ **,**

Where *r* is the risk from a single spore and *N* is the dose of spores. Thus, the probability (*r*) of death after ingestion of a single spore for a germ-free mouse is 0.067 (6.7%). This value should be used in a risk assessment for infant botulism.

Table 22.1 Parameters for dose-response curves in Figure 22.1

	Germfree	CRAS flora	LC flora	LCB flora
No. of death	29	11	21	27
No. exposed	30	30	42	48
Proportion killed	0.966667	0.366667	0.5	0.5625
r (risk from a single spore)	0.067	0.009	0.0139	0.0165

Infant mammals begin to acquire microorganisms during the birth process and different species of microorganisms appear sequentially in the gastrointestinal tract until all available

Wells *et al*. (1982) studied the effect of adding different intestinal flora on the resistance of germ-free mice to challenge with a dose of 50 *C. botulinum* spores (**Figure 22.1**). Limited intestinal flora increased the ID_{50} to about 100 spores.

A risk assessment for cattle should assume that the ID_{50} for adults cows is >100,000 on the basis of the data of Moberg and Sugiyama (1979).

22.3 Heat resistance of *C. botulinum* spores

This ranges from values such that spores can be killed by 80°C for 10 minutes, to those that are unaffected by boiling. The constituents of compost may have a protective effect on spores, and so increase their heat resistance. Thus at least some spores may survive heat treatment, and, if they do survive are likely to be stimulated to germinate by the heating step, followed by multiplication of cells.

22.4 Growth of *C. botulinum* spores in composting and in food

Composting processes could provide microenvironments which are either sufficiently anaerobic or where the redox potential is sufficiently reducing such that cells of *C. botulinum* can multiply. (Food, which is very rarely homogenous, has microenvironments that allow growth of particular organisms occur.) Growth occurs between 3.3°C and 40°C but is slow below approximately 12°C, so although it could perhaps be possible for cells to multiply after the heating step, there is unlikely to be a large increase in numbers within a few days.

According to the Food MicroModel, spores of *C. botulinum* could increase up to 10^7-10^8 g^{-1} of food at 19°C (**Figure 22.2**). It should be noted that anaerobic conditions suitable for growth may obtain even in foods not in closed containers. Toxin can form in loose wet mince-meat exposed to the atmosphere. Wrapping in film may lead to a reduction in the redox potential of a food.

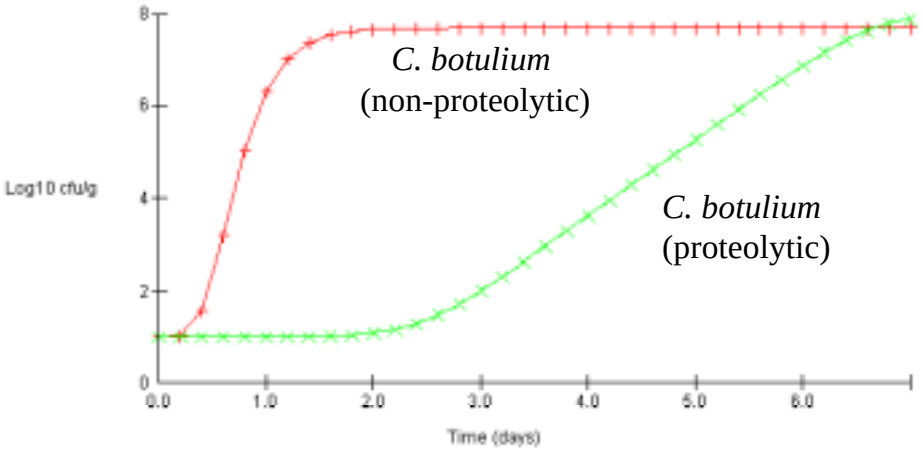

Figure 22.2 Predicted growth of *C. botulin* spores in food at 19ºC, pH 6.0 (Food MicroModel).

22.5 Survival in soil.

C. botulinum spores will survive for many years in soil. The neurotoxins are likely to completely lose activity within two weeks.

22.6 Number and frequency of spores in food.

C. botulinum was isolated from eleven 25g samples of vacuum packed bacon out of 263 samples investigated (M. Brett, pers. comm.). This 4.18% of bacon samples. This is probably representative of meat products. Unfortunately, most studies do not enumerate *C. botulinum,* or at least the figures are not published. This makes a quantitative risk assessment for *C. botulinum* difficult to undertake.

22.7 Risk assessment for infant botulism – a "What-if?" scenario

Some 2,039 tonnes are bacon are distributed to catering outlets weekly in the UK (**Table 2.6**). This is equivalent to 106,000 tonnes y^{-1}. Thus, 1,060 tonnes of bacon (1%) would be discarded to catering waste annually. Of this 4.18% (i.e. 44.4 tonnes) would be positive for *C. botulinum* spores. According to **Figure 22.2**, spores could grow up to 10^8 g^{-1} of meat. Assuming this happened in just 0.01% of the meat (i.e. that 0.01% of meat in the waste bin had the appropriate anaerobic conditions), then 4,443 g of bacon would contain counts of 10^8 spores g^{-1}. The total loading would be 4.43 x 10^{11} spores in the catering waste. Composting/biogas/storage will be effective at destroying the vegetative *C. botulinum* cells on the meat. However, assuming 10% of the vegetative cells managed to convert to heat-resistant spores, and assuming the total compost produced annually in the UK is 500,000 tonnes (**Section 5.2**), then the predicted spore concentration in the compost is 88,600 spores $tonne^{-1}$ or 0.089 g^{-1}. Using a value of r = 0.067 in **Equation 12**, an infant ingesting 1 g of compost would be exposed to a risk of 0.006 of developing infant botulism. This is a potential concern, and is well in excess of 10^{-6} $person^{-1}$ $year^{-1}$ risk of death judged as acceptable by the HSE.

22.8 Note on *C. botulinum* spore densities in soil

C. botulinum is commonly found in soil samples. Indeed soil densities range from 1-6 kg^{-1} (Great Britain) to 2,500 kg^{-1} in potato fields in The Netherlands (www.okstate.edu/ag/fapc/fsw/cbot/cbotpm.htm). The level "estimated" above for compost is 88 kg^{-1} – albeit based on many assumptions.

22.9 Risk assessment for cattle

On the basis of the model set out in **Section 22.7**, the spore concentration in soil after tilling in of the compost would be 591 spores $tonne^{-1}$. This is 0.591 kg^{-1} of soil and well below levels naturally occuring soil (see **Section 22.8**). This suggests that the additional exposure due to compost could be immaterial (depending on the appropriateness of the assumptions made in **Section 22.7**).

A cow ingesting 0.41 kg soil cow^{-1} day^{-1} would be exposed to 88 spores of *C. botulinum* $year^{-1}$. Adult mice are resistant to challenges of $>10^5$ spores (Moberg and Sugiyama 1979). To undertake a quantitative risk assessment for cattle really requires information on whether the ID_{50} is 10^6, 10^7 or 10^8 spores. However, the possibility that risks from catering waste disposed of to land-fill could be higher than for tilling in of composted catering waste, puts the risks into perspective for animal health (**Table 9.2**).

22.10 Conclusion

Lack of data on counts of spores in foods makes a quantitative risk assessment difficult for *C. botulinum* spores. However, the possibility that risks from catering waste disposed of to land-fill could be higher than for tilling in of composted catering waste, puts the risks into perspective for animal health (**Table 9.2**). Furthermore, on the basis of numerical calculations undertaken above, it appears that compost may have little effect of levels of spores already present in the soil.

In terms of public health risk, the main risk would be of infant botulism from spores remaining in the compost. Any toxin present in contaminated food will not present a risk in compost because the protein toxin will be inactivated by the heat process. In contrast, the *C. botulinum* spores will not be inactivated by composting/biogas and will not decay on the land.

On the basis of the data available it cannot be ruled out that the risks to infants would not exceed 10^{-6} $infant^{-1}$ g^{-1} of compost ingested. However, it should be borne in mind that the spore levels “estimated” in the compost for the risk assessment are in the range of those recorded in soil and lower than those reported for potato fields in The Netherlands.

It is therefore recommended that compost produced from catering wastes containing meat should contain a warning on the package along the lines of ensuring that infants should not be exposed.

23. Plant Pathogens

Selection of candidate pathogens

CSL undertook a review of the literature to examine the fate of plant pathogens during composting. The outcome of that study indicated that plant pathogenic bacteria and plant parasitic nematodes would be effectively inactivated by the temperatures obtained during composting, although some research suggests that some nematodes may survive. Also certain plant pathogenic fungi produce hardy resting spores and together with plant pathogenic viruses were considered sufficiently robust to survive the composting process.

Data was presented in the report which showed that certain fungi required temperatures of 55 – 70 °C for periods of days rather than hours to achieve inactivation.

A visit was undertaken to DEFRA Plant Health Division in York to identify the pathogens of concern. We considered whether there was scope to distinguish lower risk 'receptors' of compost, such as arable land, from higher risk ones, such as growing medium for vegetable transplant modules. On reflection it appeared that the organisms which we (and growers) would not want to see spread on arable land would be at least as resistant to composting as those of most concern for transplants. Five were selected as being significant, primarily based on their perceived temperature tolerance and so ability to survive composting. The particular viruses selected were chosen because they can be transmitted mechanically, i.e. without the need for a living vector which would be more susceptible to inactivation by composting.

The plant pathogens selected were:

1. *Sclerotium cepivorum* (white rot of onions)
2. *Plasmodiophora brassicae* (club root)
3. *Polymyxa betae* (vector of beet necrotic yellow vein virus which causes Rhizomania)
4. Potato spindle tuber viroid
5. Pepino mosaic virus

Risk assessment

Stage 1 : Source loading – Occurrence in wastes

Consideration needs to given to how vegetables infected with these diseases would be presented to the compost process, and in particular, the probability of infectious material occurring in catering waste.

Those diseases which cause putresence (white rot) of the crop or infect parts of the plant not normally consumed (club root) may not reach catering establishments or retail

outlets because of obvious physical defect. However, if they did, they would tend to be discarded to waste rather than eaten – and therefore end up in compost. Furthermore beetroot and leaves of spinach beet, which are also hosts of rhizomania, may well be present,

Sugar beet is a grown commercially and processed on an industrial scale. It is not consumed, as a vegetable so is unlikely to be present in any significant quantity in either catering waste.

The two virus disease since they can infect produce and do not give putrescence or other clinical signs which are off-putting. The produce is therefore still used in catering and so will be presented to composting through disposal of catering waste.

For both situations it is important to clearly establish the routes of transmission and loadings in plant tissues for these pathogens to properly establish the risks.

Stage 2 – Fate and behaviour during composting

There is only limited information on the survival of plant pathogens through composting processes, including different combinations of time and temperature. The Code of Practice for the Management of Agricultural and Horticultural Waste (1998) on disposal of waste cites 60-65° for several days as giving suitable control of pathogens in low risk material.

Some data on temperature tolerance suggests temperature between 55 – 70 °C will inactivate the plant pathogenic fungi for periods of time between 1 – 4 days. Less data is available on the temperature tolerance of plant pathogenic viruses. However, studies have recovered tobacco mosaic virus after 6 weeks exposure to temperatures between 50-75 °C. However, it was not established whether this was a true temperature effect or because of inadequate composting. It has also been reported that tobacco rattle virus survived 68 °C for 6 days although again the true temperature regime of the process was not established.

Recent evidence suggests that clubroot and Tobacco Mosaic Virus (TMV) are destroyed by windrow composting with 5 turns.

Ascertain whether temperature tolerance for plant pathogenic viruses is real or an artefact of inadequate treatment.

Environmental barriers

Dilution in the soil may have a reduced effect because of mobile spores. The range of such effects is proving difficult to ascertain for the pathogens of concern.

Some viruses can survive for long time periods (50 years) in the soil.

Tomatoes grown directly in compost have no dilution effect.

Stage 3 – Infectivity

Data on infectivity does not appear to exist for plant pathogens. Instead, for the purposes of risk assessment the precautionary principle is adopted in which it is assumed that one infective propagule will cause infection.

For highly infectious agents, the degree of dispersion in the soil becomes an important factor in assessing the risk. Indeed dispersion would increase the risk.

Differences between animal and plant pathogens

Animals will only ingest a small proportion of the compost spread on a field, whereas the roots of a host crop are likely to penetrate throughout the layer into which compost has been incorporated. Some plant pathogens have motile spores which actively seek hosts by detecting root exudates. Some are known to survive in soil for upwards of thirty years.

Risk assessment models are well understood and devloped for animal pathogens – but so far none have been developed for plant pathogens (to my knowledge).

Conclusion

The feasibility of undertaking quantitative risk assessments for plant pathogens would need to be assessed together with a review of the available data. There are a huge number of plant pathogens with different strategies to consider.

More detailed consideration of the risks from plant pathogens will be given at a later stage. In the meantime, controls in existing guidance on application and use of compost should be followed.

24. Other Forms Of Composting

24.1 Home-composting

A formal risk assessment for home-composting has not been undertaken here. However, there is scope for more effective Meat Exclusion by home composters, thus minimising the amount of meat being composted. Although the temperatures may be lower, the composting process may be performed for longer time periods, thus allowing for effective pathogen destruction. Furthermore, the home-produced compost is probably used in the garden of the composter and there is not spread to land for animal grazing. The risks to animal health from home composting are therefore likely to be very low.

However, by-pass could occur through wild animals and birds removing meat from the compost heap. It should be pointed out that is no different to feeding scraps of kitchen waste containing to wild animals.

24.2 Vermiculture

This is to be addressed in the next stage of the risk assessment

25. Addressing The Specific Objectives

The overall objective of this project was to determine the risks to animal, public and plant health from the land application of various categories of animal by-products and catering wastes containing meat.

The specific objectives listed in **Section 1.1** are:-

1. To compare the risks from the following three options

- Maintain the current ban on the use of composting and Biogas to dispose of animal by-products and catering waste containing meat;
- Adopt the new EC rules;
- Adopt specific UK standards;

2. To determine any minimum standards that might be needed to reduce those risks to an acceptable level.

These are now addressed.

25.1 Maintain the current ban on the use of composting and Biogas to dispose of catering waste containing meat

The risk assessment developed here underpins the case for rescinding the ban. Indeed, the landfill route for disposal of catering waste containing meat could actually present higher risks (**Section 9**).

25.2 Adopt the new EC rules

The EU conditions of 70°C for 1 hr with a 12 mm particle size are appropriate, providing it can be shown that no by-pass occurs.

25.3 Adopt specific UK standards

The minimum proposed standards are set out as:-

1. All steps are taken to eliminate any by-pass of the composting/biogas process, including ensuring that:-

- Raw catering waste material is not keep on livestock farms;
- Birds and small mammals do not gain access to the raw material;
- Raw material is delivered to a housed reception;

2. A two-barrier composting system is used for the “meat” fraction.

3. For each composting barrier, the catering waste reaches a temperature of 60°C for two days during composting, with the composting process being continued for at least 14 days;

4. The first treatment barrier be it “in-vessel” or windrow is housed or enclosed;

5. Windrows are turned at least three times;

6. “Dirty” end is kept separate from the “clean” end; i.e. different tools and equipment are used to handle the final product and the raw material;

7. Biogas is performed at 57°C; MGRT = 5 h; HRT = 19 days

8. The maximum particle size for composting is <40 cm diameter. This includes large joints of meat, e.g. discarded after freezer failures. For biogas, a maximum 5 cm (diameter) particle size is required;

9. Animals are not allowed to graze on land to which composted catering waste has been applied for a period of 2 months.

26. References

Anon (1994) Transmissible spongiform encephalopathies: A summary of present knowledge and research. London: HMSO.

Anon (1997) The digest of agricultural census statistics, United Kingdom, 1996. Ministry of Agriculture, Fisheries and Food, The Scottish Office Agriculture and Fisheries Department, Department of Agriculture for Northern Ireland, Welsh Office. The Stationary Office.

Bendixen, H.J. (1999) Hygienic safety – results of scientific investigations in Denmark (Sanitation requirements in Danish Biogas Plants). Pp 27- 47. In: IEA Bioenergy Workshop. Hygienic and environmental aspects of anaerobic digestion.

Bendixen, H.J. and Ammendrup (1992) Safeguards against pathogens in Biogas plants. Practical measures to prevent dissemination of pathogens and requirements for sanitation. The Danish Veterinary Service.

Berrang, M.E., Buhr, R.J. and Cason, J.A. (2000a) *Campylobacter* recovery from external and internal organs of commercial carcass prior to scalding. *Poultry Science*, **79**, 286-290.

Berrang, M.E., Dickens, J.A., and Musgrove, M.T. (2000b) Effects of hot water application after defeathering on the levels of Campylobacter, coliform bacteria, and Escherichia coli on broiler carcasses. *Poultry Science*, **79**, 1689-1693.

Berrang, M.E., Ladely, S.R. and Buhr, R.J. (2001) Presence and level of *Campylobacter*, coliforms, *Escherichia coli*, and total aerobic bacteria recovered from broiler parts with and without skin. *Journal of Food Protection*, **64(2)**, 184-188.

Berry, E.D. and Koohmaraie, M. (2001) Effect of different levels of beef bacterial microflora on the growth and survival of *Escherichia coli* O157:H7 on beef carcass tissue. *Journal of Food Protection*, **64(8)**, 1138-1144.

Burrows, R., Mann, J.A. and Goodridge, D. (1974) Swine vesicular disease: virological studies of experimental infections produced by the England/72 virus. *Journal of Hygiene, Cambridge* **72**, 135-143.

Buxton, D. (1998) Protozoan infections (*Toxoplasma gondii*, *Neospora caninum* and *Sarcocystis* spp.) in sheep and goats: recent advances. *Vet. Res*., **29**, 289-310.

Chapman, P.A., Siddons, C.A. Cerdan Malo, A.T. and Harkin, M.A. (1997). A 1-year study of *Escherichia coli* O157 in cattle, sheep, pigs and poultry. Epidemiology and Infection 119, 245-250.

Cobb, S.P., Hogg, R.A., Challoner, D.J., Brett, M.M., Livesey, C.T., Sharpe, R.T. and Jones, T.O. (2002) Suspected botulism in dairy cows and its implications for the safety of human food. *Vet. Record*, **150**, 5-8.

Corry, J.E.L. and Atabay, H.I. (2001) Poultry as a source of Campylobacter and related organisms*. Journal of Applied Microbiology*, **90**, 96S-114S.

Corso, B. (1997) Likelihood of introducing selected exotic diseases to domestic swine in the continental United States of America through uncooked swill. *Rev. sci. tech. Off. Int. Epiz.* **16(1)**, 199-206.

Cottral, G.E. (1969) Persistence of Foot-and-Mouth Disease Virus in animals, their products and the environment. *Bull. Off. Int. Epiz*. **71(3-4)**, 549-568.

Crockett, C.S., Haas, C.N., Fazil, A., Rose, J.B., and Gerba, C.P. (1996) Prevalence of shigellosis in the U.S.: consistency with dose-response infromation. *Int J. Food Microbiology*, **30**, 87-99.

Department of the Environment (1995) Landfill design, construction and operational practice. Waste Management Paper 26B. HMSO.

DNV (1997) Assessment of risk from possible BSE infectivity in dorsal root ganglia. Report to Ministry of Agriculture, Fisheries and Food and the Spongiform Encephalopathy Advisory Committee, Det Norske Veritas, Ref C7831.

DNV (2002) Presentation by Philip Comer at CCFRA risk assessment seminar, 24 Jan 2002.

Donaldson, A.I. (1997) Risks of spreading foot and mouth disease through milk and dairy products. *Rev. sci. tech. Off. Int. Epiz*., **16(1)** , 117-124.

Doyle, M.P. and Schoeni, J.L. (1987) Isolation of *Escherichia coli* O157:H7 from retial fresh meats and poultry. *Applied and Environmental M*icrobiology, **53(10)**, 2394-2396.

Dubey, J.P. Kotula, A.W., Sharar, A., Andrews, C.D., and Lindsay, D.S. (1990) Effect of high temperature on infectivity of Toxoplasma gondii tissue cysts in pork. *Journal of Parasitology*, **76**, 201-204.

Dufrenne, J., Ritmeester, W., Delfgou-van Asch, van Leusden, F. De Jonge, R. (2001) Quantification of the contamination of chicken and chicken products in The Netherlands with Salmonella and Campylobacter. *Journal of Food Protection*, **64(4)**, 538-541.

Edwards S. (2000) Survival and inactivation of classical swine fever virus. Veterinary Microbiology **73** pp 175-181.

EUSES (1997) European Union System for the evaluation of substances, User Manual, Joint Research Centre, European Commission EUR 17308 EN.

Farez, S. and Morley, R.S. (1997) Potential animal health hazards of pork and pork products. *Rev. sci. tech. Off. Int. Epiz*., **16(1)**, 65-78.

FAO/WHO (2000) Hazard identification and hazard characterisation of *Salmonella* in broilers and eggs. Joint FAO/WHO Expert consultation on risk assessment of mcirobiological hazards in foods, FAO Headquarters, Rome, Italy, 17-21 July 2000.

Foodservice Intelligence (2001). The Foodservice Market Meat Monitor, Quarterly report, January – March 2001. Report for MLC.

Gale, P. (2001) A REVIEW: Developments in microbiological risk assessment. *Journal of Applied Microbiology*, **91(2)**, 191-205.

Gale, P. and Stanfield, G. (2000) *Cryptosporidium* during a simulated outbreak. *Journal American Water Works Association*, **92(9)**, 105-116.

Gale, P. and Stanfield, G. (2001) Towards a quantitative risk assessment for BSE in sewage sludge. *Journal of Applied Microbiology*, **91**, 563-569.

Gamble, H.R. (1997) Parasites associated with pork and pork products. *Rev. sci. tech. Off. Int. Epiz.*, **16(2)** , 496-506.

Gibbens, J.C., Sharpe, C.E., Wilesmith, J.W., Mansley, L.M., Michalopoulou, E., Ryan, J.B.M. and Hudson, M. (2001) Descriptive epidemiology of the 2001 foot-and-mouth disease epidemic in Great Britain: the first five months. *The Veterinary Record*, **149**, 729-743.

Guittet, M., Le Coq, H. and Picault, J-P. (1997) Risques de transmission de la maladie de Newcastle par des produits avicoles contamines. *Rev. Sci. Tech. Off. Int. Epiz*., **16(1)**, 79-82.

Haas, B., Ahl, R., Bohm, R. and Strauch, D. (1995). Inactivation of viruses in liquid manure. *Rev. sci. tech. Off. Int. Epiz*., **14(2)**, 435-445.

Haas, C.N. (1996) How to average microbial densities to characterize risk. *Water Research* **30:4**, 1,036-1,038.

Harrison, W.A., Griffith, C.J., Tennant, D. and Peters, A.C. (2001) Incidence of *Campylobacter* and *Salmonella* isolated from retail chicken and associated packaging in South Wales. *Letters in Applied Microbiology*, **33**, 450-454.

Haug, R.T. (1993) Kinetics of Heat Inactivation. Chapter 5 In: The Practical Handbook of Compost Engineering, pp 161-203, Lewis Publishers, London.

Hedger, R.S. and Mann, J.A. (1989) Swine vesicular disease. In Virus infections of porcines (M.B. Pensaert, ed.) Elsevier Science Publishers, Amsterdam, 241-250.

Henderson, R.J. (1969) The outbreak of foot-and-mouth disease in Worcestershire. An epidemiology study: with special reference to spread of the disease by wind-carriage of the virus. *J. Hyg., Camb*., **46**, 394-402.

Henderson, W.M. and Brooksby, J.B. (1948) The survival of foot-and-mouth disease virus in meat and offal. *Journal of Hygiene*, **46**, 394-402.

Herniman KAJ, Medhurst PM, Wilson JN and Sellers RF (1973) The action of heat, chemicals and disinfectants on swine vesicular disease virus. *The Veterinary Record* **93** pp620-624.

Horan, N. and Lowe, P. (2001) Pathogens in biosolids and their significance in beneficial use programmes. Final Report to UKWIR, EA and DEFRA.

Joshua, R.S., Macauley, B.J. and Mitchell, H.J. (1998) Characterization of temperature and oxygen profiles in windrow processing systems. Compost Science & Utilisation, **6(4)**, 15-28.

Kao, R.R *et al*. Science Express, November 2001/10.1126/science.1067475.

Leiby, D.A., Duffy, C.H., Murrell, K.D., and Schad, G.A (1990) *Trichella spiralis* in an agricultural ecosystem: Transmission in the rat population. *J. Parasitology*, **76(3)**, 360-364.

Lung, A.J., Lin, C.-M., Kim, J.M., Marshall, M.R., Nordstedt, R., Thompson, N.P. and Wei, C.I. (2001) Destruction of *Escherichia coli* O157:H7 and *Salmonella Enteritidis* in cow manure composting. *Journal of Food Protection*, **64(9)**, 1309-1314.

MacDiarmid SC (1991) The importation into New Zealand of meat and meat products: A review of the risks to animal health. Ministry of Agriculture and Fisheries, Wellington, New Zealand, 180 pp.

Mann, J.A. and Hutchings, G.H. (1980) Swine vesicular disease: pathways of infection. *Journal of Hygiene, Cambridge* **84**, 355-363.

McKercher, P.D., Hess, W.R. and Hamdy, F. (1978) Residual viruses in pork products. *Applied and Environmental Microbiology*, **35(1)**, 142-145.

McVicar, J.W. (1984) Quantitative aspects of the transmission of African Swine Fever. *Am. J. Veterinary Research*. **45**(8) pp1535-1541.

Moberg, L.J. and Sugyama, H. (1979) Microbial ecological basis of infant botulism as studied with germfree mice. *Infection and Immunity*, **25(2)**, 653-657.

Nichols, G.L. (2000) Food-borne protozoa. *British Medical Bulletin*, **55(4)**, 209-235.

Panina, G.F., Civardi, A., Massirio, I., Scatozza, F., Baldini, P. and Palmia, F. (1989) Survival of foot and mouth disease virus in sausage meat products (Italian salami). *Int. J. Food Microbiol*., **8**, 141-148.

Pepin, N., Russo, P. and Pardon, P. (1997) Public health hazards from small ruminant meat products in Europe. *Rev. Sci. tech. Off. Int. Epiz*., **16(2)**, 415-425.

Plowright, W. and Parker, J. (1967) The stability of African Swine Fever Virus with particular reference to heat and pH inactivation. *Arch. Gesamte. Virusforsch*, **21(3),** 383-402.

Powell, M.R., Ebel, E., Schlosser, W., Wladerhaug, M. and Kause, J. (2000) Dose-response envelope for *Escherichia coli* O157:H7. *Quantitative Microbiology*, **2(2)**, 141-163.

Scott GR (1965) The virus of African swine Fever and its transmission. *Bull. Off. Int. Epizool*. **63**(5) pp645-677.

Sellers, R.F. (1971) Quantitative aspects of the spread of foot and mouth disease. *Vet. Bull.* **41**, 431-439.

Senne, D.A., Panigrahy, B. and Morgan, R.L. (1994) Effect of compsoting poultry carcasses on survival of exotic avain viruses: highly pathogenic avian influenza (HPAI) virus and adenovirus of egg drop syndrome. *Avian Diseases*, **38**; 733-737.

Schad, G.A., Duffy, C.H., Leiby, D.A., Murrell, K.D. and Zirkle, E.W. (1987) *Trichinella spiralis* in an agricultural ecosystem: transmission under natural and experimentally modified on-farm conditions. *J. Parasitol*. **73(1)**, 95-102.

Shere, J.A., Bartlett, K.J. and Kaspar, C.W. (1998). Longitudinal study of *Escherichia coli* O157:H7 dissemination on four dairy farms in Wisconsin. Applied and Environmental Microbiology 64(4), 1390-1399.

Sidhu, J., Gibbs, R.A., Ho, G.E. and Unkovich, I. (2001) The role of indigenous microorganisms in supression of salmonella regrowth in composted biosolids. *Water Research*, **35(4)**, 913-920.

Slater, R.A., Frederickson, J. and Gilbert, E.J. (1999) The State of Composting 1999. Results of The Composting Association's survey of UK composting facilities and collection systems in 1999.

Strachan, N.J.C., Fenlon, D.R. and Ogden, I.D. (2001). Modelling the vector pathway and infection of humans in an environmental outbreak of *Escherichia coli* O157. FEMS Microbiology Letters 203, 69-73.

Stenbro-Olsen, P.W., Earle-Mitchell, R., Gartland, K.M.A. and Collier, P.J. (1995) Temperature change as an indicator of the microbial activity and maturity of municipal green waste compost windrows. *Journal of Waste Management & Resource Recovery* **2(1),** 41-46.

Sutmoller, P. and Vose, D.J. (1997) Contamination of animal products: the minimum pathogen dose required to initiate infection. *Rev. sci. tech. Off. Int. Epiz*., **16(1)**, 30-32.

Teunis, P.F.M., Nagelkerke, N.J.D. and Haas, C.N. (1999) Dose response models for infectious gastroenteritis. *Risk Analysis* **19(6)**, 1251-1260.

Tiquia, S.M., Tam, N.F.Y. and Hodgkiss, I.J. (1998) Salmonella elimination during composting of spent pig litter. *Bioresource Technology*, **63**, 193-196.

Turner, C. and Burton, C.H. (1997) The inactivation of viruses in pig slurries: A review. *Bioresource Technology*, **61**, 9-20.

Turner, C., Williams, S.M., Burton, C.H., Cumby, T.R., Wilkinson, P.J. and Farrent, J.W. (1999) Pilot scale thermal treatment of pig slurry for the inactivation of animal virus pathogens. *J. Environ. Sci. Health*, **B34(6)**, 989-1007.

Turner, C., Williams, S.M. and Cumby, T.R. (2000) The inactivation of foot and mouth disease, Aujeszky's disease and classical swine fever viruses in pig slurry. *Journal of Applied Microbiology*, **89**, 760-767.

Tuttle, J., Comez, T., Doyle, M.P., Wells, J.G., Zhao, T., Tauxe, R.V. and Griffin, P.M. (1999) Lesson from a large outbreak of Escherichia coli O157:H7 infections: insights into the infectious dose and method of widespread contamination of hamburger patties. *Epidemiology and Infection*, **122**, 185-192.

Walsh, C.P., Hammond, S.E., Zajac, A.M. and Lindsay, D.S. (1999) Survival of Toxoplasma gondii tachyzoites in goat milk: potential source of human toxoplasmosis. *J. Eukaryotic Microbiology*, **46(5)**, 73S-74S.

Warnekulasuriya, M.R., Johnson, J.D. and Holliman, R.E. (1998) Detection of *Tozoplasma gondii* in cured meats. *International Journal of Food Microbiology*, **45** , 211-215.

Wells, C.L., Sugiyama, H., and Bland (1982) Resistance of mice with limited intestinal flora to enteric colonisation by *Clostridium botulinum*. The Journal of Infectious Diseases, 146(6), 791-796.

Wilkinson PJ and Donaldson AI (1977) Transmission studies with African Swine Fever virus. *J. Comp*. *Path* **87** pp497-501.

Appendix Response To The Survey Of Meat Preparation In Kitchens

Questionnaire To Catering Establishments

Replies were received from 13 catering establishments most of which were local to WRc-NSF. These establishments discarded very little meat as most (12) reported that amount of 1 percent or less. One establishment reported discarding greater amounts of meat but the amount varied between 1 and 5 percent. The main reason was that most (8) establishments received their meat pre-butchered.

Questionnaire To Domestic Households

There were 54 replies from staff at WRc-NSF to the survey into meat consumption in domestic households. The majority (18) of respondents estimated that around 5% of their meat was discarded uncooked. One household reported discarding 10% and another 20% of the meat purchased However, a similar number of households discarded less than 5 % with estimates being given as 1 % (7) of less than 1% (10) or none (2). Data was not available for 14 households.

Details were also sought on preparation of individual meats. The households (24) that consumed beef as bone-in-steak cooked the meat intact and discarded the bone afterwards. Trimmings from pork meat were discarded in the dustbin after cooking. The skin from chicken was left on the meat if the whole bird was cooked whereas the skin from chicken portions was skinned and discarded raw. The giblets were often discarded uncooked into the bin. Other meat such as bacon was either purchased rind-less or the rind discarded after cooking but occasionally discarded raw but outside to feed the birds rather than being put in the dustbin.

Very little meat is wasted in the households although rare events such as failure of the deep freeze have resulted in significant quantities being discarded.