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Food & Rural Affairs

What is the risk of chronic wasting disease being introduced into Great Britain?

An updated Qualitative Risk Assessment

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Summary

This assessment has been provided as an update to a previous version carried out in 2012, due to new evidence about the import of lures for deer hunting which contain deer urine. The previous assessment is available at:

http://webarchive.nationalarchives.gov.uk/20140507133914/http://www.defra.gov.uk/animal-diseases/files/qra_chronic-wasting-disease-121029.pdf

The previous assessment concentrated on the incursion of disease from North America through the imports of animal feed or the movement of contaminated clothing, footwear and equipment. The results suggested that import of pet feed was a non-negligible risk, but given the unlikely contact of resident deer in GB with such non-ruminant feed, this was considered overall a negligible to very low risk. The movement of contaminated clothing, footwear or equipment (particularly hunting equipment) could pose a very low risk, although the volume of contaminated soil which would need to be ingested to give rise to an infection is likely to be higher than would be present. There is a variable level uncertainty in all these assessments.

The new assessment focuses on an additional potential route of entry: the importation of natural deer urine lures. The main conclusions from this assessment are:

- In areas of North America where CWD has been reported, given that CWD is excreted in faeces, saliva, urine and blood, and survives in the environment for several years there is a **medium probability** that the deer urine in North America contains CWD (high uncertainty; depends on the source of deer used for production).

- The risk of a deer in GB being infected per 30 ml bottle of urine imported from the USA is **very low**, albeit with **high** uncertainty. Overall it is concluded that the risk of at least one infection of deer in the UK with CWD per year from deer urine lures imported from the USA is **medium**. This assumes a high number of 30 ml bottles imported per year from all areas of the USA.

- None of the species affected by CWD in North America are present in GB. For a British species to become infected with CWD following exposure, the dose and inherent susceptibility of the species will be important. Based on current scientific evidence Red deer (*Cervus elaphus elaphus*) are susceptible to CWD, Fallow deer (*Dama dama*) are likely to be less susceptible and Roe deer (*Capreolus capreolus*) have a gene conferring susceptibility. Therefore, it is likely that given exposure to an infectious dose of CWD, deer in GB could become infected with CWD.

Overall, the probability of importing CWD into GB from North America and causing infection in British deer is uncertain but likely to be **negligible to very low** via movement of deer hunters, other tourists and British servicemen and **very low** via imported (non-

ruminant) animal feed and **medium** for the use of lures. However, if it was imported and (a) deer did become infected with CWD, the consequences would be severe as eradication of the disease is impossible, it is clinically indistinguishable from BSE infection in deer (Dalgleish *et al.,* 2008) and populations of wild and farmed deer would be under threat.

The USA has implemented a Herd Certification Programme for farmed and captive cervids. So far, 29 States are approved for HCP status (APHIS, 2015). The list includes States such as Colorado, where CWD is present, therefore it is recommended that any sourcing of such natural urine lures should be not only from States with an HCP programme, but also from a herd which is registered as being regularly tested free of CWD.

Animal urine is not considered a commodity which is subject to animal by-products legislation for imports. Internet sales are common and although a license would be required, there are no conditions for the safe sourcing of such products. Deer urine lures are also available in Europe and may be produced from carcases of hunted deer. The use of deer urine produced from a species not present in Europe (such as white tailed deer) is questioned for its value with native GB deer according to the British Deer Society survey.

Background

Chronic wasting disease (CWD) is a highly infectious transmissible spongiform encephalopathy (TSE) that is circulating in the wild and farmed cervid populations of North America. It is the only TSE maintained in free-ranging wild animal populations. A feature of CWD is that it is able to transmit both directly (animal-to-animal) and indirectly via the contaminated environment. In particular, CWD prions are able to bind to and survive in the soil in a bio-available form for many years without any decrease in infectivity. This makes eradication of the disease from a wild population increasingly challenging.

Thus far, there have been no reported cases of CWD or other TSE in deer in Great Britain (GB). This is based on surveys of wild and farmed red deer (*Cervus elaphus elaphus*) (EFSA, 2011). Given the consequences of CWD observed in North America, it is of high importance that GB remains free of the disease. Further, as the clinical signs of CWD in deer are similar to those of deer experimentally infected with bovine spongiform encephalopathy (BSE), all infected deer would need to be tested to differentiate if they were infected with CWD or BSE to minimise the risk of BSE entering the human food chain via affected venison.

In 2015, the British Deer Society (BDS) carried out an online survey of BDS and BASC members to gather evidence about the use of deer urine as a lure. Fifteen percent of respondents (~1,800) answered yes about knowing that deer urine was used as a lure. Of the respondents, less than 2% responded yes to using such a product themselves. Of those that use the product, 50% had sourced the product from the USA, while 20% use more than a litre in volume a year and ~70% is natural (as opposed to synthetic).

Hazard identification

The hazard is identified as **Chronic Wasting Disease**

Chronic wasting disease (CWD) is a transmissible spongiform encephalopathy (TSE) affecting cervids. It is the only TSE maintained in free-ranging wild animal populations; other TSE's are mostly restricted to captive domestic livestock populations or humans. Chronic wasting disease was first identified as a clinical disease of captive mule deer in Colorado in 1967 and later classified as a TSE in 1978 (Williams & Miller, 2003). The origin of the disease is unknown and may have been a spontaneous TSE that arose in deer. Currently, natural infections of CWD have been reported in mule deer (*Odocoileus hemionus hemionus*), black-tailed deer (*Odocoileus hemionus columbianus*), white-tailed deer (*Odocoileus virginianus*), Rocky Mountain elk (*Cervus elphus nelsoni*), Shira's moose (*Alces alces shirasi*) and mule deer and white-tailed deer hybrids (Hamir *et al*., 2008). Other species of elk may also be susceptible. The disease is restricted to North America except for isolated cases of infected elk being exported from Canada to South Korea (Williams & Miller, 2003). In the last decade, with increased CWD testing, a more widespread distribution of CWD within North America has been observed and the geographic distribution is increasing (APHIS, 2015) (Figure 1).

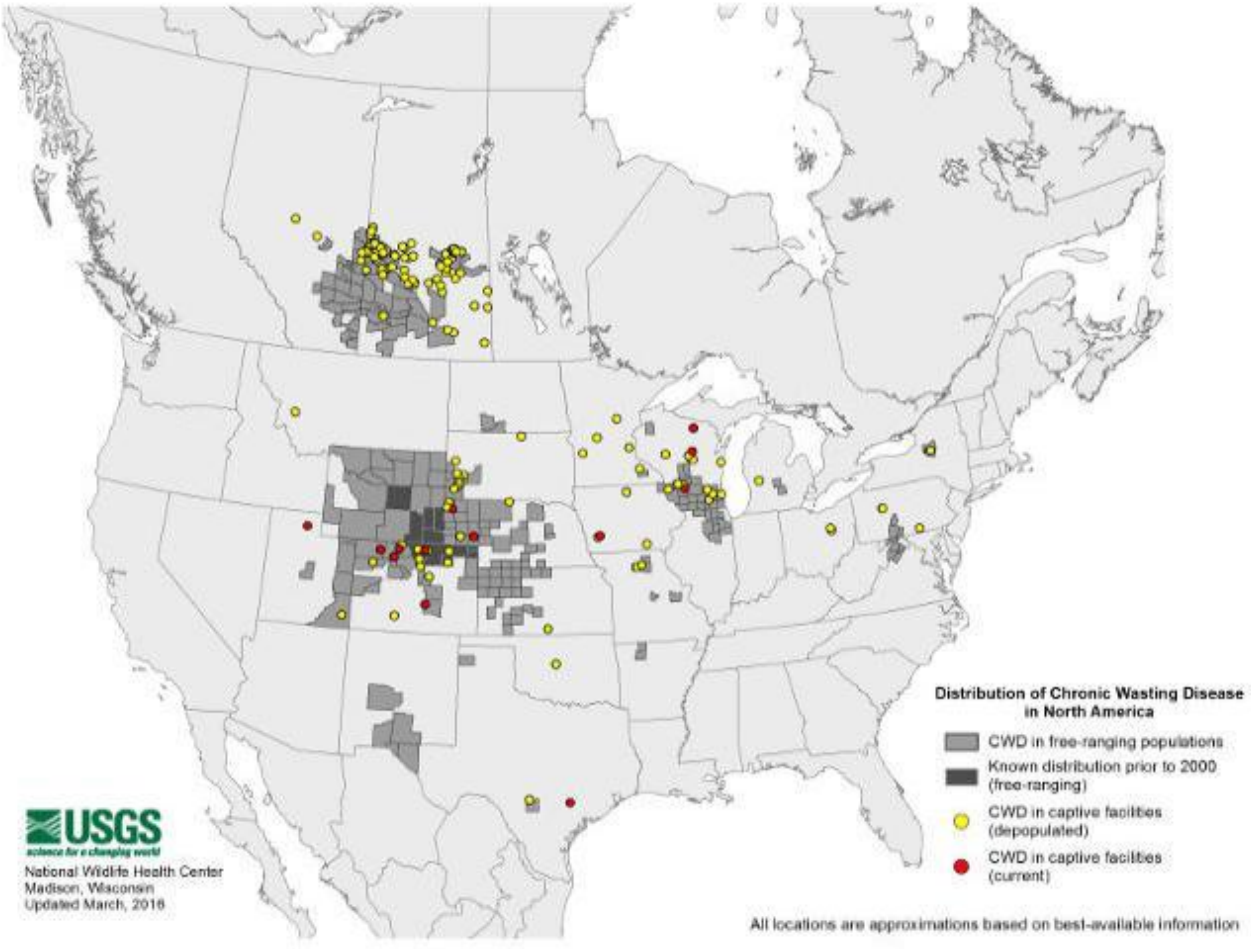


Figure 1: Current range of CWD in North America (USGS, 2016)

This more widespread distribution may be due to enhanced surveillance but also to natural migration of cervids and translocation of infected animals by humans (EFSA, 2011). Within affected areas, the prevalence varies. In the endemic area of Wyoming, for example, the prevalence of CWD in mule deer has increased from approximately 11% in 1997 to 36% in 2007 (Almberg *et al*., 2011). In such areas, population declines of deer of up to 30 to 50% have been observed (Almberg *et al*., 2011). In areas of Colorado, the prevalence can be as high as 30% (EFSA, 2011).

The clinical signs of CWD in affected adults are weight loss and behavioural changes that can span weeks or months (Williams, 2005). In addition, signs might include excessive salivation, behavioural alterations including a fixed stare and changes in interaction with other animals in the herd, and an altered stance (Williams, 2005). These signs are indistinguishable from cervids experimentally infected with bovine spongiform encephalopathy (BSE). Given this, if CWD was to be introduced into countries with BSE such as GB, for example, infected deer populations would need to be tested to differentiate if they were infected with CWD or BSE to minimise the risk of BSE entering the human food-chain via affected venison.

The duration of clinical disease is highly variable and death can occur within 4 weeks but some infected animals may survive as long as a year (Williams, 2005). The incubation period is a minimum of approximately 16 months and is more likely to be between 2 and 4 years (Williams, 2005). In affected elk, the incubation period is between 1.5 and 3 years after which they become clinically affected and may succumb less than 12 months after initial clinical signs appear (Miller *et al.*, 1998). During the pre-clinical period, the animal is infectious (Almberg *et al*., 2011).

The CWD agent or Prion Protein (P_rP^{CWD}) in affected animals is distributed firstly in the gut associated lymphoid tissues, digestive tract (e.g. tonsils, Peyer's patches, mesenteric lymph nodes) and then in the brain and spinal cord as the disease progresses (Sigurdson, 2008). Prions of CWD have also been found in muscle tissue (Angers *et al.*, 2006) (see Figure 2). The distribution and levels of PrP^{CWD} in tissues differ between species (e.g. elk versus deer).

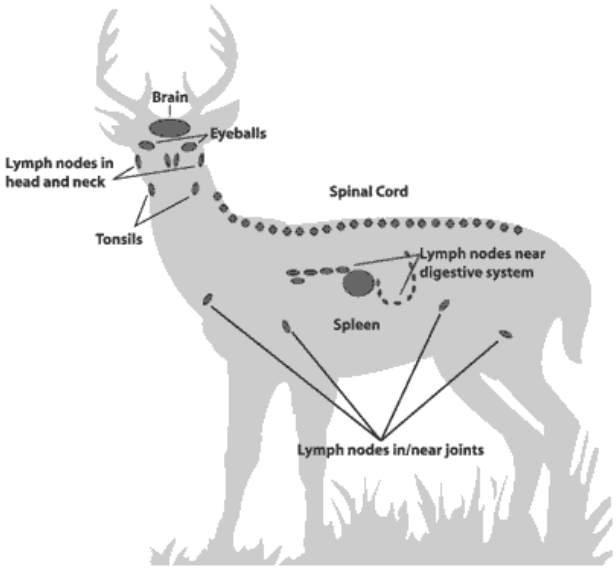


Figure 2: Diagram displaying the main organs affected by CWD in infected cervids (http://www.dnr.state.mn.us/mammals/deer/cwd/index.html)

Given its propensity to colonise the digestive tract, evidence suggests the prion is excreted in faeces (Safar *et al*., 2008), urine and saliva potentially leading to direct and indirect transmission between cervid species. Indeed, the disease is transmitted horizontally with high efficiency and circumstantial evidence suggests that environmental contamination with CWD prions contributes to the maintenance of CWD in affected areas (Safar *et al*., 2008). The rate of transmission of CWD has been reported to be as high as 30% and can approach 100% among captive animals in endemic areas (Safar *et al*., 2008). The efficiency of CWD transmission is unparalleled among TSE diseases (EFSA, 2011). Trifilo *et al*., (2007), using a murine tg mouse model, established that CWD can be transmitted via the oral route. Indeed, the distribution of PrPres in the orally infected mice (e.g. in the spleen and lymph nodes) mimicked what has been reported in deer developing CWD via natural infection (Trifilo *et al.,* 2007). Modelling studies also support the theory that transmission of CWD in deer herds is maintained by contact with a prion contaminated environment (Almberg *et al*., 2011). Scavenging of CWD-infected carcasses provides another route of releasing the prion into the environment and exposure of non-cervid species (Sigurdson, 2008). This indirect transmission route is problematic as it not only increases the basic reproductive number but also because there are very few effective mitigation strategies for reducing the risk from indirect transmission. This is due to the fact that the agent is extremely resistant in the environment and able to bind to soil particles making eradication and control of CWD a major obstacle in both farmed and free-ranging cervid populations.

Sheep and cattle may be exposed to CWD via common grazing areas with affected deer but so far, appear to be poorly susceptible to mule deer CWD (Sigurdson, 2008). In contrast, cattle are highly susceptible to white-tailed deer CWD and mule deer CWD in experimental conditions but no natural CWD infections in cattle have been reported (Sigurdson, 2008; Hamir *et al*., 2006). It is not known how susceptible humans are to CWD but given that the prion can be present in muscle, it is likely that humans have been exposed to the agent via consumption of venison (Sigurdson, 2008). Initial experimental research, however, suggests that human susceptibility to CWD is low and there may be a robust species barrier for CWD transmission to humans (Sigurdson, 2008). It is apparent, though, that CWD is affecting wild and farmed cervid populations in endemic areas with some deer populations decreasing as a result.

Thus far, CWD is restricted to North America with the exception of imported infected animals into South Korea from Canada. Surveys of wild and farmed cervid populations in the European Union between 2006 and 2010 did not identify any TSEs (EFSA, 2011). As part of this survey, 601 farmed and 598 wild red deer (*Cervus elaphus elaphus*) were tested (EFSA, 2010). These included clinical/sick animals, fallen stock, healthy shot/slaughtered animals and road killed animals. Based on the survey results, it can be concluded that the prevalence of CWD in the EU is less than 0.5%. It is important, therefore, to ensure that the disease is not introduced into Europe and establish within the

EU wild and farmed cervid population as the probability of being able to eradicate the disease would be very small.

Risk Question

This risk assessment considers the risk posed to the Great Britain (GB) deer population if chronic wasting disease (CWD) was imported from North America (i.e. Canada and the United States). The specific risk question addressed is:

What is the risk of CWD being introduced into Great Britain (GB) from North America and causing infection in deer?

To answer the above question, the risk assessment follows the OIE framework of release (or entry), exposure and consequence assessment. Specifically, it is divided into the three key areas:

1. What is the probability of introducing CWD into GB from North America?
2. What is the probability of a deer species in GB being exposed to the CWD prion?
3. What is the probability of a GB deer species becoming infected with CWD upon exposure to the prion?

Risk Assessment

Terminology related to the assessed level of risk

For the purpose of the risk assessment, the following terminology will apply (OIE, 2004):

Negligible	So rare that it does not merit to be considered
Very low	Very rare but cannot be excluded
Low	Rare but does occur
Medium	Occurs regularly
High	Occurs often
Very high	Event occurs almost certainly

Entry assessment

The routes by which CWD may be introduced into GB from North America include:

- Importation of live deer
- Importation of deer urine lures
- Importation of meat and other products derived from cervid species (e.g. trophy items including antlers, semen)
- Importation of animal feed
- Hunters and other tourists and British servicemen travelling from affected areas to GB with contaminated equipment (e.g. boots, clothing, knives)

Currently, according to the European Union Trade Control and Expert System (TRACES) database, GB does not import live cervids, 'other' animal meat products or raw hides and skins. This was the same conclusion drawn by EFSA (2004) who stated GB does not import cervids or products from North America. Therefore, the three routes which this assessment focuses on are:

1) importation of animal feed
2) importation of deer urine lures
3) importation of CWD prion on contaminated equipment and clothing/footwear of hunters or other tourists and British servicemen

Importation of animal feed

Animal feed encompasses all feed fed to farmed livestock, horses, pets, farmed fish, zoo and circus animals and also animals living freely in the wild. Currently, legislation for animal feed relating to production, and labelling and composition is harmonised at the EU level and, in GB, is the responsibility of the Food Standards Agency (FSA). In addition, Defra is responsible for ABP Regulations which includes pet food manufacturing regulation.

Pet food containing material of animal origin, according to EU Regulation (EC) No. 142/2011 on Animal By-Products, must be derived from animals inspected and passed as fit for human consumption prior to slaughter (Category 3 material). Further, the products are subject to strict microbiological criteria for *Enterobacteriaceae* and *Salmonella*. Under the EU Regulation, imported pet food produced using Category 3 processed animal proteins (PAP) must adhere to the same standards as that produced within the EU. More specifically, the imported pet food must satisfy the following criteria:

- The PAP must have been produced in accordance with the same requirements as PAP for placing on the market in the EU
- The PAP must have been sampled and tested to satisfy certain bacteriological criteria in accordance with the Regulations before release onto the EU market
- The product must enter the EU under correct Health Certification
- The Health Certification signed by the veterinarian or official inspector responsible for the rendering plant in the exporting country must verify that EU standards of

sourcing of animal by-products, processing and sampling are met for each consignment.

These requirements apply to canned pet food, processed pet food other than canned pet food, dog chews, raw pet food and flavouring innards.

According to TRACES, GB imports processed pet food from Canada and the United States of America (USA). In November and December 2015, for example, GB imported 13.6112 tonnes of processed cat and dog food (including dog chews) containing products of ungulate origin from Canada and USA. There are a limited number of processed pet food products made in the USA containing (roasted) venison for the cat and dog food market that are available in GB (e.g. Taste of the Wild pet food). Venison is high in iron content and considered a good alternative meat product for pets with intolerance to certain meat proteins. The specific amount of pet food products imported into GB from North America containing deer protein is unknown and not specified in the TRACES system, but is likely to be a small percentage of the overall amount of processed pet food imported. The TRACES database indicates that in the same period (Nov – Dec 2015) 751,418 tonnes of other products for pet food were imported into the UK from USA and Canada.

In the USA, under the Food and Drug Administration's BSE Feed Regulation (21 CFR 589.2000) most material (exceptions include milk, tallow, and gelatin) from deer and elk is prohibited for use in feed for ruminant animals. With regards to feed for non-ruminant animals, under FDA law, CWD positive deer may not be used for any animal feed or feed ingredients. For elk and deer considered at high risk for CWD, the FDA recommends that these animals do not enter the animal feed system. However, this recommendation is guidance and not a requirement by law. Animals considered at high risk for CWD include: 1) animals from areas declared to be endemic for CWD and/or to be CWD eradication zones and 2) deer and elk that at some time during the 60-month period prior to slaughter were in a captive herd that contained a CWD-positive animal. Therefore, in the USA, materials from cervids other than CWD positive animals may be used in animal feed and feed ingredients for non-ruminants. The amount of animal PAP that is of deer and/or elk origin imported from the USA to GB cannot be determined, however, as it is not specified in TRACES. It may constitute a small percentage of the very low tonnage of non-fish origin processed animal proteins that were imported from US into GB.

Overall, therefore, it is considered there is a **greater than negligible** risk that (non-ruminant) animal feed and pet food containing deer and/or elk protein is imported into GB. There is uncertainty associated with this estimate given the lack of data on the amount of deer and/or elk protein possibly being imported in these products.

Movement of hunters, other tourists and British servicemen

Probability that the environment in North America is contaminated with CWD

As outlined in Figure 1, there are 21 states and provinces in the USA and Canada where CWD has been detected in farmed and wild cervids. These include: Alberta (Canada), Arkansas, Colorado, Illinois, Kansas, Maryland, Minnesota, Missouri, Nebraska, New Mexico, New York, North Dakota, Saskatchewan (Canada), South Dakota, Utah, Virginia, West Virginia, Wisconsin and Wyoming. In these areas, the environment is likely to be contaminated with CWD prions from direct excretion of the prion in various bodily fluids of infected animals, and leaching of prions into the soil from decaying carcasses of infected animals. A summary of the current studies on CWD in faeces, urine, blood and other bodily fluids or organs is summarised in Table 1.

Table 1: Summary of the studies on CWD prion excretion

Fluid/organ	Study summary	Reference
Faeces	• Faeces were the source of CWD infection in Syrian hamsters under experimental conditions. • Prions remain intact after being passed through the digestive tract and, therefore, are a viable source of infectivity in the environment. • CWD prions are excreted in the faeces of infected mule deer 7 to 11 months prior to the onset of neurological signs (i.e. during the incubation period)	Safar *et al*., (2008) Tamguney *et al.* (2009)
Saliva	• During studies of oral transmission using a murine tg mouse model, it was observed that prior to and during clinical disease, serous and mucous glands contained PrPres. • Three naïve fawns were orally inoculated with 50ml of saliva from an infected deer in 3 doses over a 3 day period. Eighteen months post inoculation, CWD prions were detected in all 3 fawns during tonsil biopsy. • Pooled saliva from five terminally CWD infected white-tailed deer was inoculated into nine *tg1536* mice. Eight of the nine mice developed disease consistent with a TSE at 342 ± 109 days post inoculation suggesting infectious prions are present in saliva of infected cervids.	Trifilo *et al*., (2007) Mathiason *et al*., (2006) Haley *et al*., (2009)
Blood	• Two naïve white-tailed deer were inoculated intraperitoneally with 250 ml of frozen citrated blood and a further fawn with an intravenous transfusion of 250 ml freshly citrated whole blood. After 18 months post inoculation, all three fawns had CWD prions in their tonsils and retropharyngeal lymph node.	Mathiason *et al*., (2006)
Urine	• Pooled urine from five terminally CWD infected white-tailed deer was inoculated into nine *tg*	Haley *et al*., (2009)

	mice. Two of the nine mice developed disease consistent with a TSE at 370 and 376 days post inoculation suggesting infectious prions are present in the urine of infected cervids but at a lower infectivity than other bodily fluids such as saliva. • Henderson et al. (2015) report urine from CWD-infected deer contained 1 intracerebral LD_{50} per 10-20 ml. But their LD_{50} is estimated from amyloid formation rate and may be difficult to relate to an oral LD_{50} for live deer. • Henderson et al. (2015) cite Gonzalez-Romero et al. 2008 which used PMCA to show that levels of prions in neat urine were approximately equivalent to a 10^{-10} to 10^{-11} dilution of brain. • The levels in urine may be so low as to be difficult to quantify by bioassay. • Assumed estimate below that the oral LD50 for a deer may be 1,000 litres of urine from a CWD-infected deer. This assumes the oral route is 100,000-fold less efficient than the ic route.	Henderson et al. (2015)
Antler velvet	• Antlers are covered by a layer of skin, velvet, which is shed after an increase in testosterone and ossification of antlers. CWD is present at low, but detectable, amounts in antler velvet from infected elk.	Angers *et al*., (2009)

It is clear from Table 1 that CWD is excreted in several different bodily fluids and, as demonstrated in experimental studies, can be a source for onward transmission to naïve animals. Infected carcasses decaying naturally in confined areas can also lead to new CWD infections in naïve deer (Sigurdson, 2008). This was proved, experimentally, by Miller *et al*., (2004) during a study of environmental transmission. Specifically, 3 naïve mule deer were stocked in an $800m^2$ paddock in which a naturally infected mule deer had died and decomposed approximately 1.8 years prior. In a second paddock, a further 3 naïve mule deer were placed where infected mule deer had resided 2.2 years earlier and contaminated the environment with their faeces (Miller *et al*., 2004). The experiment was conducted in 3 replicates. In total, 3 out of 12 and 1 out of 9 deer were infected by being exposed to an infected decomposed carcass or residual excreta, respectively.

The CWD prion has also been detected in water. Specifically, very low levels (below infectious levels) were detected in a water sample from melting winter snow-pack from an endemic area (Nichols *et al*., 2009). The data showed persistence of CWD prions in water, accumulated levels of which, it is hypothesised, may promote transmission within deer herds.

Once in the environment, TSE prions can bind to soil particles and remain infectious (Saunder *et al*., 2010). Indeed, Johnson *et al.*, (2006) demonstrated that the disease-

associated form of the prion protein can bind to all soil mineral surfaces and is preserved in a bioavailable form. Further, in a later study, Johnson *et al*., (2007) observed that prions bound to the soil mineral montmorillonite (Mte) significantly enhanced disease penetrance and reduced the incubation period compared to unbound prions. The reason why binding to Mte or other soil components enhances transmissibility is unclear but it may provide some protection for the prion in the gut against denaturation allowing more agent to be absorbed by the animal (Johnson *et al*., 2007). Further, binding to the soil particles maintains prions near the soil surface increasing the probability of animal exposure (Russo *et al*., 2009).

In addition to the enhanced infectivity, prions can remain in the soil for several years as the agents are resistant to inactivation by most chemical agents, radiation and heat (Johnson *et al*., 2006). Seidel *et al.* (2007), for example, demonstrated that scrapie agent (strain 263K) remains persistent in soil over a period of at least 29 months and remains highly infectious to Syrian hamsters in oral inoculation experiments. In Iceland during an epidemiological investigation of scrapie, a TSE of sheep and goats, Georgsson *et al*., (2006) reported that the scrapie agent survived on a farm for at least 16 years. However, Russo *et al*., (2009) demonstrated experimentally that reactive soil components such as manganese oxides may contribute to the inactivation process of TSE prions in soil. The authors did not study CWD prion specifically but the study highlights the complexity of the effect the inorganic and organic constituents in soil may have on prion survival and infectivity.

In summary, in endemic areas, there is a **medium** probability that the soil and surrounding environment is contaminated with CWD prions and in a bioavailable form. In rural areas where CWD has not been reported or only very recently reported and deer are present, there is a **greater than negligible** risk the soil is contaminated with CWD prion.

Movement of deer hunters, other outdoor tourists and British servicemen between North America and GB

The probability a person comes into contact with CWD prions varies depending upon their place of residence and/or their involvement with outdoor pursuits (e.g. hunting). In this assessment, focus is given to the following groups of people:

- Residents in CWD affected areas travelling to GB (particularly the countryside) and British tourists travelling to CWD affected areas
- North American hunters travelling to GB to hunt/stalk deer and British hunters travelling to North America to hunt deer
- British servicemen training in and/or near CWD affected areas

All other people (e.g. city tourists and residents) are considered to pose a lower risk of being exposed to CWD in North America and, therefore, arriving in GB with contaminated clothing, footwear and/or equipment.

There are limited data on the number of North American tourists, stratified by location of residence, arriving in the UK. Therefore, it is not possible to ascertain of the 3.89 million

visitors from the United States (US) to the United Kingdom (UK) in 2006, for example, how many were from CWD affected areas. Likewise, there is no breakdown of where in the USA the 4.5 million UK visitors travelled in 2008. This is a significant data gap in the assessment.

Hunting in the US is a popular sport with 4% of the population (10.1 million) involved in deer hunting in 2006 (USFWS, 2011). Of these people, 4.7 million only hunted deer whilst the remainder hunted deer and other species (e.g. small game, bears). Further, 58% of deer hunters were involved in wildlife watching activities and other outdoor pursuits (e.g. hiking, fishing); this is compared to 31% of the general public (USFWS, 2011; TAMS, 2007). Individuals that hunted whilst on a trip were likely to come from northern and western states (e.g. Alaska, Wyoming, North Dakota, South Dakota, Idaho and Montana) compared to highly urbanised states. In Canada, those individuals participating in hunting activities are most likely to do so within Canada with the majority taking a trip within their own province or region (TAMS, 2006). Canadian hunters were also more likely than other Canadian pleasure travellers to participate in wilderness activities and hiking (TAMS, 2006). There are no data collated on the number of hunters from North America travelling to the UK to stalk/hunt deer and, vice versa, there are no data on the number of UK residents hunting in North America. However, in order to hunt in GB with your own rifle, a visitor firearms permit has to be obtained from the police force in one of the devolved countries. In 2011, 123 licences were granted by the Scottish Police Force for non-EU residents (BASC, pers. Comm., 2012). This includes not only individuals from North America but also Norway and other non-EU countries (BASC, pers. Comm., 2012). The number of hunters arriving without their own rifle and participating in an organised hunting package/holiday is unknown. However, it is likely the total number of hunters from North America is in the low hundreds; the actual number, however, is highlighted as a significant data gap.

As well as tourists, British servicemen frequently move between North America and GB. In particular, British Army servicemen use the Canadian Forces base at Suffield, Alberta to take part in extensive training exercises. Suffield is located in the southwest of Alberta and comprises of 2,690km^2 of prairie landscape. The eastern boundary is designated as a wildlife management area and is south of a wildlife management area in which CWD was reported as recently as 2011 (www.srd.albert.ca). Consequently, the servicemen have the potential to be in close contact with areas where CWD is present.

In summary, given the volume of tourists, hunters and servicemen moving between GB and North America, the probability of at least one person travelling to/from a CWD affected area and, in doing so, contaminating their clothing, footwear and/or equipment prior to arriving in GB is **greater than negligible**. For deer hunters, specifically, the risk is likely to be greater given the increased contact with deer and their environment. However, there is significant uncertainty associated with these estimates.

Probable amount of CWD prions on contaminated boots and equipment

Given that a hunter or tourist walks in areas which are contaminated with CWD, it is possible that they will collect soil on their boots and other equipment. This likelihood will increase if the hunter has shot and handled a CWD infected deer resulting in contamination of the hunting equipment (e.g. knives) and their clothing and they subsequently arrive in GB with this equipment, footwear and clothing. Further, the soles of hiking boots tend to retain more soil than those of normal shoes. Wilkinson (2010), for example, removed 0.1 g of soil from hiking boots after returning to GB from a 2-month research visit to Canada. The amount of CWD prion in this amount of soil will depend upon the density of CWD infected animals excreting prions into the environment and the type of soil; CWD prion binds to clay soil, for example. Animal mortality sites could also be hotspots of CWD prion given the highly infectious nervous system matter entering into the environment and soil (Saunders *et al*., 2010).

Importation of deer urine lures

It is well established that urine from CWD-infected deer contains CWD infectivity. The urine collected for deer lures has no processing and is immediately refrigerated and bottled (Anon 2015b). There is no inactivation of the CWD agent in the urine and thus all infectivity present at the point of bottling in the USA will still be present at the point of use in the UK. However, under EU rules, (EC Regulation 1069/2009), urine from farmed deer should fall under the definition of “manure” and therefore the import of such a product, if unprocessed, is not allowed, according to Regulation (EC) 142/2011. Nevertheless, the processing required for bottling prior to retail is unlikely to affect the CWD prion and if it did fulfil the requirements in 142/2011, the active ingredient in the urine would no longer be effective. It is therefore presumed that the urine is considered “unprocessed” under EU law. The import and transit of urine from wild deer is not covered by 1069/2009.

A survey conducted by the British Deer Society (BDS) in July 2015 suggested small number of hunters in GB were aware of and used deer urine lures (http://www.bds.org.uk/index.php/news-events/135-deer-urine-lure-survey-july-2015).

For the purpose of the qualitative risk assessment developed here it is necessary to estimate the probability that a 30-ml bottle of lure contains urine from an infected deer. This requires an estimate of the proportion of deer herds in the USA which are infected with CWD together with the within herd prevalence.

The distribution map of CWD in US shows it is present mainly in central states (Figure 1). However, Virginia in the east of the country has recorded seven recent cases of CWD (Anon 2015a). Some US manufacturers claim to take steps to prevent urine being taken from infected animals eg by sourcing from farms where the deer are randomly tested for CWD (Anon 2015a). However, if disease is already present and testing is not carried out regularly, captive populations are not necessarily disease free (Strausser 2014). Urine-based deer lures have been known to be collected from domestic white-tailed deer herds and therefore there is a recognised risk. This is reflected by 6 US States which have

banned the use of natural deer urine for lures, as the deer urine may be sourced from CWD-endemic areas in the USA as well as from areas free of CWD. For example, the US State of Virginia is banning the use of urine-based deer lures on July 2015 and Vermont from 2016 due to the risk of spread of CWD. Alaska banned their use in 2012 (Anon 2015a). Pennsylvania Game Commission has banned urine-based deer lures and acknowledged that there is no way to detect their use (Strausser 2014). On the basis of unpublished data (J. Manson, Pers. Comm.) it appears that up to 50% of deer herds can be infected with 80-90% of animals infected within some herds. It is therefore assumed that probability that a 30-ml bottle of deer urine lure imported from the USA is sources from an infected deer is **medium**.

Exposure assessment

Importation of animal feed

Once in GB, the use of animal feed is subject to the TSE Feed Ban and ABP Regulations. The BSE-related feed ban prohibits the feeding of PAP and gelatine from ruminants to ruminants (including farmed deer) or non-ruminant farmed animals. Further, ruminants must not be fed any animal protein or feedstuffs which contain animal protein except for milk, milk-based products and colostrum, eggs and egg products, gelatine from non-ruminants and hydrolysed proteins derived from non-ruminants or from ruminant hides and skins. Therefore, in accordance with the current ban, farmed deer should not be directly exposed to (i.e. feed) imported animal feed containing any PAP. Therefore, assuming this ban is adhered to correctly the risk of farmed deer being exposed to animal feed containing deer protein from North America is considered **negligible** but with associated uncertainty. However, given that non-ruminant feed produced in the USA may contain deer and elk PAP, it is theoretically possible that wild deer may be exposed to deer protein in legally imported non-ruminant feed. For this to occur, wild deer would need to access non-ruminant feed (e.g. pig, fish and chicken feed) on farms near their habitat. Alternatively, wild deer may be exposed to CWD prion in the faeces of pets that have consumed and digested imported, contaminated pet feed. The frequency in which these routes may occur is unknown and is considered to be a **greater than negligible** risk with associated uncertainty.

Movement of hunters, other tourists and British servicemen

The pathways by which naïve deer can be exposed to CWD contaminated soil and prions on equipment and clothing from people arriving to GB from North America are variable and highly uncertain. In principle, in order to expose a deer to CWD prions, the traveller (hunter, tourist or serviceman) would need to transfer the CWD prion from their clothing and/or equipment to the environment in which deer habit. The latter will depend upon the behaviours of returning GB residents or tourists and the probability of entering into and walking around deer territory. In GB, there are two main deer populations (wild and farmed

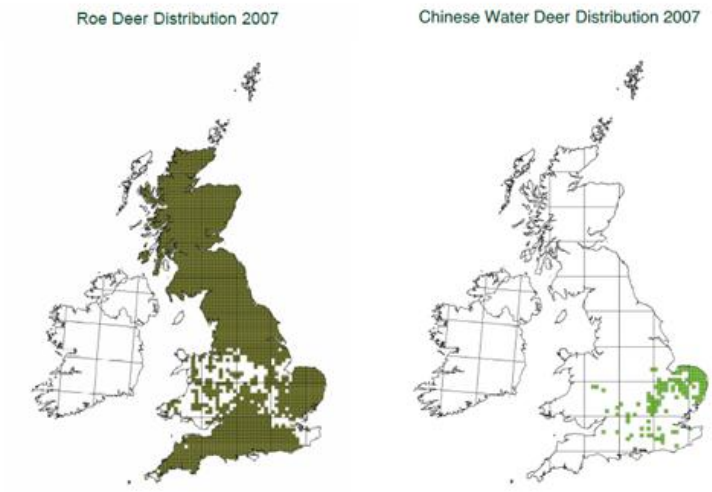
or park deer) each of which will have differing risks of exposure given the type and frequency of contact with people. Each population type is considered in turn.

Wild deer

There are 6 species of wild deer residing in GB including: Red deer (*Cervus elaphus*), Roe deer (*Capreolus capreolus*), fallow deer (*Dama dama*), muntjac (*Muntiacus reevesi*), sika (*Cervus nippon*), and Chinese Water deer (*Hydropotes inermis*). The British Deer Society implemented a survey in 2007 to ascertain the distribution of these deer species across the UK. The survey provides the presence of deer on a standard template of 10km grid squares (www.bds.org.uk). A further survey was conducted in 2011 of which the results will be published later in 2012. The deer distribution as ascertained from the 2007 survey is summarised in Figure 3.

It can be seen from Figure 3 (below) that deer are widely distributed across the UK with Roe deer being the most widespread. Chinese Water deer are the smallest deer population with approximately 700 deer.

Deer hunters, particularly, are most likely to be in direct contact with wild deer and their habitat compared to other tourists and returning GB residents. During the stalking and/or hunting of deer, there is opportunity for CWD prion on the hunter's boots, clothing and/or equipment to be transferred to the environment. The amount transferred will depend upon the measures taken to remove soil etc from the equipment prior to stalking. Assuming that CWD prion is transferred to the environment, there is an uncertain probability that a deer will come into contact with the CWD prion.



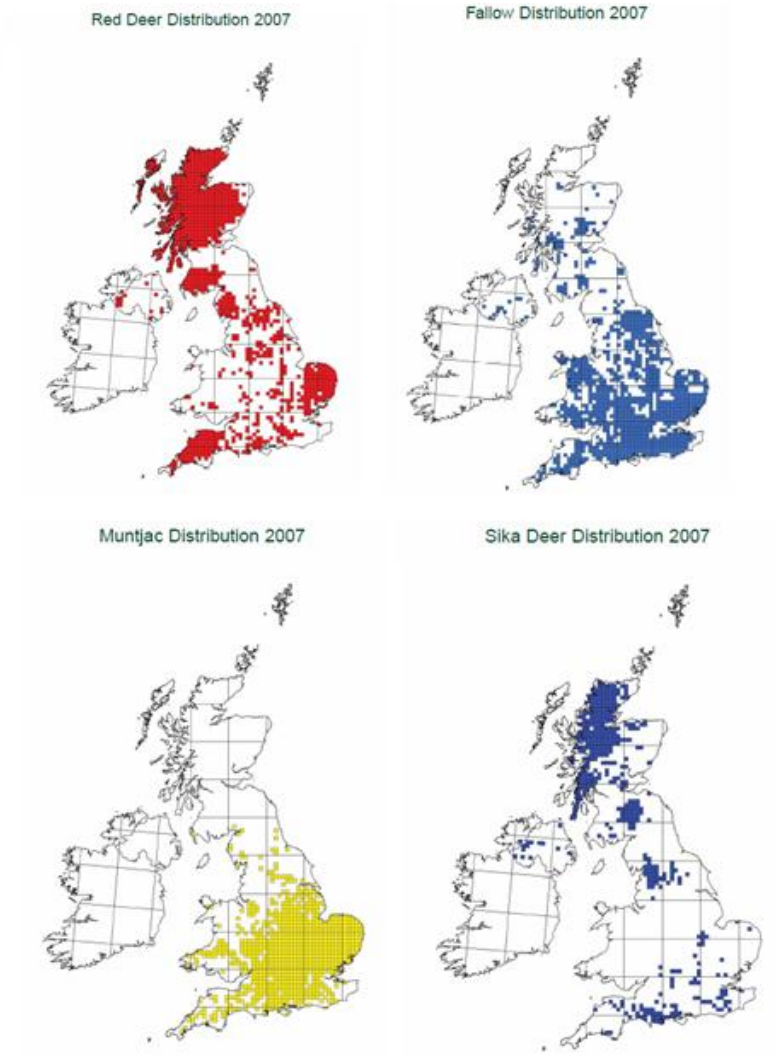


Figure 3: Distribution of the six deer species in the UK in 2007 (British Deer Society Deer Distribution Survey, http://www.bds.org.uk)

Farmed and park deer

Deer farming is a relatively recent enterprise. There are two systems currently used for managing enclosed deer: park and farm deer systems. In the park system, deer are raised in a park type setting and allowed to roam freely and may be provided with some supplemental feed. Farmed deer, in contrast, following conventional agricultural practices and may be housed in the winter and nutritional supplements are provided where necessary. In this farming system, there are several categories including calf rearers, calf finishers, breeder finishers and producer/processors (www.bdfpa.org). In 2011, according to the June Agricultural census, there were approximately 21,000 farmed deer on commercial agricultural holdings in England. The Economic Report on Scottish Agriculture (2011) cited that within Scotland, Wales, England and Northern Ireland there were 30,910 farmed deer. It is less likely that tourists, deer hunters and British servicemen will come into contact with conventionally farmed deer compared to park deer. The total park deer population in GB is unknown. However, in 2005, based on annual population control culling of about 8,000 animals, it was estimated that there were approximately 40,000 park deer. These deer are distributed across several parks (some of which are famous tourist

sites) where wild and/or exotic species of deer can roam and be viewed. These are outlined in Table 2.

Table 2: Summary of parks in Great Britain where wild and/or exotic deer roam (The British Deer Society, www.bds.org.uk)

Country	Park	Species
England	Ashton Court, Bristol	Red, Fallow
	Bolderwood Deer Sanctuary, Minstead, Hampshire	Fallow,
	Bradgate Park, Charnwood Forest, Leicestershire	Fallow, Red
	Bushy Park, Hampton Hill, London	Fallow, Red
	Chatsworth Park, Chatsworth, Derbyshire	Fallow, Red
	Dunham Massey, Altrincham, Cheshire	
	Dyrham Park, Chippenham, Wiltshire	Fallow
	Grimsthorpe Castle Park and Gardens, Bourne, Lincolnshire	
	Helmington Hall, Stowmarket, Suffolk	Red
	Holkham Estate, Wells-next-the-Sea, Norfolk	Fallow
	Knole Park, Sevenoaks, Kent	Fallow, Sika
	Lodge Park & Sherborne Estate,	
	Lyme Park, Disley, Cheshire	Red, Fallow
	New Forest Wildlife Park, Longdown, Hampshire	European Bison, Red deer
	Petworth Park, Petworth, Sussex	Fallow
	Prinknash Deer and Bird Park, Cranham, Gloucester	
	Raby Castle, Staindrop, County Durham	Red, Fallow

	Richmond Park, Richmond, London	Red, Fallow
	Snettisham Park Farm, King's Lynn, Norfolk	Red
	South West Deer Rescue and Study Centre, Wayford	Red, White Red, Fallow, Axis, Roe, Japanese Sika
	Tatton Park, Knutsford, Cheshire	
	Wentworth Castle, Stainborough	Red, Fallow
	Wildwood Trust, Hern Bay, Kent	Roe, Fallow, Red
	Woburn Abbey, Woburn Park, Bedfordshire	Sika, Axis, Chital, Barasingha, Chinese Water, Rusa, Pere David
	Wollaton Park, Woolaton, Nottinghamshire	Red, Fallow
Wales	Abergavenny Priory Deer Park, Abergavenny	
	Dinefwr House, Kinefwr Park, Llandeilo, Carmarthenshire	Fallow
	Margam Country Park, Port Talbot	Red, Pere David, Chital, Hog, Barasingha, Roe, Muntjac, Chinese Water
Scotland	Beecraigs Country Park, Linlithgow, West Lothian	Red
	Glengoulandie Deer Park, Aberfeldy, Perthshire	Red
	Highland Wildlife Park, Kingussie, Inverness-shire	Red, reindeer
	Jedforest Deer and Farm Park, Jedburgh	
	The Scottish Deer Centre, Cupar, Fife	Nine species of deer

It is evident from Table 2 that there are several locations in GB where tourists and returning residents may come into contact with park deer and, in doing so, potentially expose the deer to CWD on their contaminated clothing and/or footwear. Further, given the volume of tourists and other travellers moving between North America and GB, there are potentially multiple opportunities for CWD prions to be transferred from clothing, boots and/or equipment to the environment. It has been observed that multiple exposures to low levels of CWD prions in the environment and increased infectivity of CWD when prions are bound to the soil are influential factors in transmission (Anger *et al*., 2009). Given the nature of their management, there is a restricted area (or environment) in which park deer inhabit enabling them to have a potentially higher probability of coming into contact with any CWD transferred to the environment by a tourist or returning GB resident compared to wild deer in a free-ranging environment. Therefore, it is considered that farmed and park deer may have a higher probability of exposure to CWD transferred to the environment than wild deer given the restricted habitat range and higher frequency of contact with tourists and returning GB residents.

Exposure of UK deer to infected urine

For the purpose of the qualitative risk assessment developed here it is necessary to estimate the probability that some of the contents (i.e. 10-ml) of each 30-ml bottle of deer urine lure is ingested by UK deer.

The degree of exposure depends on whether deer are likely to lick urine in the woods, and where the urine is placed by the hunter. Some hunters claim it is unlikely deer will lick the urine (Anon 2015a). The main risk reduction factor is the fact that deer roam over a very large area and female deer (those not attracted) would be unlikely to come in contact with it. The risk of exposure to female deer therefore may be relatively low. This raises the question of deer bucks which are attracted to the lure and whether they lick it. The aqueous solvent component of the urine will evaporate or soak into the ground. However, it is well known that TSE infectivity does not leach into the ground but attaches to soil particles, which could be eaten by grazing deer. Although the infectivity may be less bioavailable compared to a deer actually imbibing the contents of a 30-ml bottle of deer urine lure the exposure of single bucks attracted to the lure **cannot be assumed to be negligible.** Furthermore it cannot be assumed that the lured deer are shot and the deer lure may continue to attract deer for some time after the hunter/stalker has departed. It is not known how long the lure would work for. CWD infectivity will persist in the environment for long periods of time, and bind to the soil surface. Therefore, there is the possibility that the CWD infectivity in deer lure urine may persist for months, such that deer could be exposed at a later date. Overall therefore, given the fact that the primary use of a deer lure is to attract bucks, it is assumed here that the probability that some of the contents (i.e. 10-ml) of each 30-ml bottle of deer urine lure is ingested by UK deer is medium.

Given that a deer within GB is exposed to CWD bio-available prions in the environment, the probability of becoming infected is dependent upon the infectious dose and the susceptibility of the animal to the prion. The majority of research into CWD has been

conducted in North America where it has been shown that the following species are naturally infected with CWD (Hamir *et al*., 2008):

- Mule deer (*Odocoileus hemionus hemionus*)
- Black-tailed deer (*Odocoileus hemionus columbianus*)
- White-tailed deer (*Odocoileus virginianus*)
- Rocky Mountain elk (*Cervus elphus nelsoni*)
- Shira's moose (*Alces alces shirasi*)

None of these species are present in GB. However, EFSA (2010) considered that red deer (*Cervus elaphus*), a species present in GB (see Figure 3), is likely to be susceptible to CWD and was a species specifically targeted in the EU survey for CWD. This stems from the fact that red deer have a close genetic relationship to Rocky Mountain elk. This hypothesis has been supported by recent experimental studies that have demonstrated red deer become infected with CWD after oral inoculation of brain tissue from infected Rocky Mountain elk (Balachandran *et al*., 2010). Specifically, two of the four 2-month old red deer, showed clinical signs by 585 days p.i. and all deer had CWD prion in the brain, spinal cord and other organs at necropsy (Balachandran *et al*., 2010). Further, Martin *et al*., (2009) demonstrated in a similar study of four European red deer, that red deer can become infected upon inoculation of 5g of infected brain homogenate from four CWD elk and hence the species is susceptible to CWD.

Hamir *et al*., (2008) undertook a study to ascertain if fallow deer (*Dama dama*), another British deer species, could be experimentally infected with CWD brain suspension from infected elk or white-tailed deer. The authors concluded that it is possible to transmit CWD to fallow deer via the intracerebral route but the pathological features of CWD in the deer differs from those observed in white-tailed deer or elk (Hamir *et al*., 2008). It was further concluded that it might not be possible to transmit CWD via a more natural route or, alternatively, a higher dose of inoculum is required leading to a longer incubation period (Hamir *et al*., 2008).

Initial studies into the PRioN Protein (PRNP) gene variability in European red deer and roe deer suggest that these species have a PRNP genetic background that is compatible with TSE susceptibility, including CWD (EFSA, 2011). It is important to note, however, that no experimental studies on roe deer have been conducted verifying this hypothesis.

There are no data on the susceptibility of the other free-ranging deer species present in Britain (muntjac (*Muntiacus reevesi*), sika (*Cervus nippon*), Chinese Water deer (*Hydropotes inermis*)) to CWD. Further experimental studies would be required to investigate the susceptibility of these species to CWD. Therefore, on the basis of current scientific understanding, it is likely that given exposure to an infectious dose to CWD, deer in GB could become infected with CWD. Whether the amount of CWD prion that could be transferred from clothing, boots and/or other equipment into the deer's environment is enough to induce infection given that the infectious dose is extremely small (Saunders *et al*., 2010) is uncertain. However, given that the amount of soil ingested is likely to be very small, the probability of ingesting an infectious dose via this route is considered **negligible**

to very low. The probability of ingesting an infectious dose via consumption of non-ruminant feed is likely to be higher and may be **very low**, with associated uncertainty.

The CWD agent is relatively dilute in deer urine compared to brain and spinal cord material with 1 ic LD_{50} per 10 ml. The LD_{50} determined by Henderson et al. (2015) is an intracerebral ID_{50} in cervidized transgenic mice. The oral ID_{50} in deer would be in a much larger volume of urine, because the oral route may be 100,000-fold less efficient than the intracerebral route in terms of TSE transmission (Gale et al 1998). Thus, in terms of oral LD_{50}, there may be one in a 1,000,000 ml i.e. 1 m^3 or 1,000 litre volume of deer urine. Therefore a deer would have to ingest 1,000 litres of urine to have a 50% chance of being infected through the oral route. Of course the CWD agent would be concentrated as the urine evaporated from the tree trunk.

The main sources of uncertainty are:-

1. The amount of urine ingested by the deer. A bottle of Tink's "69 Doe-in-Rut Buck" is about 30 ml and boasts 100% natural doe oestrous urine (Anon 2105d). A bottle from an infected doe would thus contain about 3 i.c. LD_{50} units. The bottles come with an easy to use squirt top, so it seems relatively small amounts are used. There are also gel forms which do not freeze as fast, last longer in the rain, and do not dry out so quickly.

2. The magnitude of oral/i.c. barrier

Assuming a deer drank 10 ml of urine from each 30 ml bottle, then that deer would ingest 10^{-5} $LD_{5.}$ It is generally assumed that there is no threshold dose for TSEs (Gale 2006) and the risk of CWD infection in GB deer per 30 ml bottle imported is therefore 0.69 x 10^{-5} (Gale 1998). This is **very low**.

Table 1: Risk of infection of deer in GB per 30 ml bottle of deer urine lure imported from the USA

Step in pathway	Risk	Uncertainty
Entry (probability a 30-ml bottle contains urine from an infected deer)	Medium	Low
Exposure (probability some of 30-ml bottle is ingested by UK deer)	Medium	High
Consequence (probability that deer is infected given exposure)	Very low	Medium
Overall risk	Very low	High

Overall the risk of a deer in GB being infected per 30 ml bottle of urine imported from the USA is **very low**, albeit with **high** uncertainty.

The next question is how much deer urine is imported into GB from the USA per year. If there are n 30-ml bottles, then the risk is calculated as:-

$$p_{n\_bottle} = 1 - (1 - p_{one\_bottle})^n$$

Hundreds of gallons of urine are sold every year just by one company in the USA (Anon 2015b). It is difficult, however, to estimate the volume of deer urine imported into the UK per year from the USA. That some respondents to the BDS survey reported they used upwards of 1litre per year, is consistent with importation of a high number of 30-ml bottles. Assuming that the number, n, of 30-ml bottles imported annually is high, then the risk of CWD infection in at least one deer in the UK per year will be **medium** (given the probability, of infection of a UK deer from one 30-ml bottle is very low (see Table 1)).

Overall it is concluded that the annual risk of at least one infection of deer in the UK with CWD from deer urine lures imported from the USA is **medium**. This assumes a high number of 30 ml bottles imported per year from all areas of the USA.

Control and risk management options

In order to reduce the potential amount of CWD prion entering GB on boots and clothing, it is important to meticulously clean off all adherent material prior to departing from North America. As CWD is a highly resistant agent, where possible, equipment should be soaked in a solution of bleach that has 20,000 parts per million of active chlorine for one hour. However, it is acknowledged it is impractical to soak hunting boots, clothing or firearms, for example, in strong concentrations of bleach.

For deer urine lures, the import of unprocessed natural deer urine is not allowed under the EU Animal By-Product legislation. However the import of such commodities including internet sales would still require licenses, but it would be a voluntary action on behalf of the importer to ensure the urine is sourced from safe herds. One option is to ensure the sourcing is from herds in States which are currently registered under the National CWD Herd Certification Programme which has recently been introduced in the USA. Otherwise the use of synthetic of European-produced lures should be promoted by the industry and stakeholder groups.

Conclusions

There is significant uncertainty associated with estimating the risk of CWD entering the UK via movement of people (tourists, hunters and British servicemen) and importation of animal feed. This partly stems from the lack of data on these two importation routes. Given this uncertainty, the probability of importing CWD into GB from North America and causing infection in British deer is likely to be **negligible to very low** via movement of deer hunters, other tourists and British servicemen and **very low** via imported (non-ruminant) animal feed. However the risk of natural deer urine lures from the USA containing CWD

PrP is considered **high** (reflected by some US States banning the use) and the probability of such a commodity, if used in significant volumes, leading to CWD infection in GB populations is considered to be **medium** (lack of susceptibility in certain species and limited use by hunters and stalkers in GB) but with a high level of uncertainty.

The consequences of CWD, however, are severe with the minimal possibility of eradicating the disease from a wild cervid population and populations of wild and farmed deer would be under threat.

Current research indicates that of the six free-ranging deer species in the UK, red deer are susceptible to CWD. This deer species is concentrated in distinct areas of the country (e.g. North of Scotland) and one of the key species which hunters, in particular, stalk. It is important, therefore, that the risk of this species being exposed to CWD is minimised by taking appropriate precautionary measures.

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