Disease Risk Analysis for the Conservation Translocation of the Eurasian Beaver (Castor fiber) to England

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Disease Risk Analysis for the Conservation Translocation of the Eurasian Beaver (Castor fiber) to England

Donald, H., Common, S. and Sainsbury, A.W.

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Project details

This report was commissioned to inform Natural England's advice to government on the reintroduction of beavers in England.

Beaver reintroduction is a topic of increasing interest in England. Although species reintroductions are a key conservation tool used to help restore species populations and/or ecosystem function, species translocations can facilitate the movement of parasites and risk animals encountering parasites that they normally would not be exposed to. Risks from disease associated with wildlife translocations arise because individual translocated specimens are a 'biological package', consisting of the host and all the associated viruses, bacteria, fungi and other parasites that the animal or plant may naturally harbour. Reintroduced beavers may, therefore, act as a mechanism for the introduction of new or previously eradicated parasites or may establish new transmission routes for the infection of humans, domesticated livestock and existing wildlife.

Disease risk analysis is a qualitative risk assessment method undertaken in the planning stage of a conservation intervention such as a reintroduction. During a disease risk analysis, the risk that infectious and non-infectious hazards will precipitate during or following an intervention is analysed in the absence of mitigating measures. Subsequent mitigating measures are proposed to reduce this risk. Preventing the introduction of alien parasites to native populations is crucial because parasite invasions have the potential to cause catastrophic mortality outbreaks in potentially immunologically naïve populations.

Disease risk analysis and the evaluation of mitigation measures is, therefore, a key step in understanding and controlling any disease risks for humans, livestock and wildlife that may arise from a reintroduction or translocation of wildlife.

This report provides an update to existing disease risk analyses for beavers in Britain and investigates the translocation pathways specifically relevant to reintroductions in England.

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Natural England Project manager

Dr Claire Howe, Horizon House, Deanery Road, Bristol, BS1 5AH

Contractor

Dr Helen Donald, Dr Sophie Common and Dr Tony Sainsbury. Institute of Zoology, Zoological Society of London, Regent's Park, London, NW1 4RY

Authors

Dr Helen Donald BA MBA BVetMed MSc Wild Animal Health MRCVS
Dr Sophie Common BVetMed MSc Wild Animal Health MRCVS
Dr Tony Sainsbury, BVetMed MRCVS CertZooMed DVetMed CertAP DipECZM (Wildlife Population Health) FHEA

Institute of Zoology,
Zoological Society of London
Regent's Park
London NW1 4RY

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Further information

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Executive summary

In a disease risk analysis on the conservation translocation of free-living beavers from Norway, or Great Britain, to England, 78 hazards (73 infectious and five non-infectious) were evaluated and twenty-one received detailed analysis. Of the latter twenty-one, 14 were of high or medium risk of precipitating disease in beavers or sympatric mammals, including people without mitigation: hantaviruses (PUUV); gram-negative enteric bacteria; Streptococcus castoreus; Stichorchis subtriquetrus; Trichinella species; Toxoplasma gondii; Emmonsia crescens; SARS-CoV-2; road traffic collisions; persecution; captivity during translocation; Yersinia enterocolitica and Y. pseudotuberculosis; Leptospira species and Echinococcus multilocularis.

Seven of these 14 are stressor-associated and very careful attention to translocation protocols will be required to reduce the risk from these hazards. If Natural England concludes that the benefits of translocation outweigh the costs, we recommend that a disease risk management and post-release health surveillance protocol, which includes attention to stressor-related hazards, is drawn up. Stressor-associated parasite hazards can be commensal and an important component of biodiversity, and efforts should be made to conserve these parasites following translocation.

The spread of Echinococcus multilocularis, a cestode parasite which causes severe disease in people, through Scandinavia over the last ten years has increased the risk from disease since an analysis was last carried out on the importation of this parasite to the UK in 2012. Given that Trichinella species are also a zoonotic risk for people from beaver translocation from the continent, our analysis shows that translocations from Great Britain to England are less of a risk than translocations from Norway to England.

Evidence shows that ‘source hazards’ constitute the greatest risk of epidemic disease following translocation and, given that free-living beavers in Great Britain are of uncertain origin, if beavers in Great Britain are used for translocations we recommend that a comprehensive, methodical post-release disease surveillance plan is formulated and enacted. The free-living beaver populations in Great Britain or Norway are a potential source of unidentified hazards. Since unknown parasites have given rise to severe epidemics as a result of translocations, this disease risk analysis should be continually updated as new information becomes available, the literature scrutinised and immediate efforts made to use retrospective sample archives for parasite microarray and multi-organ parasite screens.

The transparent method of disease risk analysis used in this work, adapted by Disease Risk Analysis and Health Surveillance (DRAHS) at the Zoological Society of London (ZSL) for use in free-living wildlife from the World Organisation for Animal Health (OIE) Import Risk Analysis, and conforming to IUCN guidelines, allows for ready re-analysis and revised risk estimation. If the benefits of translocation are seen by Natural England to exceed the costs, and translocation of beavers proceeds, the disease risk analysis should be regularly updated on the basis of improvements in epidemiological and pathological knowledge and the results of post-release health surveillance.

The risks from disease in the conservation translocation of beavers currently held in enclosures, or any other captive facility, was not considered in this disease risk analysis. If there is a need to use captive beavers in a future translocation programme, revision of this disease risk analysis will be required.

This disease risk analysis must be regularly reviewed, as new evidence relevant to the threat of disease to mammal populations following beaver translocation becomes available, if it is to effectively assess and manage the risks from disease from beaver translocation.

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Forward

Natural England commission a range of reports from external contractors to provide evidence and advice to assist us in delivering our duties. The views in this report are those of the authors and do not necessarily represent those of Natural England.

1. Introduction

The Eurasian Beaver (Castor fiber) is believed to have become extinct in Great Britain during the 16th century as a result of human persecution, primarily hunting for fur, meat and castoreum (Nolet and Rosell, 1998). Across the species' range, exploitation reduced population size in the late 1990s to approximately 1200 individuals over eight discrete locations (ibid.). Following greater protection, reintroductions and natural dispersal, numbers in Europe have now recovered to over one million across 32 European countries, with the addition of some non-native Canadian beavers (Castor canadensis) in Russia, Luxembourg and Finland (Halley et al., 2012), with human-beaver conflict requiring careful management in some areas (Campbell-Palmer et al., 2015b). Small free-living populations are currently found in Scotland and England as a result of authorised and unauthorised releases. In addition, there have been licensed imports to captive facilities in England. Interest in the beaver's potential role as a keystone species in ecosystem restoration, specifically its ability to alter landscapes to the benefit of other species and for flood mitigation (Gaywood et al., 2008) has fed enthusiasm for reintroduction of the species in Great Britain.

1.1 Beavers in Great Britain

There are currently at least five known populations of free-living beavers in Great Britain: Knapdale and in the region surrounding Tayside in Scotland (Jones and Campbell-Palmer, 2014; Campbell-Palmer et al., 2018); the River Otter in Devon, the River Tamar in Devon and the River Stour in Kent (Claire Howe, pers. comm.). Beavers in Knapdale were imported from Norway in 2008 as part of a formal trial regulated by Scottish National Heritage (Jones and Campbell-Palmer, 2014). The Tayside beavers, first sighted in 2006, are of unknown origin but genetic testing of 25 individuals indicated that they were from three distinct lineages of German, most probably Bavarian, origin with heterozygosity and allelic richness comparable to the Bavarian source population (McEwing et al., 2015). This diversity suggests that the Tayside population is derived from multiple releases. Beavers on the River Otter were first sighted in 2007 and five were trapped and found on genetic analysis to be closely related and from either Bavaria or Baden-Wurtemberg (Brazier et al., 2020). The origin of the beavers on the Rivers Tamar and Stour is less certain but is believed to be Bavaria and Norway, and Poland and Bavaria respectively (Claire Howe, pers. comm.).

There are less certain reports of free-living beavers in at least one site in Wales and several sites in England which are of unknown number, origin and date of release (Jones and Campbell-Palmer, 2013). In addition, approximately 40 captive beavers are currently held in approximately 20 fenced sites, commonly known as 'enclosed releases', such as Ham Fen, Kent with further releases currently in progress (Claire Howe, pers. comm.) and in an unknown number of zoos, wildlife parks and other captive collections. Until 2018 the majority of beavers for enclosed releases were sourced from Bavaria, but subsequent releases have been sourced predominantly from the free-living wild population on the

River Tay in Scotland, with remaining animals sourced from enclosure sites in England and, in one case, from Bavaria (Heydon et al. 2021).

1.2 Health and disease of free-living beavers in Great Britain

The precise origin of some free-living beavers in Great Britain is unknown. The release of some beavers was not subject to disease risk analysis and they may harbour parasites novel to Great Britain.

2. Assessing the risks from disease in wildlife translocations for conservation purposes

Wildlife translocations for conservation purposes (reintroduction, reinforcement, ecological replacement and assisted colonisation) have become a key conservation tool to help restore species and/or ecosystem functions (IUCN 2013). Risks from disease associated with wildlife translocations arise because individual animals moved are a biological package, consisting of the host and all its associated parasites (Davidson and Nettles, 1992). The potential impact of infectious disease on the outcome of wildlife conservation interventions has only recently been recognised and detrimental effects may occur in the focus species or in other species within the wider destination ecosystem. The IUCN (2013) recommended health monitoring of animals involved in translocation programmes and current scientific opinion is that a disease risk analysis (DRA) should be conducted before a translocation takes place to in order to address the significant disease risks of translocation and to inform appropriate mitigation measures (Davidson and Nettles, 1992; Leighton, 2002; Miller, 2007; Sainsbury and Vaughan-Higgins, 2012).

DRA provides a structured, evidence-based process that can help decision makers understand the risks of disease-causing agents on translocation objectives and make decisions in light of these risks (Jacob-Hoff *et al.*, 2014). Several methods have been described. In 2012, Sainsbury and Vaughan-Higgins described a method for conducting a DRA for conservation translocations adapted from the OIE's (Murray *et al.,* 2004) approach for domestic animals. This DRA process follows a similar structure to the OIE's guidelines for DRA in domestic animal movements between countries (Murray *et al.*, 2004) but includes (i) hazards not known to cause harm (ii) infectious agents as hazards based solely on novelty to the source or in the destination (iii) hazards based on stressor effects (iv) non-infectious hazards and (v) ignores country borders and assesses the risk from parasite hazards on the presence or absence of geographical and ecological barriers in the translocation pathway. A series of steps are completed in the DRA: (1) mapping out the translocation pathway, (2) defining geographical and ecological barriers (3) hazard identification, (4) justification of hazard status, (5) risk assessment, (6) risk management and (7) risk communication. Disease is defined as any change in structure or function and can be infectious or non-infectious in origin. Non-infectious agents include trauma and toxins.

Sainsbury and Vaughan-Higgins' (2012) method ('the ZSL method') has been used for 23 translocation and reintroduction programmes conducted over the last 20 years.

3. Aims of this disease risk analysis for beaver reintroduction

The aim of this study was to assess the risks of disease related to the conservation translocation of beavers from either Norway, or any free-living population from any area of Great Britain, to England. Translocation of captive beavers from zoological collections, wildlife parks or any collection which houses or has housed exotic species, or where any component of the translocation pathway includes such collections, including enclosed releases, was not considered. The risk from disease from the translocation of beavers held in enclosures (enclosed releases) in Great Britain was omitted because of our developing understanding that at least some individual beavers from these groups of enclosure beavers have been exposed to exotic, non-native rodents in zoological collections, directly or indirectly, at some time over the last four decades. Considerable further work would be required to assess the risks from disease from beavers held in enclosures in Great Britain.

It is important to note that if, in the future, the translocation pathway is altered and, for example, includes (i) beavers from zoological collections, (ii) beavers that have been temporarily housed in zoological collections, or (iii) beavers in enclosures; a revised disease risk analysis would be required. Our previous work has shown that the risk from disease to a conservation translocation programme is comparatively high if animals are housed in zoological collections (Bobadilla Suarez *et al.*, 2017) primarily due to breach of ecological barriers and the potential for contraction of alien parasites from different ecological and geographical zones. Specifically, beavers that have been held captive in collections that have held, or are holding, exotic rodents may be directly or indirectly infected with novel parasites that present a hazard to the beavers themselves or other animals at the destination site(s). Severe disease outbreaks have been associated with translocations in which novel parasites have been introduced to immunologically naïve populations (see section 4.1).

We have communicated the findings from this DRA to Natural England. The intention is that Natural England can use this disease risk analysis, in the context of other evidence, for example ecological feasibility, to make a decision on the favourability of reintroduction and on the source of beavers for that intervention.

4. Materials and methods

In this report we use the ZSL method (Sainsbury and Vaughan-Higgins, 2012) described above, as developed from previous qualitative DRA methods for wildlife (Davidson and Nettles, 1992; Leighton, 2002) and domestic animals (Murray *et al.*, 2004) and modified by Bobadilla-Suarez *et al.* (2017) and Rideout *et al.* (2017) to: describe the translocation pathway; assess geographical and ecological barriers; identify disease hazards; assess the magnitude and probability of disease occurring, and propose methods to mitigate the risk from disease associated with the reintroduction of free-living beavers to England. Disease risk assessment was carried out according to the method described by the OIE (Murray *et al.,* 2004; Bruckner *et al.,* 2010).

4.1 Translocation Pathway(s) and geographical/ecological barrier considerations

A translocation pathway is a description of the route of the translocated animals that illustrates the points at which different types of hazards may potentially harm translocated individuals or the recipient ecosystem (Bobadilla-Suarez *et al.*, 2017). A major consideration in any given translocation pathway is whether any geographical (rivers, mountain ranges, seas) or ecological barriers are to be crossed, for example by bringing species that would normally be separated by habitat or behaviour into either direct or indirect contact with each other, thereby facilitating the spread of parasites that could not occur without human intervention. If a translocation crosses geographical or ecological barriers, then there is an increased probability of translocated or recipient populations being exposed to novel infectious agents.

This assessment is crucial because empirical evidence shows that the major epidemics of disease associated with translocations have arisen from source hazards (Cunningham, 1996; Sainsbury and Vaughan-Higgins, 2012; Viggers *et al.*, 1993). Source hazards are parasites present at the source but not at the destination (until the translocation occurs). An assumption that source and destination hazards are absent or minimal in a given translocation gives the translocation manager confidence that the overall risk from disease of a given translocation is markedly reduced. If source and destination environments are not separated by barriers, and populations of the translocated species, closely related or sympatric species and their parasites are contiguous, source and destination hazards do not require consideration and the overall risk from disease in the translocation may be reduced (Bobadilla Suarez *et al.*, 2017).

In this disease risk analysis, two potential source populations were considered: free-living beavers in Norway and free-living beavers in Great Britain. This disease risk analysis has not considered the translocation of beavers from, or in, captive collections such as fenced enclosures, wildlife parks or zoos, or translocations in which beavers are temporarily housed in wildlife parks or zoos, or any collection which houses or has housed exotic species. Animals in some captive collections, including zoos, are considered to have crossed an ecological or geographical barrier, as described above, because their proximity

to exotic species creates the potential for parasite transfer and the acquisition of non-native parasite species.

4.2 Hazard Identification

To identify hazards, we searched the scientific literature, examined unpublished data and sought experts' opinions. We used the search engines of Google Scholar, PubMed, Web of Knowledge and the ZSL library services.

We identified parasites (micro- and macro-parasites) known to be present in Rodentia, and specifically beavers, as well as multi-host parasites, using the scientific literature both in Great Britain and overseas, including a disease risk analysis undertaken for Eurasian beavers in Great Britain (Girling *et al.,* 2019b). Through consideration of (i) geographic distribution, (ii) occurrence (iii) pathogenesis and (iv) diseases associated with each parasite and (v) evidence for a negative impact on population numbers, we assigned, when possible, each hazard to an appropriate category as defined below (justification of hazard status). In carrying out our evaluation, we included evidence for susceptibility of beavers, other rodents and other mammals to each potential hazard, or similar agents of disease. We considered not only known pathogens, but also apparent commensal parasites, since the pathogenicity of many parasites of free-living wild animals is unknown. The translocation and the adaptation to the new environment could act as stressors and therefore alter the normal host-parasite dynamics resulting in disease. We also considered non-infectious agents or events and their association with disease, and similarly assigned these to their respective hazard category.

CARRIER HAZARDS were defined as commensal parasites, or parasites which do not ordinarily cause disease in the host animal following infection, which when the host is under stress associated with translocation or is subjected to factors that affect parasite dynamics, such as alterations in host density, may cause disease in transit or at the release site.

TRANSPORT HAZARDS were defined as those hazards that may be encountered during the transport (between the source and destination sites) which may be novel to the translocated animals and/or the release environment. Translocated animals can be a potential vehicle for introduction of these hazards to the destination site. Transport hazards are also those infectious agents moved with materials such as transport boxes, equipment, food and water.

POPULATION HAZARDS were defined as those non-infectious and infectious agents present at both the source and destination sites which potentially could have a negative impact on population numbers at the destination.

SOURCE HAZARDS were defined as a hazard present at the source site which would be novel at the destination site. Conversely, **DESTINATION HAZARDS** were defined as infectious agents present at the destination but not the source.

If no geographical or ecological barriers are crossed in a translocation then it may be assumed that there are no source or destination hazards (Bobadilla Suarez *et al.*, 2017).

4.3 Disease risk assessment

We assessed the risk of disease from each hazard using the method described by Sainsbury and Vaughan-Higgins (2012), with amendments provided by Bobadilla Suarez *et al.* (2017) and Rideout *et al.* (2017) and using the foundation provided by the OIE (Murray *et al.,* 2004).

4.3.1 Release assessment

Where relevant, we determined the biological pathways that might permit a beaver from the donor site to be released while infected with a parasite and the likelihood of its occurrence.

4.3.2 Exposure assessment

We described the biological pathways that might permit beavers and sympatric species at the destination to be exposed and infected with the parasite and the probability of this occurrence. We then described the processes required for the agent to disseminate through beavers and sympatric species populations and the probability of dissemination occurring.

4.3.3 Consequence assessment

We assessed the likelihood and severity of biological, economic and environmental consequences associated with the entry, establishment and spread of the hazard.

4.3.4 Risk estimation

Using the method described in Murray *et al.* (2004), we combined the results of the release, exposure, and consequence assessments to qualitatively assess the risk of disease associated with the hazard (negligible, very low, low, medium or high).

In our method, destination and population hazards have already “entered” the destination environment and a release assessment is not carried out for these hazards.

It is important to note that these estimates will be influenced by the information available and the risk attitudes of the specialists undertaking the DRA and therefore a reasoned, informed and transparent discussion of the risks of disease associated with each hazard is included within the DRA to justify each probability or risk estimation.

5. Results

5.1 Translocation Pathway

Two possible pathways were considered: the translocation of (i) free-living beavers from Norway and (ii) free-living beavers from Great Britain, to England. The destination site(s) remain unknown at this stage but are considered to be at any location in England.

5.2 Geographical and ecological barriers evaluation

The distance between source and destination site(s) is unknown as both have yet to be selected but could be as great as 2000 km if considering southern Norway as a source and 500 km if considering Scotland. Norway and England are separated by the North Sea. We do not know of any free-living rodents or fresh-water mammals which are contiguous between Norway and England. Many species of birds migrate seasonally between the two countries and could act as a potential route for parasite transfer. However, parasites infectious for birds may not be infectious for rodents. It therefore seems prudent to consider that a geographic barrier exists between Norway and England for the purposes of disease risk analysis. We have additionally considered the risk associated with the proximity of Norwegian beavers to neighbouring Swedish beaver populations. Populations inhabit the areas surrounding waterways which breach the 1600 km border between the two countries, such as the river Klarälven (Hartman, 1995).

The origin of some beavers in Great Britain is uncertain and, as stated above, there is evidence that at least some Tayside free-living beavers originated from Bavaria. The introduction of these beavers has potentially broken an ecological and geographical barrier, no specific disease risk analysis was undertaken prior to their importation and these beavers may have brought non-native parasites into Great Britain. Beavers in the Tayside area of Scotland are now known to have extended their range as far south as the outskirts of Stirling and into the Forth catchment (Campbell-Palmer *et al.*, 2018). They may move hundreds of kilometres when dispersing and cross watersheds in pursuit of new territories or mating opportunities (ibid.) and so continued natural dispersal seems likely.

Sympatric rodent and other mammalian species that are susceptible to the same parasites may be considered to increase the effective population size (Mathews *et al.*, 2006). Beavers live in close proximity to brown rats (*Rattus norvegicus*) and bank voles (*Myodes glareolus*), two ubiquitous species in Great Britain, with population numbers estimated at 7 million (Mathews *et al.*, 2018) and 27.4 million (ibid.) respectively. There are also robust populations of other small mammals that would be expected to overlap in habitat occupation with beavers such as, but not limited to, field voles (*Microtus agrestis*), pygmy shrews (*Sorex minutus*) and water shrews (*Neomys fodiens*). It is therefore probable that sympatric mammalian species form contiguous populations for parasite transfer purposes in many areas of Britain. Since non-native beavers have only recently (within decades) been translocated to Scotland, and other parts of Great Britain, it will be assumed that

there has been insufficient time for parasites to be transferred to all parts of England, and these free-living, recently reintroduced, beavers in Scotland, and other parts of Great Britain, will be assumed to cross ecological and geographical barriers if they are translocated to England. As a result, our analysis has included evaluation of the risks from disease posed by source and destination hazards for the translocation of free-living beavers from either Norway, or Great Britain, to England.

5.3 Hazard Identification

Seventy-eight potential hazards were identified (73 infectious hazards and five non-infectious hazards). Twenty-one of these were identified as requiring full disease risk analysis in order to determine the risk of disease that they presented as a consequence of beaver translocation. A list of the hazards receiving full disease risk analysis is provided in Table 1 and listed here by hazard category:

- Fully assessed SOURCE HAZARDS included *Francisella tularensis*, hantaviruses, specifically Puumala-virus (PUUV); *Echinococcus multilocularis* and *Trichinella* species.
- Fully assessed CARRIER HAZARDS included *Leptospira* species; *Yersinia enterocolitica* and *Y. pseudotuberculosis; Mycobacteria* species.; *Emmonsia crescens;* gram-negative enteric bacteria; *Streptococcus castoreus; Stichorchis subtriquetrus; Toxoplasma gondii* and *Eimeria* species.
- Fully assessed POPULATION HAZARDS included Road Traffic Collision; Persecution; Captivity During Translocation; *Toxoplasma gondii* and SARS-CoV-2.
- Fully assessed DESTINATION HAZARDS included hantaviruses, specifically Seoul-virus (SEOV) and Tatanale-virus (TATV).

There may be a need to evaluate TRANSPORT HAZARDS once a transit route between the source and destination sites has been formulated.

In addition, we evaluated the risks from disease associated with *Giardia duodenalis*, *Cryptosporidium parvum* and *Mycobacterium* species (risk to domestic and free-living wild animals).

The remaining fifty-seven potential hazards received detailed scientific review as described in Appendices and Table 8. The scientific reviews showed that these hazards were, at least currently, of very low or negligible disease risk as a result of the translocation of beavers. These hazards should be re-evaluated with each succeeding translocation as information may become available and our understanding improves.

Table 1 Potential hazards identified for the translocation of beavers (Castor fiber) to England and for which full disease risk analysis was carried out

POTENTIAL HAZARD		Beaver susceptibility to infection and/or disease**	Other *Rodentia* susceptibility to infection and/or disease	Reference	Hazard Category
Viral	Hantaviruses – SEOV, TATV Hantaviruses – PUMV	N/K	Yes	Duggan *et al.*, 2017; Pounder *et al.*, 2013; Thomason *et al.*, 2017 Vapalahati *et al.*, 2003	Destination Source
	SARS-CoV-2	N/K	Yes	Chan *et al.*, 2020; Bao *et al.*, 2020	Population
Bacterial	*Leptospira* species	Yes (I, D)	Yes	Nolet *et al.*, 1997	Carrier
	Francisella tularensis	Yes (I, D)	Yes	Morner *et al.,* 1988a; Mörner & Sandstedt, 1983; Schulze *et al.*, 2016	Source
	Yersinia pseudotuberculosis and *Y. enterocolitica*	Yes (I, D)	Yes	Nolet *et al.*, 1997	Carrier
	Gram-negative enteric bacteria	Yes (I, D)	Yes	Pratama *et al.*, 2019; Pilo *et al.*, 2015; Dollinger *et al.*, 1999	Carrier
	Streptococcus castoreus	Yes (I, D)	No	Lawson *et al.*, 2005; Schulze *et al.*, 2015	Carrier
	Mycobacterium species	Yes (I, D)	Yes	Gavier-Widen *et al.*, 2012; Nolet *et al.*, 2007	Unclassified Carrier
Endoparasites	*Stichorchis subtriquetrus*	Yes (I, D)	No	Demiaszkiewicz *et al.*, 2014	Carrier
	Echinococcus multilocularis	Yes (I, D)	Yes	Barlow *et al.,* 2011; Britton and Barlow, 2019	Source

POTENTIAL HAZARD		Beaver susceptibility to infection and/or disease**	Other *Rodentia* susceptibility to infection and/or disease	Reference	Hazard Category
	Trichinella species	Yes (I)	Yes	Seglina *et al.*, 2015; Rozycki *et al.*, 2020	Source
Protozoa	*Toxoplasma gondii*	Yes (I, D)	Yes	Herrmann *et al.*, 2013	Carrier Population
	Giardia duodenalis	Yes (I)	Yes	Paziewska *et al.*, 2007; Tsui *et al.*, 2018; Sroka *et al.,* 2015	Unclassified
	Cryptosporidium parvum	Yes (I)	Yes	Paziewska *et al.*, 2007; Mackie, 2014	Unclassified
	Eimeria species	Yes (I)	Yes	Demiaszkiewicz *et al.*, 2014; Campbell-Palmer *et al.*, *submitted*	Carrier
Fungi	*Emmonsia crescens*	Yes (I, D)	Yes	Morner *et al.*, 1999; Dolka *et al.*, 2017	Carrier
Non-Infectious	Road traffic collisions	Yes	No	Brazier *et al.*, 2020; Campbell-Palmer *et al.*, 2015b; Stefen, 2018	Population
	Captivity during translocation	Yes	No	Harrington *et al.*, 2010; Goodman *et al.*, 2012	Population
	Illegal persecution	Yes	No	Campbell-Palmer *et al.*, 2015b; Stefen, 2018	Population

* Because of the paucity of data available on both infectious and non-infectious hazards in free living beavers, a qualitative judgement of beaver susceptibility to some hazards, based on expert opinion, was used when it could not otherwise be supported by evidence in the scientific literature. Beavers were considered to be “likely susceptible” to those parasites isolated in closely phylogenetically related species but also to those multi-host parasites known to infect many other mammalian families and orders.

** N/K = Not Known; I = Infection; D = Disease in Species

5.4 Disease risk analyses

Full disease risk analysis was performed on 21 hazards which hazard identification indicated required such detailed evaluation (see Appendices 1 - 19). One hazard was estimated to be of negligible risk (*Mycobacterium* species (risk to domestic and free-living wild animals)), three hazards were estimated to be very low risk (Hantaviruses (SEOV/TATV); *Giardia duodenalis*; *Cryptosporidium parvum*), three low risk (*Francisella tularensis; Eimeria* species; *Mycobacterium* species (risk to beavers)), 11 medium risk (Hantaviruses (PUUV); gram-negative enteric bacteria; *Streptococcus castoreus*; *Stichorchis subtriquetrus; Trichinella* species*, Toxoplasma gondii; Emmonsia crescens;* SARS-CoV-2*; road traffic collisions; illegal persecution; captivity during translocation) and three high risk (*Yersinia enterocolitica and Y. pseudotuberculosis; Leptospira* species; *Echinococcus multilocularis*).

*Risk evaluated at 5 May 2020. The risk of disease in beavers from SARS-CoV-2 will fluctuate as infection prevalence in humans changes temporally and spatially and this hazard may need to be re-evaluated if, and before, beaver translocation proceeds.

6. Discussion

In this disease risk analysis for the conservation translocation of free-living Eurasian beavers from Norway, or Great Britain, to England we have described the translocation pathway; assessed geographical and ecological barriers to the spread of parasites; identified, reviewed and evaluated 78 (73 infectious and five non-infectious) potential hazards; and carried out a full disease risk analysis on 21 selected hazards. Both translocation pathways (from Norway or Great Britain) were found to be crossing geographical barriers and consequently an in-depth and detailed disease risk analysis was required which included source and destination hazards in addition to carrier and population hazards. No transport hazards have been identified to date but when the specific translocation route has been determined these hazards can be reviewed.

Of the 21 hazards selected for full disease risk analysis, 11 were medium risk (hantaviruses (PUUV); gram-negative enteric bacteria; *Streptococcus castoreus*; *Stichorchis subtriquetrus; Trichinella* species*, Toxoplasma gondii;* SARS-CoV-2; *Emmonsia crescens*; road traffic collisions; illegal persecution; captivity during translocation) and another three high risk (*Yersinia enterocolitica and Y. pseudotuberculosis; Leptospira* species; *Echinococcus multilocularis*) for disease as a consequence of translocation. Of those 14 high and medium risk hazards, seven are triggered by stressors and later in this discussion we set out how to minimise the effects of these stressor-related hazards as a group.

Two non-infectious hazards were assessed as medium and may be a threat to small populations of beavers post-translocation: road traffic collisions and persecution. There is reliable evidence of beaver persecution in free-living populations, including in Scotland, and local community involvement in translocation projects would be beneficial to combat this hazard. Consideration of traffic density in the vicinity of release sites will assist in the mitigation of road traffic collisions.

Zoonotic hazards of high and medium risk. The disease risk analysis identified three zoonotic hazards of high or medium risk of disease in the human population. *Echinococcus multilocularis* was analysed as of high risk of disease to people. We consider it a high priority in undertaking beaver translocations to maintain the UK's infection-free status from this cestode because of the severe biological and economic consequences which would result from its incursion. There remains a possibility that unlicensed imports of beavers in the past have already introduced this parasite to Great Britain and for this reason we recommend that, should this population be used for translocations to England, robust and comprehensive disease surveillance is used to monitor the population post-release. Given (i) the further spread of *Echinococcus multilocularis* through Scandinavia since Roberts *et al.* (2012) carried out their disease risk analysis for the importation of this parasite to the UK with beavers, and (ii) the understanding that *Echinococcus multilocularis* could have evaded detection in foxes in Norway due to sampling statistics, we estimated that the risk of *Echinococcus multilocularis* incursion is greater from the translocation of free-living Norwegian beavers

than those from Great Britain. Further reduction in risk can be achieved by prioritising free-living beavers proven to have been born in Great Britain for translocations to England.

Trichinella species were analysed as of medium risk for disease in the human population. Maintaining the UK's infection free status for this nematode parasite is, like for *Echinococcus multilocularis,* important given the severity of the disease in people and the high economic costs of disease prevention should *Trichinella* species become endemic in the UK. As for *Echinococcus multilocularis* the risk from disease is reduced if a choice is made to translocate beavers from Great Britain rather than Norway.

Puumala-virus (PUUV), a hantavirus, represents a medium risk source hazard if Norway is chosen as the source for beavers, given the associated disease syndromes in people. There is uncertainty in the likelihood that beavers can be infected on release, and pre-translocation screening using stored archive samples would be of value to improve our risk estimation. If translocation proceeds, further information on prevalence of PUUV infection in beavers can be gathered.

The elevated risk from these three zoonotic infectious agents if Norway is chosen as the source population leads us to recommend free-living beavers in Great Britain as the source for translocations. If Norway was selected as the desired source for non-disease reasons, we recommend the disease risk analysis for all three of these agents is revised to ensure it is up to date before translocation proceeds.

SARS-CoV-2 was considered of medium risk of disease in translocated beavers but the prevalence in humans is likely to fluctuate as control of the pandemic continues, and the distribution of the virus changes temporally and spatially. Disease risk assessment for SARS-CoV-2, and risk management options, may need to be updated if beaver reintroduction is chosen as a course of action.

Stressor-associated disease and translocation of beavers. In our disease risk analyses, seven of the high and medium risk hazards were precipitated by stressors. Translocation has been shown through detailed research to be a substantial stressor for all animal species (Dickens *et al.,* 2010) and therefore detailed planning of disease risk management for beaver translocation is imperative.

Stressors such as translocation may reduce immunocompetence and consequently immunocompromised individuals will be more susceptible to disease if infected, or from commensal organisms that do not ordinarily cause disease in healthy individuals. We have identified nine stressor-related hazards for which we anticipate a risk of disease (seven of which are high or medium risk), based on cases of previous morbidity and mortality in beavers. In previous translocations, fatalities have been attributed to yersiniosis, leptospirosis and mycobacteriosis (Nolet *et al.*, 1997). In addition, enteric disease from *Stichorchis subtriquetrus* infection (Howie, 2009); adiaspiromycosis (Dolka *et al.,* 2017); gram negative enteric bacteria (Cranwell, 2009); *Toxoplasma gondii* (Hermann *et al.,* 2013) and *Streptococcus castoreus* (Lawson *et al.*, 2005) may have contributed to mortalities in beavers triggered by stressors.

It is widely understood and accepted that stress can lead to immunocompromise (Dhabhar and McEwen, 1997; Dickens *et al.*, 2010; Glaser and Kiecolt-Glaser, 2005). Stress has been suggested to be an inevitable component of animal translocations, which can occur at multiple stages including capture, transport and captivity (Dickens *et al.*, 2009, 2010; Teixeira *et al.*, 2006). Dickens *et al.* (2010) state that all translocated animals will be chronically stressed to some extent when released. Further to this, several reintroduction failures, including of rodents, have been attributed to stress. For example, stress was considered to be a key factor in the failure of a reintroduction programme of Vancouver Island marmots (*Marmota vancouverensis*) in Canada, in which all six died within a year of release (Bryant *et al.*, 2002). Shen *et al.* (2016) experimentally demonstrated that transportation stress can alter the immunity of chronically infected mice leading to the reactivation of dormant bradyzoites and acute toxoplasmosis. This process may be similar in other rodents, including beavers. It is therefore essential that measures are taken to minimise stress to beavers at all stages of the translocation process.

Disease risk management and post-release health surveillance (DRM PRHS). Principles of good disease risk management in translocations will reduce the risk from disease for a high proportion of the hazards we have analysed. For example, the risk of exposure to parasite hazards will be reduced through good hygiene during the translocation process. Maintaining high standards of biosecurity should be standard practice and substantial knowledge of efficient methods is available from our previous work and reported in Vaughan-Higgins *et al.* (2017). We have provided disease risk management recommendations to reduce the risk from disease in each disease risk analysis. Our standard practice developed over 30 years of monitoring translocations in England is to convert the disease risk analyses recommendations into a comprehensive, evidence-based, practically orientated Disease Risk Management and Post-Release Health Surveillance (DRM PRHS) protocol. If Natural England decides, following a review of evidence, that translocation of beavers to England is warranted, we strongly recommend that a DRM PRHS protocol is formulated.

DRM PRHS and minimizing the effects of stress. Given the evidence that seven stressor-associated hazards are of high or medium risk to this proposed translocation, the DRM PRHS protocol will consider methods to minimise stress in detail. Some preliminary comments are made here.

Contact with humans should be reduced wherever possible and care should be taken to ensure that human scent is not present within beaver crates or enclosures, for example through wearing gloves (Campbell-Palmer and Rosell, 2010, 2013). During the process of trapping beavers, appropriate traps should be used and checked regularly in order to ensure beavers do not remain in traps for long periods of time. When contact with beavers is necessary, for example to move them from traps to transport containers, reduction of surrounding noise, movement and minimal handling times should be implemented (Campbell-Palmer and Rosell, 2015).

Appropriate stocking densities should be observed during any periods of captivity, including transport. Beavers of the same family should be trapped and housed together, and minimal trapping intervals should be present between trapping members of the same

family (Campbell-Palmer and Rosell, 2013). It is also important that beavers from different families are not housed together (Campbell-Palmer and Rosell, 2013). During transportation, sufficient absorbent bedding, ventilation, food and water should be provided. Including used bedding from an individual in transport crates may also help to reduce stress (Campbell-Palmer and Rosell, 2010).

The captive periods for free-living beavers should be kept to a minimum. Quarantine can ensure that enclosures are as naturalistic as possible in many cases. Access to fresh water deep enough to allow beavers to fully submerge is essential, along with appropriate shelter, space and substrate to allow expression of normal behaviours such as digging (Campbell-Palmer and Rosell, 2010). It is also important that family groups of beavers are housed out of sight of other groups, for example throuh the addition of visual barriers to closely positioned enclosures (Campbell-Palmer and Rosell, 2010).

Collection of samples, for example for parasites, should be collected non-invasively wherever possible to reduce the necessity of repeated handling, general anaesthetic and/or confinement. Consideration should also be given to the timing of releases, avoiding winter months when lower temperatures and food shortages may increase the risk from stressor-associated disease.

Further information on animal stress physiology and its effects can be found in Dickens *et al.* (2010). Detailed consideration of stress mitigation will be made in the DRM PHRS protocol.

Parasite conservation and translocation of beavers. Commensal parasites which induce disease in the presence of stressors are an important component of biodiversity and, as such, efforts should be made, if possible, to conserve them at the same time as keeping disease under control. Careful use of therapeutic protocols can allow for prevention without elimination, while maintaining host immune responses, as we have shown in the conservation of the commensal parasite, *Isospora normanlevinei*, which was associated with stressor-associated disease in reintroducing cirl buntings to Cornwall (McGill *et al.* 2010). The Eurasian beaver harbours at least one species-specific parasite, the beaver beetle *Platypsyllus castoris* (see Appendix 20), and parasite conservation should, we argue, therefore be an integral and important component of a DRM PRHS protocol.

Disease risk analysis method. The disease risk analysis reported here has been completed using the ZSL method described by Sainsbury and Vaughan Higgins (2012) and deployed in 23 wild animal translocations to date. This ZSL method uses the foundation of the OIE's disease risk assessment (Murray *et al.*, 2004), a reasoned, logical and transparent approach which adheres to, and contributed to, IUCN guidelines, in DRA. Transparency is crucial to make the qualitative judgements of release, exposure and consequence absolutely clear to stakeholders. Transparency of method and results also ensures that in each succeeding beaver translocation, the risks from disease can be easily and quickly reassessed, ensuring lessons are learned and improvements made. In addition, the disease risk analysis can be utilised by managers of future translocations in

the same or closely related species, anywhere in the world. Information from previously published, transparent, evidence-based disease risk analyses, for example Roberts (2012), has been utilised in this disease risk analysis reported here.

Rapid turnaround of this disease risk analysis. DRAHS have completed 23 disease risk analyses for conservation translocations using the ZSL method but we have never completed a complex DRA (involving source and destination hazards) as rapidly as in this instance. To complicate our work the aims of the disease risk analysis were modified twice within four months. Our ability to turn this DRA around, given these constraints, reflects well on our developing expertise in disease risk analysis for conservation translocations and the hard work of the team involved. However, given the pressure to complete this disease risk analysis within such a short period, some literature for example from Russia has been unavailable, and we have not had sufficient time to request peer review of some of the information in some analyses. To ensure good decision making over forthcoming months, assuming translocation proceeds, further reflection and peer review, will be essential.

Unidentified and poorly understood hazards in the source populations. Geographical or ecological barriers are crossed in this translocation whatever the source population chosen (free-living beavers in Norway or Great Britain). Therefore, either source population may harbour non-native parasites and indeed four source hazards of high or medium risk have been identified and analysed. The risk from source hazards requires careful and thorough analysis because empirical evidence shows that the major epidemics of disease associated with translocations have primarily arisen from these hazards (Sainsbury and Vaughan-Higgins, 2012). For example, chytridiomycosis in amphibians arose as a result of transfer of the causal infectious agent, *Batrachochytrium dendrobatidis*, to novel hosts and environments, and the disease has subsequently led to extinctions of many amphibian species (Scheele *et al.*, 2019). Closer to home, squirrelpox viral disease illustrates the same threatening process, in decimating populations of red squirrels in Great Britain, following the introduction of the squirrelpox virus with grey squirrels in the 19th century. In both examples the parasites were not known to science at the time the first epidemics of disease occurred. In addition, the squirrelpox epidemic was undetected for decades and has continued for over a century since the first outbreak, which shows that immediate positive translocation results do not preclude later disease outbreaks. The parasites and diseases of the Eurasian beaver are poorly described and evaluated, and it remains a realistic possibility that beaver populations in either Great Britain or Norway harbour an unidentified, novel parasite capable of inducing an epidemic in naïve rodent populations in the UK. In undertaking this disease risk analysis, we have been alert to the need to detect source hazards of greatest risk to translocation and have used the criteria set out by Rideout *et al.*, (2017) to scrutinize the potential hazards to assess the likelihood that these parasites would give rise to an epidemic. We searched for recently identified parasites or new virulent strains of known pathogens, and will continue to scrutinise the published literature, grey literature and reports before translocation proceeds.

In order to assist in identifying unknown parasites which may present a source hazard for the translocation of beavers, we recommend retrospective screening of stored beaver sample archives, from both healthy and diseased animals using, for example DNA microarrays, which can rapidly screen samples for genetic sequences from viruses, bacteria, protozoa and fungi. Sequences are cross-referenced against a databank of known organisms to identify the closest match. Screening programmes would be advantageously carried out before translocation goes ahead so that disease risk analyses can be reassessed.

In addition, uncertainty as to the origin of many beavers already present in Great Britain, and the risk of parasites yet to be identified and described in beavers, means that sustained post-release health surveillance of beaver populations will be required. A coordinated, methodical and systematic approach to clinical and pathological examination of all beavers found sick or dead is crucial to improve our understanding of beaver parasites and to ensure early detection of parasites which may cause disease outbreaks in other, naïve hosts. Historically, due to technology or time limitations, pathogens may have been missed on screening. For example, PCR testing and microarrays are relatively novel technologies which have greatly improved detection of viruses in particular; however, even nowadays, such techniques are not routinely deployed in standard post-mortem examination.

Beavers from Great Britain for conservation translocation to England. There is currently support within the beaver conservation community for careful use of the resource offered by the expanding populations of beavers in Great Britain, for example beavers in Tayside and surroundings. Free-living beavers in Great Britain are, in some cases, of uncertain origin, not subject to disease risk analysis prior to importation. If plans are made to utilise these beavers for translocation to England, we strongly recommend that their uncertain origin and potential to harbour non-native parasites is appreciated. Therefore, we recommend that, following translocation, substantial resources are placed in health and disease surveillance of beaver and sympatric rodent populations in the vicinity of the release site(s). Assuming Natural England approves reintroduction to England, we would be able to map out this surveillance programme as a component of the DRM PRHS protocol.

The influence of beaver translocation on the control of mycobacteria in England. There are severe economic costs to the control of mycobacteria in domestic livestock in England. Therefore, we have considered whether there is any additional risk from mycobacteria to livestock as a consequence of beaver translocation. Scientific evidence shows that rodents in the British Isles are not an important reservoir of *Mycobacterium bovis* (Delahay *et al.*, 2007): for example prevalence in the wood mouse was 0.006% (n = 333) and in the yellow-necked mouse 2.78% (n = 36). There are no reported cases of mycobacterial disease in beavers attributable to *M. bovis.* Detailed research in the UK has established the most important hosts for *M. bovis* and they do not include rodents. Therefore, we considered the risk from beaver translocation to the control programme for *M. bovis*-associated tuberculosis in livestock in England is negligible. Notwithstanding this evaluation, we recommended (i) beavers for translocation are selected from areas, such

as Scotland and Norway, currently *M. bovis*-free and (ii) stringent biosecurity protocols are adhered to in beaver translocations. We are confident that biosecurity protocols, as we have previously used in DRAHS-led translocations (Vaughan-Higgins *et al.,* 2017), will prevent risk from the translocation process. There is a low risk from mycobacteria as a carrier hazard for beavers, as a consequence of the stress of translocation, and associated with *Mycobacterium avium* (MAC) complex infection.

This disease risk analysis must be reviewed on the basis of changing evidence. This disease risk analysis will require regular review in the light of changes in evidence and knowledge on the diseases of threat to beavers and sympatric species following beaver translocation, if it is to effectively assess the risks from disease in translocation.

Appendix 1 Disease Risk Analysis for the Source and Destination Hazard Hantaviridae

Hantaviruses are notifiable RNA viruses (Order Bunyavirales, Family Hantaviridae) found primarily in rodent, bat and insectivore reservoir hosts and identified as a significant emerging zoonotic risk in Europe (ECDPC, 2019a).To date, 48 species of hantavirus have been identified (Forbes et al., 2018). However, in the host, viral species identification is difficult due to the cross-reactivity of antibodies with viral antigen (Vaheri et al., 2008) especially if using saliva samples (Jameson et al., 2014). For example, Seoul-virus (SEOV) cross-reacts with Hantaan-virus (HTNV) and Sin Nombre-virus (SNV) with PUUV and there may be other unidentified cross-reacting species (ibid.). Definitive diagnosis is by RT-PCR for viral antigen and sequencing from tissue samples.

Each species of hantavirus has traditionally been regarded as host-species specific causing mostly asymptomatic and persistent (possibly lifelong) infection in its reservoir host but only transient, spillover infections in other animal species (Forbes et al., 2018). As reservoir hosts are chronically infected, both antibodies and viral antigen should be detectable (Vaheri et al., 2008); the presence of antibodies without antigen is indicative of transient and probable spillover infection (Forbes et al., 2014). The hantaviruses of interest with regard to this DRA, identified in Europe, with their primary reservoir host are shown at Table 2.

Table 2 Hantavirus species identified in Europe with reservoir hosts (From Klingstrom et al., 2002; Heyman et al., 2002; Pounder et al., 2013)

Hantavirus	Reservoir host
Seoul-virus (SEOV)	Rattus norvegicus (brown rat) and Rattus rattus (black rat)
Puumala-virus (PUUV)	Myodes glareolus (bank vole)
Tula-virus (TULV)	Microtus arvalis (common vole)
Tatenale-virus (TATV)	Microtus agrestis (field vole)
Dobrava-virus (DOBV)	Apodemus flavicollis (yellow-necked mouse)
Saaremaa-virus (SAAV)	Apodemus agrarius (striped field mouse)
Topografov (TOPV)	Lemmus sibericus (Siberian lemming)
Khabarovsk (KBRV)	Microtus fortis (reed vole)

However, hantaviruses may have the potential to spread to new reservoir hosts. Phylogenetic analysis of hantavirus sequences by Zhang (2014) suggests cross-species transmission has occurred historically. Specifically, SEOV has been found in several rat species (Holmes and Zhang, 2015). There is also evidence that HTNV and SEOV have expanded their host ranges in China based on the identification of SEOV antigen in shrews and HTNV in house mice and brown rats (Fang et al., 2015). A meta-study of all peer-reviewed reports of hantavirus infections between 1971 and 2015 found several instances of interspecies sharing, particularly in voles (Millholland et al., 2018). Additionally, Schmidt-Chanasit et al. (2010) challenged assumptions of viral co-evolution with host species, concluding that TULV is a promiscuous hantavirus with a large range of susceptible hosts.

PUUV, in common with its reservoir host, the bank vole, is widely distributed throughout continental Europe. Figure 1 shows host distribution and recorded cases of infection in humans. An average of 50 cases a year in humans are reported in Norway and rarely in southern Sweden (Vapalahti et al., 2003). The incidence of DOBV is predominantly in Eastern Europe and the Balkans (ibid.).

Figure 1 The distribution of Myodes glareolus and human hantavirus infections. The distribution of Myodes glareolus and human hantavirus infections. Rodent figure indicates

countries where PUUV sequences are available from M. glareolus; dots indicate human hantavirus infections caused by PUUV; black dots indicate cases confirmed by cross-neutralisation tests or RT-PCR and sequencing. (Source: Vapalahti et al., 2003).

SEOV is thought to have originated in China and has been found in wild rats in the UK, Belgium and France and in pet rats in Sweden (Ling et al., 2019). SEOV has not been found in rats in Germany (Hoffman et al., 2018). It is not known whether SEOV is present in Norway.

Source Hazard - Justification of Hazard Status

Until recently only SEOV had been identified in the UK, in both pet and wild rats (Webster and Macdonald, 1995; Jameson et al., 2014; Duggan et al., 2017). However, a novel arvicoline virus (Tatenale-virus) was identified in a field vole in northern England from samples collected and sequenced between 2009 and 2011 (Pounder et al., 2013) and a closely related virus in 17% (n=8/48) of field voles examined in Kielder Forest in 2015 (Thomason et al., 2017). Thomason et al. (2017) concluded that the divergence in the two viruses was strongly suggestive of long-standing endemicity (ibid.) which may suggest that Tatanale virus is also prevalent in other areas of Britain. Pounder et al. (2013) additionally noted that Tatanale virus antibodies cross-reacted with PUUV antigen. The primary hosts for PUUV and DOBV, respectively the bank vole and yellow-necked mouse (Apodemus flavicollis), are widely present in the UK, but there is reportedly no evidence for infection of either of the host species in the UK (Duggan et al., 2017).

A number of cases of hantavirus infection have been recorded in humans in the UK but it is not always known with certainty which species was involved as the serotype is often not recorded (Bennett et al., 2010) or may have been misattributed due to cross-reactivity (Duggan et al., 2017). The earliest human cases noted were in Northern Ireland in the 1990s and were most probably attributable to SEOV (Clement et al., 2014). Infections with HTNV, SEOV, DOBV and PUUV (n=19, 26, 2 and 1 respectively) were recorded in a seroprevalence study on behalf of Public Health England of 545 fancy rat owners and workers at high occupational risk of exposure to hantavirus infection and 300 randomly selected control samples in England between 2013 and 2014 (Duggan et al., 2013). However, as HTNV is not known to exist outside central and Eastern Asia and DOBV and PUUV have not been found in studies of wildlife in Great Britain, it was concluded that positive test results might be due to cross-reaction with another hantavirus such as TATV. Clement et al. (2014) similarly cautioned that the PUUV cases reported in two other studies (Lloyd, 1991; Jameson et al., 2013) in Great Britain could be attributable to cross-reaction rather than indicating a true wildlife reservoir of PUUV in Great Britain.

As PUUV is endemic in bank voles in Scandinavia, including Norway, and is not believed to be present in the UK, iIt should therefore be considered as a potential source hazard for the translocation of beavers from Norway.

Risk Assessment

Release assessment

Hantaviruses may persist for some time outside the host. For example, PUUV and TULV have been shown to remain infectious for up to 11 days at room temperature and up to 18 days at 4°C (Kallio et al., 2006). Cool and damp conditions may prolong viral survival (Forbes et al., 2018). Infection is by aerosol inhalation of viral particles or intense contact with hosts such as biting, grooming and sharing food resources (ibid.). Juvenile rodents may be protected from infection for up to 80 days by maternal antibodies and prevalence in male rodents is higher, probably due to intra-specific aggression and dispersal distances (Kallio et al., 2013). Co-infection with parasites is variably positively and negatively associated with virus infection in bank voles (Deter et al., 2008; Salvador et al., 2011).

Bank voles and beavers in Norway are likely to be sympatric in riparian margins. Chronically infected rodent hosts will shed PUUV in urine, faeces and saliva which may persist in the environment for up to 18 days in cool, damp conditions. There is a low probability that beavers could be exposed to viral particles when foraging on land. It is not known whether beavers are susceptible to infection with hantaviruses either as reservoirs through host switching or as accidental hosts. Girling et al. (2019) found no evidence of hantaviruses from kidney tissue and urine samples from 20 free-living beavers examined between 2010 and 2015 from Knapdale and Tayside in Scotland, Telemark, Norway, and Bavaria, Germany using a pan-hantavirus nested PCR. There are not believed to be any other reports of testing in beavers in Europe (ibid.). There is therefore a very low likelihood of a translocated beaver being infected.

Exposure assessment

Studies in laboratory rodents have shown that chronic hantavirus infection may result in occasional or no viral shedding (Forbes et al., 2018). However, a capture-mark-recapture investigation of naturally occurring PUUV infection in bank voles suggested that free-living host animals may be infectious for life (Voutilainen et al., 2015), and shed virus in urine, faeces and saliva (ibid.). Gastrointestinal transmission has also been demonstrated experimentally (Witkowski et al., 2017).

Accidental hosts are not believed to be infectious. The only exception to this is occasional reports of human-to-human transmission of Andes-virus (ANDV), a hantavirus species specific to South America which is believed to have unique anti-inflammatory properties that enable it to evade the host's salivary anti-viral mechanisms (Forbes et al., 2018). Accidentally infected hosts are believed to clear infection quickly and are not considered a source of infection to other animals (Klingstrom et al., 2002). Host-switching of hantaviruses has been reported and so there is a very low likelihood that an infected beaver could act as a reservoir and shed virus PUUV into the environment through its urine, faeces or saliva or could infect con-specifics by fighting, grooming or food-sharing.

If beavers were persistently infected, there is a low likelihood that new beaver colonies at the destination could act as a reservoir of infection to sympatric species and humans. In particular, as the known host for PUUV, bank voles, are native to Great Britain and likely to share habitat in riparian margins with released beavers, there is a medium likelihood that sympatric bank voles could be exposed to and infected with PUUV. These animals could then act as a reservoir for disease transmission. There is a medium likelihood of dissemination of PUUV at the destination.

Consequence assessment

There is a very low likelihood that one beaver will become infected with PUUV. Infection of rodent reservoir hosts is believed to be asymptomatic; however, subtle histopathological changes have been recorded in infected animals in combination with a robust antibody response (Simmons et al., 2002). Spillover infection to closely related sympatric species is known to occur but it is not known whether clinical disease results (ibid.). Simmons et al. (2002) reported that experimental infection of Syrian hamsters (Mesocricetus auratus) with PUUV, SEOV and DOBV resulted in asymptomatic serological conversion. Klingstrom et al. (2002) further suggested that accidental spillover infections of non-reservoir hosts result in rapid clearing of the virus. However, experimental infection of immunocompromised mice with SEOV resulted in chronic wasting disease (Golden et al., 2015). There is a very low likelihood of a disease outbreak in beavers or sympatric rodents at the destination.

PUUV is known to cause disease in humans. In 2017, the last year for which data is available, Germany recorded the highest number of cases of human hantavirus infection of any country in Europe, at 1717 cases compared to 26 in Norway, 158 in Sweden (mostly from northern Sweden) and 0 in the UK (ECDC, 2019). Baden-Wurttemberg, in south-west Germany, and Bavaria account for the majority of cases in humans in Germany (ECDC, 2014). Two clinically significant syndromes have been recognized in humans (GOV.UK, 2019): Haemorrhagic fever with renal syndrome (HFRS) and Hantavirus pulmonary syndrome (HPS). Of these, only HFRS is known in Europe, usually causing a milder form of disease known as nephropathia epidemica (NE) (Klingstrom et al., 2002). In rare cases, infection may lead to chronic conditions such as Guillain-Barre syndrome (ECDPC, 2019). There is a medium likelihood of disease in humans in contact with infected beavers during the translocation.

Risk estimation

The likelihood of a beaver from Norway being exposed to PUUV at the source is low and the probability of infection is very low. The likelihood of dissemination to con-specifics and sympatric species is medium. There is a very low likelihood of a disease outbreak in rodents and a medium likelihood of disease in humans. The overall risk from PUUV as a novel source hazard is VERY LOW for rodents and MEDIUM for humans.

As hantaviruses have been shown to cause morbidity in immunocompromised mice, if beavers are subsequently found to be susceptible to infection, this DRA may have to be updated to consider the risks to beavers of hantaviruses as a carrier hazard.

Destination Hazard - Justification of Hazard Status

As data on the distribution of hantaviruses in rodent reservoirs in the UK and Europe is scant, beavers imported from Norway, or that have previously been imported from Germany (and currently free-living or in enclosures in Great Britain), may be naïve to SEOV and Tatanale hantaviruses which may be present at the destination site(s). Hantaviruses should therefore be considered as a destination hazard for the translocation of beavers.

Risk Assessment

Exposure assessment

Prevalence of the newly-identified Tatenale-virus (TATV) found in field voles is not known but this virus is believed to be of long-standing endemicity in the UK so may be widely distributed throughout the country. It has not been reported outside the UK. Hantaviruses may have the potential to host-switch but, to date, there has been no evidence of TATV exposure or infection in other rodent species. Chronically infected rodents will shed the virus in urine, faeces and saliva. As beavers forage in woodland and scrub on riparian margins there is a low likelihood of a beaver being exposed to TATV and a very low probability of at least one beaver being infected. The only other hantavirus known to be present in wildlife in the UK is SEOV, identified in brown rats. Although there has been limited host switching from rats to other murines and shrews in China, SEOV has not, to date, been found in other species in Europe and there is no recorded infection of beavers. As rats and beavers may occupy similar habitat, there is a medium likelihood of contact and exposure to SEOV through viral shedding via faeces, urine and saliva but a low likelihood of infection of beavers.

Consequence assessment

There is a very low likelihood of one beaver being infected with TATV and a low probability of one beaver being infected with SEOV. As no cases of disease have been recorded in beavers and it appears that accidental rodent spillover hosts do not usually experience clinical disease, the likelihood of disease associated with hantaviruses in translocated beavers and failure of the reintroduction is very low.

Risk estimation

There is a low likelihood of exposure of beavers to TATV and a very low likelihood of infection. There is a medium likelihood of exposure to SEOV and a low likelihood of infection with SEOV. The risk of morbidity and/or mortality is very low. The overall risk is VERY LOW.

Disease Risk Management

Risk evaluation

The level of risk associated with hantaviruses as either a source or destination hazard for beavers and other rodents is very low; the risk for humans is medium. Preventative measures for the risk management of hantaviruses as a destination and source hazard should be employed.

Risk management options

Hantavirus-associated disease should be considered as a differential in any sick beaver or other rodent examined during reintroduction. Detailed pathological examination should be carried out of beavers found dead during and after translocation and samples collected for diagnosis of hantaviral disease dependent on the pathological signs. Retrospective PCR testing of stored beaver tissue samples for hantavirus antigen or a pooled microarray for viral RNA as well as convenience blood sampling for serological conversion would be valuable to improve our understanding of hantavirus prevalence in beavers.

As hantaviruses can cause morbidity and mortality in humans, staff and volunteers working with beavers during reintroduction or post-release health surveillance should be reminded of the zoonotic risks and of the need to deploy good hygiene practices. Specifically, the wearing of masks to reduce the risk of aerosol inhalation when handling beavers is recommended.

Appendix 2 Disease Risk Analysis for the Population Hazard SARS-CoV-2

SARS-CoV-2 is the name given to the newly evolved coronavirus which at the time of writing is responsible for a global pandemic of severe acute respiratory syndrome (SARS), known as Covid-19, in humans (Gorbalenya et al., 2020). The virus belongs to the Betacoronavirus genus within the Coronaviridae family (de Groot et al., 2012; Masters, 2006). Coronaviruses are enveloped RNA viruses which cause numerous diseases across mammalian and avian species and have the largest genomes among all RNA viruses (de Groot et al., 2012; Masters, 2006). SARS-CoV-2 is a close relative of the human and bat severe acute respiratory syndrome coronaviruses (SARS-CoVs) which have given rise to several outbreaks of disease in people over the past 20 years (Gorbalenya et al., 2020; R. Lu et al., 2020; Wassenaar and Zou, 2020).

Justification of Hazard Status

Although some coronaviruses are host specific, others are found in a range of hosts (Drexler, Corman and Drosten, 2014). It appears that SARS-CoV-2 is likely to infect and replicate in numerous mammalian species other than humans and there is growing evidence to support its role as an anthropozoonosis, which we review here. Closely related coronaviruses to SARS-CoV-2 have been found to replicate in several free-living wild animal species. SARS-CoV-like viruses have been isolated from Himalayan palm civets (Paradoxurus hermaphroditus) which have been shown experimentally to be susceptible to disease from two separate virus isolates (Guan et al., 2003; Wu et al., 2005; Shi and Hu, 2008). Evidence of infection with SARS-CoV has also been detected in raccoon dogs (Nyctereutes procyonoides) and numerous bat species (Rhinolophus spp.) although clinical disease was not reported (Guan et al., 2003; Li et al., 2005; Cheng et al., 2007; Wassenaar and Zou, 2020). These studies provide evidence that free-living wild animal species could be infected with the closely related SARS-CoV-2 and may be at risk of clinical disease as a result.

There have been numerous reports to suggest that the SARS-CoV-2 pandemic originated from free-living wild animal sources, as is thought to be true for 60-70% of emerging diseases (Schlottau et al., 2020; Shi et al., 2020). Several preliminary reports have highlighted the ability of SARS-CoV-2 to infect 18 non-human mammalian hosts (Common et al., 2021): domestic cats (Felis catus), domestic dogs (Canis familiaris), racoon dogs, transgenic house mice (Mus musculus), North American deer mice (Peromyscus maniculatus), domestic ferrets (Mustela putorius furo), American mink (Neovsion vison), Egyptian fruit bats (Rousettus aegyptiacus), Syrian hamsters (Mesocricetus auratus), Malayan tigers (Panthera tigris jacksoni), Amur tigers (Panthera tigris altaica), African lions (Panthera leo), snow leopards (Panthera unicia), pumas (Puma concolor), rhesus macaques (Macaca mulatta), long-tailed macaques (Macaca fascicularis), African green monkeys (Chlorocebus aethiops) and common marmosets (Callithrix jacchus) (Bao et al., 2020; Chan et al., 2020; Deng et al., 2020; Goumenou, Spandidos and Tsatsakis, 2020; Lu et al., 2020; McAloose et al., 2020; ProMed International Society for Infectious

Diseases., 2020a, 2020b; Schlottau et al., 2020; Shi et al., 2020; Wang et al., 2020; World Organisation for Animal Health (OIE) (2020); Zhang et al., 2020). In 16 of these mammalian species (racoon dogs, Malayan tigers, Amur tigers, African lions, snow leopards, pumas, domestic cats, Syrian hamsters, North American deer mice, American mink, domestic ferrets, transgenic house mice, rhesus macaques, long-tailed macaques, common marmosets and African green monkeys), infection has been associated with disease (Bao et al. 2020; Chan et al. 2020; Freuling et al. 2020; ProMed International Society for Infectious Diseases., 2020b; Schlottau et al. 2020; Shi et al. 2020; World Organisation for Animal Health (OIE) (2020). Domestic pigs (Sus scrofa domesticus), domestic chickens (Gallus gallus domesticus) and domestic ducks (Anas platyrhynchos) are not thought to be susceptible to infection with SARS-CoV-2 (Schlottau et al., 2020; Shi et al., 2020).

The virus has been shown to replicate effectively in the upper respiratory tract of ferrets (Shi et al., 2020). Two ferrets in the study developed fever and loss of appetite 10 to 12 days after experimental inoculation with the virus. Post-mortem examination of these animals showed evidence of lymphoplasmacytic perivasculitis and vasculitis increased numbers of type II pneumocytes, macrophages, and neutrophils in the 24 alveolar septa and alveolar lumen, and mild peribronchitis in the lungs, suggesting that ferrets are susceptible to the clinical disease associated with SARS-CoV-2. Furthermore, SARS-CoV-2 outbreaks have now been confirmed in over 20 separate American mink farms in the Netherlands, Denmark and Spain (infocusvj.org 2020). Those reports which are published to date suggest that the outbreaks occurred after an infected worker exposed the mink to SARS-CoV-2 (Molenaar et al., 2020; Oreshkova et al., 2020). In the Netherlands, Oreshkova et al (2020) reported that an increased mortality rate was noticed in two separate farms: 1.2% at one, and 2.4% at the other, which are double and triple the expected mortality rate of 0.6% respectively (Oreshkova et al., 2020). Respiratory signs of watery nasal discharge were noted, with some animals exhibiting severe respiratory distress. Eighteen deceased animals from each farm were submitted for post-mortem examination; all 36 animals tested positive for SARS-CoV-2 using qPCR on throat swab, and 34 out of 36 on rectal swab. Twenty eight out of 36 (77.8%) had macroscopic evidence of interstitial pneumonia. In seven of these cases, histopathology of lungs was undertaken and severe diffuse interstitial pneumonia with hyperaemia and alveolar damage was found. In both of these farms, clinical signs of Covid-19 were present and SARS-CoV-2 confirmed in workers before mink, suggesting exposure of the mink by infected humans followed by dissemination among the mink. Transmission of SARS-CoV-2 between mink by direct contact is highly unlikely as each was housed with an impermeable barrier separating them from other animals, suggesting that indirect transmission through fomites, dust or droplets occurred. Inhalable dust was collected at three locations in each farm and tested for SARS-CoV-2 in three/six samples (1/3 at one farm and 2/3 at another) suggesting this as a likely transmission route (Oreshkova et al. 2020).

Schlottau et al. (2020) also experimentally inoculated nine fruit bats intranasally with SARS-CoV2, which resulted in transient respiratory tract infection. Virus replication was detectable in the nasal epithelium, trachea, lung and lung associated lymphatic tissue, and

infectious virus was isolated from the nasal epithelium and trachea of one animal after four days. Viral DNA was also detected in the nasal epithelium of one out of three in-contact bats after 21 days post-contact, suggesting that transmission is possible within this species (Schlottau et al., 2020).

There is evidence to suggest that domestic cats are susceptible to Covid-19 disease. Shi et al. (2020) showed that the virus replicates effectively in cats and can transmit between them via respiratory droplets. Moreover, two juvenile cats in the same study which were experimentally inoculated with SARS-CoV-2 were found to have severe lesions in the nasal and tracheal mucosal epithelia and lungs, highlighting their susceptibility to the disease (Shi et al., 2020). This finding is supported by results of a preliminary study into populations of domestic cats in Wuhan, China. 102 serum samples were collected from domestic cats after the outbreak of Covid-19 in humans, and 14.7% (n=15/102) were positive for the receptor binding domain (RBD) of SARS-CoV-2 by indirect enzyme linked immunosorbent assay (ELISA), suggesting that SARS-CoV-2 infected the cat population in Wuhan during the outbreak (Zhang et al., 2020). There are also several case reports of owned domestic cats testing positive for SARS-CoV-2, for example a case in Belgium, a case in Hong Kong, and two cases in the USA (News.gov.hk, 2020; ProMed International Society for Infectious Diseases, 2020c; USDA Animal and Plant Health Inspection Service, 2020). These feline cases are of further concern when considered alongside the reports of captive Malayan tiger and African lion from which duplicate nasal and oropharyngeal swabs tested positive on qPCR for SARS-CoV-2 in the USA (Calle, 2020; World Organisation for Animal Health (OIE), 2020). The animals had shown mild respiratory disease signs after contact with an infected keeper (Calle, 2020). Other reports of disease in large cats have since been reported (McAloose et al., 2020; Wang et al., 2020).

Since the Covid-19 outbreak was first reported, four domestic dogs have tested positive for SARS-CoV-2, and all had been in contact with an infected owner. None of the dogs showed signs of clinical disease, and although one dog died during the infection period, it was 17 years old and had multiple underlying diseases which were attributed as the cause of death rather than Covid-19 (Goumenou et al., 2020). Over 3,500 dogs, cats and horses (Equus caballus) showing respiratory disease (species numbers not reported) were screened for SARS-Cov-2 by IDEXX laboratories in South Korea in February and March 2020 and none were found to 25 be positive (IDEXX, 2020). This suggests that even if it is possible for them to become infected, occurrences are likely to be rare, given the 7,755 human patients with confirmed COVID-19 in Korea as of the 13th March 2020 (Covid-19 National Emergency Response Center, 2020).

It has been shown that entry of SARS-CoV-2 to host cells requires binding of the viral spike protein (S) to the SARS-CoV receptor human angiotensin converting enzyme 2 (hACE2) (Hoffmann et al., 2020), as is the case for SARS-CoV (Li et al., 2003; Kuba et al., 2005). hACE2 transgenic mice have been used as a disease model and compared to wild type mice (Bao et al., 2020). When intranasally inoculated with SARS-CoV-2, hACE2 transgenic mice show clinical signs of weight loss along with multiple histopathological changes including interstitial pneumonia. Viral RNA was detected in the lungs of transgenic mice by quantitative PCR at one, three, five and seven days after inoculation

but never in controls or wild-type mice. Infectious SARS-CoV-2 was isolated from inoculated transgenic mice, but never from wild-type mice or controls (Bao et al., 2020). This study highlights the importance of the hACE2 enzyme for entry of SARS-CoV-2 into host cells, leading to infection.

A preliminary study by Chan et al. (2020) investigated the genetic components of several mammalian species with the aim to identify an appropriate animal disease model for SARS-CoV-2. They found that that rhesus macaque ACE2 is 100% identical to human ACE2 at the interface region. Syrian hamster and common marmoset ACE2 proteins are were also found to be highly similar to human ACE2, each differing by only 3-4 mutations. Syrian hamsters were therefore identified as a possible disease model. In the experimental section of their study, Syrian hamsters were consistently infected with SARS-CoV-2 after nasal inoculation. Infected animals displayed a range of clinical signs including rapid breathing and weight loss. Histopathological changes two days after experimental inoculation included diffuse alveolar destruction and protein-rich fluid exudate, mononuclear cell infiltration, and alveolar collapse with haemorrhage. Bronchiolar lumens were filled with cell debris and epithelial cell swelling, focal cilia loss, and mononuclear cell infiltration into the epithelium and lamina propria was noted in the trachea. Histopathological respiratory tract changes appeared to peak around seven days post inoculation, with an increase in pulmonary cellularity and lung consolidation. After 14 days, only mild pulmonary congestion and inflammation were still detectable and gas exchange structures were restored to normal. Moreover, experimentally infected hamsters consistently infected naïve hamsters housed within the same cage, resulting in similar clinical signs (Chan et al., 2020). This study provides evidence that hamster ACE2 can bind with SARS- CoV-2 S receptor enabling cell entry and infection. Another study by Griffin et al (2020) showed experimentally that North American deer mice, closely related to Syrian hamsters, are also susceptible to infection with SARS-CoV-2. Although none of the mice in this study showed outward signs of disease (other than appearing ‘ruffled), post-mortem signs included occasional discrete foci of interstitial pneumonia and inflammatory signs on histopathology. Moreover, direct contact between infected and naïve mice consistently resulted in direct transmission to and infection of the naïve animals.

It is likely that species susceptibility to SARS-CoV-2 is intrinsically linked to the similarity of their ACE2 gene to that of human ACE2. Although this has not been investigated in Eurasian beavers, it is feasible that they may be susceptible to SARS-CoV-2 given that other rodent species, namely Syrian hamsters, have an ACE2 gene similar enough to human ACE2 to allow infection. ACE2 has been sequenced in Ords kangaroo rat (Dipodomys ordii), a closely related species to the beaver (Doronina et al., 2017), but not in any members of the Castoridae family (National Centre for Biotechnological Information, 2020). The relatedness of the kangaroo rat ACE2 to human ACE2 has also not been analysed. Therefore, it is not possible at this stage to determine whether beavers are susceptible to SARS-CoV-2. As far as we are aware, to date there have been no coronaviruses isolated from, or detected in, beavers nor have there been any coronavirus serological studies showing positive results in beavers. Guan et al., (2003) tested numerous species from a wet market in China for coronavirus using PCR during the

SARS-CoV outbreak, including three beavers, none of which were positive despite several other animals from different species from the same market testing positive. 26 Nevertheless, the limited available research means that we cannot rule out the possibility that beavers are susceptible to SARS-CoV-2.

Cases of SARS-CoV-2 infection have been reported in humans throughout the translocation pathway, including over 45 000 confirmed cases in the UK, and over 5000 confirmed cases in Norway as of 6th April 2020. The evidence to date shows that at least seven mammal species, including three rodent species, appear to be susceptible to disease associated with SARS-CoV-2. There has been no research specifically into the epidemiology of the virus in beavers, however SARS-CoV-2 is present both at the source and destination and therefore may represent a population hazard to reintroduced beavers.

Risk Assessment

Exposure assessment

Human exposure

Human exposure is likely to occur through direct contact with other humans, aerosol droplets in the air, or contact with contaminated surfaces (Kampf et al., 2020; Rothan and Byrareddy, 2020). The probability of human exposure to SARS-CoV-2 is medium. Human infection is thought to occur through contact of viral particles with exposed mucous membranes including the eyes, nose and mouth (Lu et al., 2020; Zheng, 2020). Faecal-oral transmission may also be possible (Xiao et al., 2020; Yeo et al., 2020; Zheng, 2020). The probability of infection after exposure is high.

It is thought that transmission of SARS-CoV-2 between humans occurs primarily via direct contact or through aerosol droplets spread by coughing or sneezing from an infected individual (Kampf et al., 2020; Rothan and Byrareddy, 2020), as is the case for other members of the Coronaviridae family (de Groot et al., 2012). Viral RNA has been detected in nasal washes of ferrets inoculated with the virus, as well as in several upper respiratory tract structures of inoculated and exposed cats (Shi et al., 2020). SARS-CoV-2 has also been detected in faeces of humans, a Malayan tiger and an African lion and is thought to be present in the faeces of bats (Calle, 2020; Holshue et al., 2020; Wassenaar and Zou, 2020) therefore faecal-oral transmission may also be possible, as for other closely related coronaviruses (Yeo et al., 2020). Rectal swabs taken from experimentally inoculated ferrets tested positive for viral RNA, though at lower levels than nasal washes. Infectious virus was not detected in any rectal swabs. In the same study, rectal swabs from experimentally inoculated beagles also tested positive for viral RNA (Shi et al., 2020). The basic reproduction number, R0, of SARS-CoV-2 in humans has been estimated to be 2 (Li et al., 2020; Liu et al., 2020a). The probability of dissemination through the human population is high.

Beaver exposure

During translocation of beavers, there are several opportunities for beavers to be exposed to SARS-CoV-2, mainly through direct contact with infected humans or contact with surfaces contaminated by infected humans. Coronaviruses have been shown to persist on inanimate surfaces for up to nine days and, at low temperatures, persistence can be as long as 28 days (Ijaz et aI., 1985; Kampf et al., 2020). Exposure through contact with infected surfaces could occur in beavers, as can occur for humans (Kampf et aI., 2020). Throughout the translocation pathway, beavers could be exposed at capture, during the quarantine period in captivity, during transport and at release. There is a medium probability that beavers will be exposed to SARS-Cov-2 during the translocation process.

There is no evidence to suggest that if beavers are exposed, they will become infected, but two other rodent species have been infected after experimental intranasal inoculation, and the lack of research in this area means the eventuality of beavers becoming infected cannot be ruled out. There is a medium likelihood that beavers will become infected with SARS-CoV-2 if exposed. The probability of the virus being disseminated amongst the reintroduced beaver population is medium, since rodent to rodent transmission has been shown for Syrian hamsters. Animal to animal transmission has also been shown for domestic cats and ferrets.

Consequence assessment

There is a low likelihood of beavers being infected at the reintroduction site.

The pathogenesis of SARS-CoV-2 in other rodents, particularly free-living wild rodents is unclear, although the literature so far suggests that severe disease and death is unlikely to occur after exposure. Covid-19 disease has been shown to occur in one non-transgenic species of rodent infected with SARS-CoV-2 in the laboratory, the Syrian hamster (Chan et al., 2020). Wild-type house mice did not appear to be susceptible in a separate study (Bao et al., 2020), implying that susceptibility is likely to be variable among rodent species. No coronavirus has ever been detected in a beaver.

There is a low likelihood that beavers will be susceptible to clinical disease if infected. Clinical signs in infected Syrian hamsters were considerable but did not result in mortality. Responses in other susceptible species have been variable and the limited available research suggests that severity may vary on a case-by case basis. It has been hypothesised that higher infective doses may lead to increased disease severity in humans; human patients with severe clinical signs had higher nasal viral loads than those with mild clinical signs (Y. Liu, Yan, et al., 2020b). However, until experimental challenge studies are undertaken for SARS-CoV-2, this will remain speculative. At this stage we estimate that there is a low probability of severe disease and mortality in beavers if they were to become infected.

The likelihood of Covid-19 disease outbreak within the translocated beaver population as a result of exposure to SARS-CoV-2 and the failure of the translocation is low. The likelihood

of severe economic and environmental consequences as a result of this failed translocation is low.

Risk estimation

At the time of writing, (05 May 2020), the probability of exposure of humans is medium and probability of infection is high. There is a high probability of dissemination through the human population. There is a medium likelihood that beavers will be exposed to SARS-CoV-2 through contact with workers at different stages of the translocation process and a medium likelihood of infection in beavers at the reintroduction site. There is a medium likelihood of dissemination within the beaver population at the release site. The probability of an outbreak of disease in the beaver population and the failure of the translocation is low. The overall risk is estimated to be MEDIUM.

Disease Risk Management

Risk evaluation

Simple preventative measures are likely to reduce the risk of SARS-CoV-2 to translocated Eurasian beavers.

Risk management option

The most important preventative management measure would involve reducing the exposure of translocated beavers to SARS-CoV-2 through direct contact. Since the majority of naturally occurring animal cases have been thought to have occurred as a result of anthropozoonosis, it is important to prevent exposure of beavers to infected humans. Simple measures such as appropriate personal protective equipment (PPE) for personnel in contact with beavers is likely to reduce the probability of exposure.

Moreover, since SARS-CoV-2 is active for long time periods on inanimate surfaces, proper disinfection of traps, captive enclosures, food bowls and any other possible fomites is essential to reduce the probability of transmission between humans and beavers. It is important that this is followed at every stage of the translocation pathway, including initial trapping, transport, captivity and release. Disinfectants containing 0.1% sodium hypochlorite or 62-71% ethanol lead to effective inactivation of the virus and so would be appropriate (Kampf et al., 2020). Notwithstanding, all specific products should be analysed to ensure they are safe and licenced for use around animals.

Appendix 3 Disease Risk Analysis for the Carrier Hazard Leptospira species

Leptospires are globally distributed Gram-negative, spirochete bacteria belonging to the genus Leptospira that currently comprises about 20 species of varying pathogenicity and as many as 300 recognized serovars (Adler, 2015). Nomenclature is complex, comprising species, serogroup, serovar and strain (Levett, 2001). Infected mammals may shed leptospires in their urine with warmth and moisture favouring leptospire persistence in the environment (Birtles, 2012a). Leptospires have been shown experimentally to survive for up to several months in water at room temperature and for up to 7 weeks in soil (Levett, 2001). Cases reportedly peak in summer following periods of hot, dry weather (ibid.). Infection is from contaminated watercourses via mucus membranes or skin lesions or, less commonly, by direct contact with infected animals' urine (Evangelista and Coburn, 2010).

Justification of Hazard Status

Different Leptospira species and serovars have evolved to exploit different mammal species as reservoir hosts and it has been shown that almost every mammal species can serve as a carrier (Adler and de la Pena Moctezuma, 2010). Leptospires do not survive well in acid conditions so animals producing alkaline urine such as herbivores are more prolific shedders (ibid.). Rodents, in particular, rats, are considered among the most important reservoirs of some Leptospira species, including zoonotic serovars. Other mammals in environments where rats are believed to be the main reservoir tend to harbour the same Leptospira serovar but it is not known whether they also play a reservoir role or are accidental (incidental) hosts (ibid.). Aquatic rodents, including the muskrat (Ondatra zibethicus), coypu (Myocastor coypus) and water vole (Arvicola amphibius) have been shown to harbour leptospires (Aviat et al., 2009; Meyer-Scholl et al., 2012; Gelling et al., 2015). It is recognized that an animal can be a reservoir host for one serovar but susceptible to infection and disease as an accidental host from another (Levett, 2001).

Reservoir hosts are usually asymptomatically and chronically infected and may shed bacteria for extended periods (Adler and de la Pena Moctezuma, 2010). However, chronic disease in reservoir hosts causing interstitial nephritis, renal fibrosis and failure has been reported in wild rats and experimentally induced in rats inoculated with L. interrogans serovar Copenhageni (Monahan et al., 2009). Additionally, severe disease has been experimentally induced in immunocompromised mice inoculated with L. interrogans serovar Icterohaemorrhagiae (Evangelista and Coburn, 2010). As a result, it would appear that animals within reservoir host groups may under certain circumstances experience either chronic or acute leptospirosis following infection with Leptospira serovars that do not normally cause disease in the host species.

In humans, leptospirosis is an important emerging zoonotic disease of which the most severe form involves multi-system organ complications, known commonly as Weil's Disease or Syndrome (Evangelista and Coburn, 2010). Susceptibility and severity of disease is believed to vary with infective dose, serovar, strain, host species and individual

MHC variation (Monahan et al., 2009). Infection of humans can result in a range of symptoms from mild flu-like illness to jaundice, pulmonary haemorrhage and kidney failure with occasional reports of aseptic meningitis and myocarditis (Schreiber et al., 2015). Histopathological examination of beavers, infected with pathogenic strains of Leptospira species found dead, recorded lung haemorrhage as the most common lesion, consistent with fatal cases in humans (Marreros et al., 2017).

Leptospira species are ubiquitous in both potential source and destination sites. As translocation is a known stressor (Dickens et al., 2010), beavers, either as accidental or reservoir hosts, may therefore be susceptible to disease when immunocompromised by stress and so Leptospira species should be considered as a carrier hazard for the translocation of beavers.

Risk Assessment

Release assessment

Beavers at the source site(s) may be exposed to and infected by Leptospira species in the environment via mucous membranes or skin abrasions as leptospires can survive in water for several months and shedding by infected reservoir hosts is prolonged.

There is scant evidence for Leptospira species in Norway. Akerstedt et al. (2010) reported a prevalence of 9.9% in red foxes (Vulpes vulpes) tested by MAT serology for L. interrogans serovar Icterohaemorrhagiae between 1994 and 2005 (n=20/202). However, 0/52 Norwegian beavers tested by PCR of kidney tissue (Girling et al., 2019c) were positive for leptospiral antigen and we are not aware of any other studies finding evidence of leptospiral infection in beavers in Norway. 9/30 beavers trapped in Norway for release in Scotland as part of the Knapdale trial tested positive on MAT (ibid.) but this was towards the end of their 6 months rabies quarantine in the UK and so infection in the UK cannot be ruled out as none of the serovars identified was novel to the UK (Goodman et al., 2012). Of these beavers, four were positive for L. interrogans serovar Icterohaemorrhagiae and nine for L. interrogans serovar Copenhageni. On retrapping, one beaver remained seropositive to L. interrogans serovar Icterohaemorrhagiae.

0/25 beavers trapped in the Tayside region of Scotland (origin unknown) tested positive on MAT serology or urine or kidney PCR (Girling et al., 2019c). Additionally, Leptospira species were not isolated from any of the 18 beavers examined post-mortem in the UK to date that have been reported to us. 3/6 beavers trapped in Devon as part of the River Otter trial (origin unknown, presumed Bavaria) were positive on MAT but the serovars were all known to be present in the UK (ibid.). Similarly, 2/9 Bavarian beavers (wild-caught or captive-bred) were positive by kidney PCR or MAT but to serovars already present in the UK (ibid.). None of these beavers was positive for L. interrogans serovar Icterohaemorrhagiae or L. interrogans serovar Copenhageni.

As animals infected with leptospires have been found in potential source sites and Leptospira species are considered to be ubiquitous, beavers at the source site(s) are

therefore highly likely to be exposed to and infected as beavers have been shown to be susceptible to infection. There is therefore a high likelihood of an infected beaver being translocated and released.

Exposure assessment

As infected beavers may shed leptospires for prolonged periods and leptospires are able to survive for prolonged periods in the environment, there is a high probability of beavers and other mammals being exposed to Leptospira species at the destination site(s). Many mammal species are susceptible to infection and those that are already or become infected have the potential to become long term carriers and to contribute to the maintenance of the agent at the destination site(s) by shedding leptospires in their urine into water and adjacent habitat. There is therefore a high likelihood that mammals at the destination site(s) will disseminate Leptospira species to other mammals.

Consequence assessment

There have been 21 reported cases of leptospiral infection associated with mortality in Eurasian beavers in mainland Europe (Nolet et al., 1997; Woll et al., 2012; Giovannini et al., 2012; Marreros et al., 2017). The serovar was not reported in every case but has included five associated with infection with L. interrogans serovar Icterohaemorrhagiae and five with L. interrogans serovar Copenhageni (Marreros et al., 2017; Nolet et al., 1997). However, leptospiral infection, including of L. interrogans serovar Icterohaemorrhagiae, has been found on serology in Eurasian beavers without clinical signs (Goodman et al., 2017; Girling et al., 2019c). Girling et al. (2019c) concluded that previously reported mortalities may have been associated with other factors such as concurrent infection with other parasites.

Marreros et al. (2017) reviewed the histopathology of lung and kidney tissue and serology from 13 free-living beavers found dead in Switzerland between 2010 and 2014. The authors noted multifocal haemorrhages with variable levels of associated inflammation on histopathology of lung samples from all 13 beavers and interstitial fibrosis in renal tissue from two thirds (n=8/12) of the beavers. PCR testing confirmed the presence of leptospiral antigen in nine of the 11 beavers tested with five beavers PCR-positive in both lung and kidney tissue. Sequencing identified genotypes of leptospiral strains in the L. interrogans serovar Icterohaemorrhagiae and L. interrogans serovar Copenhageni serovars (serogroup Icterohaemorrhagiae). Ten of the 11 beavers for which blood samples were available were positive on MAT (titre => 1/100) for leptospiral antibodies with the highest titres (1/3200) to serovars Icterohaemorrhagiae, Copenhageni and Verdun (serogroup Icterohaemorrhagiae). All but one of the beavers was in poor body condition and leptospirosis was cited as the cause of morbidity and mortality in all cases.

The histopathology samples from beaver lung and kidney tissues examined by Marreros et al. (2017) exhibited features associated with both acute and chronic leptospiral infection. Low levels of inflammatory infiltrate in lung tissue, seen in accidental hosts such as humans or dogs experiencing acute leptospirosis (ibid.), were noted in some sections while interstitial renal fibrosis, associated with chronic rather than acute leptospirosis

(Monahan et al., 2009) were noted in sections from other beavers. Marreros et al. (2017) therefore concluded that beavers are capable of being both acutely and chronically infected i.e. can act as both accidental and reservoir hosts of pathogenic leptospires. As both forms of infection, acute and chronic, have been variably observed following infection with L. interrogans serovar Icterohaemorrhagiae in beavers, it seems probable that immunocompetence to leptospiral infection is similarly variable in the species.

Immunocompetent beavers infected with pathogenic Leptospira species would be expected to mount a humoral antibody-mediated response to infection and recover quickly without experiencing clinical disease. However, the observation of signs of chronic infection such as bacterial colonization of renal tubules and interstitial renal fibrosis in beavers suggest that some individuals may become chronically infected with the potential to become reservoir hosts.

Additionally, since translocated beavers will be under stress, there is a high likelihood that infected beavers will experience clinical disease, leptospirosis. Acute leptospirosis associated with the stress of translocation has been previously observed in beavers (Nolet et al., 1997). Of 58 beavers translocated from Germany to the Netherlands, Nolet et al. (1997) reported that three beavers were found dead in association with leptospiral infection between 24 and 31 days post-release. Of the 58 beavers released, 57 were released in the autumn and 43 had undergone general anaesthesia shortly prior to release for the intra-peritoneal implementation of radio-transmitters. The stress of trapping, handling and captivity could therefore increase the susceptibility of beavers to disease and increase the likelihood of morbidity and mortality from leptospirosis. However, once precipitating stressors are removed, it is probable that any infected beavers will remain as asymptomatic carriers and so the risk of severe disease is low and the overall risk of reintroduction failure is low.

Risk estimation

There is a high probability of beavers being exposed to Leptospira species at either the source or destination site and a high likelihood of infection. The risk of dissemination to other animals at the destination site(s) is high. There is a high probability that the stress of translocation may precipitate acute disease in infected beavers and result in the failure of the translocation. The overall risk from disease caused by Leptospira species is HIGH.

Risk Management

Risk evaluation

Based on the risk assessment above, preventative measures should be employed to reduce the risks from Leptospira species as a carrier hazard.

Risk management options

Diagnosis of exposure is usually by micro-agglutination test (MAT) serology, identifying host antibodies to specific leptospiral serovars or serogroups. Where antibodies are

detectable on MAT, a minimum titre of 1/100 is usually regarded as indicative of infection although, given the specificity of the MAT, lower levels may be interpreted as confirming exposure (IOE 2018). A titre of over 1/400, consistent with a four-fold increase, is regarded as indicative of current or recent infection (Girling et al., 2019c).

However, it may be up to three to four weeks before a positive test is returned following infection (Schreiber et al., 2015) so acute infection may be missed on serology. Additionally, host-adapted strains appear to trigger only minimal serological response in reservoir (carrier) hosts compared to accidental hosts (Shearer et al., 2014) and bacteraemia may be transient (OIE, 2018) so serology is not a reliable means of identifying whether a host is actively shedding leptospires and so potentially infectious (Aviat et al., 2009). Serology is therefore likely to be of limited value in identifying infected beavers and infected beavers may be healthy and not necessarily of risk to other beavers or mammals.

Isolation of bacteria by urine culture or PCR of urine is a preferred method of identifying carriers but leptospires are fastidious and incubation is lengthy, potentially up to 30 weeks (Birtles, 2012a) and leptospire shedding may be intermittent, so carriers may be missed on testing (ibid.). If pathological findings are suggestive of leptospirosis, PCR testing of kidney tissue for leptospiral nucleic acid at post-mortem, followed by sequencing, in conjunction with histopathology, is currently regarded as the gold-standard method of identifying leptospiral-associated disease and should be considered as part of routine post-mortem examination of all beavers found dead or euthanized on welfare grounds if signs suggest leptospirosis is a differential.

Measures should be undertaken to reduce stress in beavers undergoing translocation. Specifically, handling, invasive testing, journey times and human presence, and scent, at capture and release sites should all be kept to the lowest practical level. General anaesthesia for clinical examination or implantation of tracking devices is not recommended due to the associated stress of additional handling and confinement.

Appendix 4 Disease Risk Analysis for the Source Hazard Francisella tularensis

Justification of Hazard Status

Francisella tularensis is a small, gram negative coccobacillus which is one of five species within the Francisella genus, family Francisellaceae. It is the aetiological agent of tularemia, an infectious and zoonotic septicaemic disease. Tularemia was first described in 1911 in rodents exhibiting plague-like clinical signs (McCoy, 1911) and the bacteria later identified after isolation from Californian ground squirrels (Otospermophilus beecheyi) (McCoy and Chapin, 1912). F. tularensis has since been isolated from over 250 species and is considered to have the broadest host range of all zoonotic agents (Gyuranecz, 2012; Mörner, 1992). Eurasian beavers have been implicated as reservoir hosts of F. tularensis and one case of clinical disease has been reported (Morner et al., 1988a; Mörner and Sandstedt, 1983; Schulze et al., 2016). Tularemia is a complex disease, and many aspects of the epidemiology are poorly understood, including transmission cycles and reservoir hosts (Hestvik et al., 2015). Mammals within the orders Lagomorpha and Rodentia are thought to be particularly important within the parasite's lifecycles (Gyuranecz, 2012).

Four subspecies of F. tularensis are currently recognised: F. tularensis subsp. tularensis, F. tularensis subsp. holarctica, F. tularensis subsp. mediasiatica and F. tularensis subsp. novicida. The moderately virulent F. tularensis subsp. holarctica is the causative agent of disease in Europe (Gyuranecz, 2012). F. tularensis subsp. holarctica is associated with aquatic ecosystems. Aquatic mammals, including Eurasian beavers, have been implicated as reservoirs of the bacterium in countries where the disease is endemic (Mörner and Sandstedt, 1983). F. tularensis subsp. holarctica can also be transmitted by haematophagous arthropods, including mosquitos (Aedes aegypti) and ticks (Ixodae species) (Akimana and Kwaik, 2011; Gyuranecz, 2012; Maurin and Gyuranecz, 2016; Petersen et al., 2009; Thelaus et al., 2014; Výrosteková, 1993). Mosquitoes become infected through the aquatic cycle during their larval stages, but are not considered to be true reservoirs as transovarial transmission has not been shown, suggesting that the infection will die with the mosquito (Petersen et al., 2009). The tick Dermacentor reticulatus is thought to be a true reservoir of F. tularensis subsp. holarctica and transmits the parasite between mammals in Central Europe through a separate terrestrial cycle (Keim et al., 2007).

Francisella tularensis is widespread across continental Europe and its current geographic range encompasses Czech Republic, Finland, France, Germany, Liechtenstein, Netherlands, Norway (Personal communication, Turid Vikøren, 11th February 2020), Sweden and Switzerland. It is also suspected to be present in Italy, Denmark and Russia, and has previously been reported in Austria, Belgium, Bulgaria, Hungary and Poland, although is currently absent in these areas. The bacterium is currently considered to be absent from the United Kingdom (OIE, 2020).

Risk Assessment

Release assessment

There are two known transmission cycles of F. tularensis: the aquatic and terrestrial cycles. F. tularensis is highly adaptable to a wide range of arthropod vectors (Petersen et al., 2009), and it is possible that an infected arthropod could be released at the destination alongside translocated beavers from Norway. Prevalence of F. tularensis within the European tick population has been reported as between 0 and 3% (Hubálek and Halouzka, 1997).

Hare and rodent species, such as lemmings (Lemmus lemmus), are important hosts and have also been implicated as reservoir species in previous outbreaks (Berdal et al., 1996; Larssen et al., 2011; Morner et al., 1988b; Nordstoga et al., 2014). The bacterium can be transmitted directly through environmental contamination with bodily discharges such as faeces and urine, leading to alimentary or aerogenous infection (Friend, 2006; Gyuranecz, 2012; Gyuranecz et al., 2010; Reintjes et al., 2002). These routes of infection are particularly important during winter, when arthropod density decreases (Morner et al., 1988b).

In the aquatic cycle, aquatic mammals including voles (Microtus species), muskrats and beavers are thought to be important hosts and contribute to environmental contamination through shedding of live bacteria in secretions (Mörner and Sandstedt, 1983; Schulze et al., 2016). Contamination from carcasses can also occur (Gyuranecz, 2012; Schulze et al., 2016). F. tularensis subsp. holarctica has been detected in water and sediment samples from areas in which tularemia is endemic in both outbreak and non-outbreak years. This indicates that environmental persistence may contribute to the complex epidemiology of the disease (Berdal et al., 1996; Broman et al., 2011).

F. tularensis has not been found in beavers in Great Britain during testing in the River Otter Beaver Trial and monitoring of the Scottish populations at Knapdale and Tayside. Serum Polymerase Chain Reaction (PCR) and Serum Enzyme Linked Immunosorbent Assay (ELISA) was used to test 29 beavers in Knapdale, with no positive results noted (Gaywood et al., 2015; Goodman, 2014). At Tayside, PCR of blood was negative for F. tularensis in all 17 live trapped animals, as well as PCR of blood or tissue samples of six carcasses submitted for post-mortem examination. Serum PCR was performed on five live-trapped animals as part of the River Otter Beaver Trial, and all were negative for F. tularensis (Campbell-Palmer and Girling, 2019).

Cases of tularemia in Norway have been sporadic in humans, wildlife and domestic species over the past century but showed an increase in 2019 (Agren et al 2019). 116 human cases of tularemia were reported in Norway between 1926 and 1972 along with sporadic identification of F. tularensis in lemmings and Ixodes species of tick (Pearson, 1975; Výrosteková, 1993), while an additional 179 cases of disease in humans was reported in 2019 (Agren et al 2019). A report published in 2014 described a case of tularemia in a domestic dog in Norway after ingestion of an infected mountain hare (Lepus timidus), suggesting an alternate source of infection within the country (Larssen et al.,

2011). More recent outbreaks in humans and domestic dogs were linked to increased free-living lemming populations and subsequent contamination of drinking water. Lemmings are now widely considered to be the main reservoir in Norway (Berdal et al., 1996; Larssen et al., 2011; Nordstoga et al., 2014). Human tularemia outbreaks have been associated with increased population numbers of free-living rodent reservoirs (Larssen et al., 2011) and with insect bites (Agren et al 2019).

There is a high likelihood that Eurasian beavers in Norway will have been exposed to F. tularensis through contaminated water sources during these outbreak periods. A recent report of tularemia diagnosis in 16 hares (Lepus species) from the Eastern part of Norway in 2019 (Personal communication, Turid Vikøren, 11 February 2020) confirms that the disease is currently occurring within the country. It is possible that free-living beavers in Norway were exposed to F. tularensis through environmental contamination at this time. There have been no known surveys of F. tularensis infection or tularemia in free-living Eurasian beavers in Norway, and it is therefore not possible to conclude that these animals have not been exposed and infected with F. tularensis over the last decade. It is also unclear for how long beavers shed the bacterium after infection and whether they may become persistent shedders.

In the neighbouring Sweden, tularemia has been considered to be endemic in wildlife for the past decade and widely prevalent in domestic animal populations before this (OIE, 2020). The number of cases in humans showed a marked increase in 2019 (Agren et al 2019). Furthermore, exposure to the bacterium has been detected in free-living Eurasian beavers in Sweden using serological studies. Positive antibody titres were found in 21% (n= 23/110) of investigated beavers in one study (Morner and Sandstedt, 1982). The beaver is likely to be important in the epidemiology of tularemia in Scandinavia, and could act as a reservoir of F. tularensis, although the bacterium has never been isolated from this species in Sweden (Morner et al., 1988a; Mörner and Sandstedt, 1983; Tärnvik et al., 1996). In Sweden, several beaver populations are distributed close to the Norwegian border. Populations inhabit the areas surrounding waterways which breach this border, such as the river Klarälven (Hartman, 1995). There is a risk that Norwegian beavers have been exposed to F. tularensis through contact with Swedish beaver populations in these areas.

While it is known that beavers in parts of Europe, including Sweden, have been exposed to F. tularensis, there is a lack of evidence on the proportion infected and the persistence of infection. In other rodent species infection rates appear to be low. In one study, 547 small rodents were trapped in Finland and multiple samples tested using PCR. F. tularensis DNA was unequivocally detected in liver samples of only five field voles.

There is a medium likelihood that, at the time of translocation from Norway to England, beavers will be infected with F. tularensis. There is a low likelihood that free-living beavers in Great Britain and in enclosures are infected with F. tularensis because some beavers from these populations originate from geographic areas in which the parasite occurs.

Exposure assessment

There is a medium likelihood of exposure of mammals at the release site to F. tularensis. Eurasian beavers carrying the bacteria when translocated to England from Norway, or already residing within Great Britain and enclosures, could lead to contamination of water sources and exposure of susceptible species via this route. Alternatively, direct transmission through aerosol, gastrointestinal secretions or urine could lead to infection of susceptible rodents and lagomorphs at the destination. Stowaway infected arthropods translocated alongside the beavers from Norway may also transmit F. tularensis through feeding on animals at the release site. Once exposed, there is a high likelihood of infection of mammals at the release site and dissemination through these mammal populations.

There is a medium likelihood that arthropods within Great Britain will be exposed and infected with F. tularensis. If one infected translocated beaver is bacteraemic when released, arthropod vectors residing at the destination site could be exposed through feeding on this animal.

There is a low likelihood of human exposure to F. tularensis at the destination through contamination of water sources. Human to human transmission does not occur (Tärnvik et al., 1996; World Health Organisation, 2007), meaning that dissemination amongst the human population in the face of an outbreak would not occur. Once the source of infection is identified the outbreak would be self-limiting.

Consequence assessment

In humans, clinical signs of tularemia are variable, but when associated with contaminated water sources are commonly fever and pharyngitis (considered the ‘oropharyngeal form’). An ulceroglandular form can also occur as a result of insect bites. In general, disease as a result of F. tularensis subsp. holarctica in Europe is generally less severe than disease caused by F. tularensis subsp. tularensis in North America (Larssen et al., 2011). Clinical signs can be non-specific and so without appropriate testing it is not possible to distinguish tularemia from other septicaemic diseases (Nordstoga et al., 2014; Tärnvik et al., 1996). The disease course is thought to be dose-dependent, with individuals exposed to higher doses more likely to die acutely than to become chronic shedders (Ellis et al., 2002; Frederick and Stewart, 1975; Staples et al., 2006; World Health Organisation, 2007). To our knowledge, no cases of tularemia have been reported in humans working with beaver translocations. Several outbreaks of tularemia have occurred in Europe, including Norway, but appear to be sporadic and are associated with contaminated water sources as a result of increased populations of lemming reservoirs (Larssen et al., 2011). The likelihood of a tularemia outbreak in humans living downstream of beaver release sites is low. The likelihood of negative consequences to humans as a result of a disease outbreak, including severe clinical signs, is high.

Clinical signs of tularemia vary between other mammal species. Mountain hares in Sweden appear to die of acute disease with non-specific clinical signs. Post-mortem examination findings included pinpoint necrotic foci throughout abdominal organs (Morner et al., 1988b). A more chronic course has been reported in brown hares (Lepus

europaeus) in central Europe, although post-mortem examination findings are comparable to those in mountain hares (Gyuranecz et al., 2010). One case of tularemia in a Eurasian beaver has been reported in Germany, demonstrating the possibility of disease occurring in this species; findings post-mortem were comparable to those in other free-living species (Schulze et al., 2016).

The probability that one beaver translocated from Norway into Great Britain is infected is high, and from Great Britain, either free-living or in a fenced enclosure, into England is medium. Eurasian beavers are susceptible to tularemia, but the disease appears to be rare and only a single case has been reported, noted above. Those beavers exposed to F. tularensis and infected are not likely to show clinical signs and instead will act as reservoirs (Morner et al., 1988b; Mörner and Sandstedt, 1983). There is a very low likelihood of systemic disease leading to death in an infected beaver and of an outbreak in the translocated beaver population and of biological and economic consequences through failure of the reintroduction.

There is a low likelihood of cases of disease in humans in contact with contaminated water sources. Cases of tularemia in humans would be limited by the fact that human to human transmission is not thought to occur (Tärnvik et al., 1996; World Health Organisation, 2007). There is a very low likelihood of economic consequences as a result of increased resource requirement of trained staff including vets, doctors and government agency workers to manage cases of the disease (Tärnvik et al., 1996; World Health Organisation, 2007).

As far as we are aware, no autochthonous cases of tularemia have been diagnosed in Great Britain and the differing epidemiological risk factors between continental Europe and Great Britain underlying the absence of disease in Great Britain are uncertain. There is a low likelihood of disease outbreaks in exposed susceptible mammalian species, particularly from the orders Rodentia and Lagomorpha, including several endangered species including the already endangered hazel dormouse (Muscardinus avellanarius), water vole and red squirrel (Sciurus vulgaris).

Risk estimation

There is a medium likelihood that Eurasian beavers translocated from Norway will be infected with F. tularensis and a low likelihood that beavers translocated from Great Britain will be infected. There is a low likelihood that an infected arthropod vector will be translocated alongside the beavers. There is a medium probability of exposure and a high probability of infection of mammals at the destination and dissemination through mammal populations. There is a medium probability that arthropods at the destination will be exposed and infected with F. tularensis if an infected beaver is released. There is low likelihood of exposure of people and negligible likelihood of dissemination through the human population. There is a very low likelihood of a disease outbreak in the translocated beaver population and a low likelihood of a disease outbreak in other susceptible mammalian species. There is a low likelihood of sporadic disease in people. The overall risk is LOW.

Risk Management

Risk management options

The following serological tests are available for F. tularensis: microagglutination, indirect immunofluorescent assay or ELISA-type western blot assay (Hepburn and Simpson, 2008; Maurin and Gyuranecz, 2016; Tärnvik and Chu, 2007; World Health Organisation, 2007). PCR testing of secretions to detect active shedding is available (Sting et al., 2013). Both serological and PCR tests would be valuable for research purposes if possible and to modify the disease risk analysis in future years.

Treatment of all beavers with anti-parasitic agents prior to transport should be considered to avoid co-transport of arthropod vectors infected with F. tularensis to the destination site. If Norway is chosen as the source, investigations into the conservation status of native arthropods should be undertaken and consideration given to conserving these species.

Appendix 5 Disease Risk Analysis for the Carrier Hazards Yersinia enterocolitica and Yersinia pseudotuberculosis

The genus Yersinia comprises twelve species of Gram-negative coccobacilli (Martin et al., 2009) of which Y. enterocolitica and Y. pseudotuberculosis are associated with disease in mammals in Europe (Najdenski, 2012). Both Y. enterocolitica (YE) and Y. pseudotuberculosis (YP) consist of serotypes of varying pathogenicity associated with the disease, yersiniosis, in a wide range of species globally, particularly in northern Europe (ibid.).

Justification of Hazard Status

Both YE and YP are considered to be ubiquitous with numerous species of wild mammals, including rodents, and birds acting as subclinical carriers (ibid.). A study in Scandinavia found 8% (n=12/154) prevalence of YE in free-living small rodents (Kapperud, 1975). However, in both Sweden and Norway, domestic pigs are believed to be the primary reservoir of YE (Lindberg, 2018; Jorgensen et al., 2018). Additionally, both YE and YP have been confirmed in Sweden in a wide range of birds, including those known to migrate to the UK (Niskanen et al., 2003), for example the barnacle goose (Branta leucopsis).

In the UK, YE was isolated from faecal samples from free-living wild animals in Dorset, including the bank vole, between 1986 and 1989 (Healing and Greenwood, 1991) and YP from free-living birds and mammals including, prior to its extirpation, the coypu, mouse and field vole (Mair, 1973). Infection with either YE or YP has not been found to date in screening of free-living beavers (n = 65) in Great Britain (Campbell-Palmer et al., 2015b; Campbell-Palmer and Goodman, 2019; Goodman et al., 2014). However, a gravid female in good body condition was found to be infected with Y. frederikensii following re-release in Devon (Campbell-Palmer et al., 2018).

Susceptibility to yersiniosis probably varies from species to species but sporadic outbreaks of disease resulting in high mortality have been reported in a wide range of wildlife species (ibid.). Additionally, stressful conditions such as cold and wet weather, limited food availability, overcrowding and capture may precipitate clinically significant disease in sub-clinical carriers (Gasper and Watson, 2001). Disease incidence is reported to be higher in winter months (Najdenski, 2012).

Yersiniosis has been cited as the cause of deaths in Eurasian beavers, either in isolation or in combination with other diseases in three studies (Nolet et al., 1997; Platt-Samoraj, 2015; Stefen, 2018). For example, of 57 beavers translocated from Germany to the Netherlands between 1988 and 1994, four died with yersiniosis associated with either YE or YP, including one which had been vaccinated prior to translocation against YP, in the first three months following release (Nolet et al., 1997). Nolet et al. (1997) suggested that stress from territorial conflict and food shortages contributed to disease susceptibility in

these translocated beavers as they had all settled in habitats of poor quality compared to other translocated beavers.

A wild-caught beaver from Norway (M08K33) which died during quarantine in the UK with severe enteritis and focal hepatic necrosis was found to have an Escherichia coli bacteraemia; histopathology was reported to be suggestive of yersiniosis (Cranwell,2009c). It was suggested that suspected yersiniosis in this beaver (M08K33) and another (M08K20), might be a result of prolonged confinement in captivity (Cranwell, 2009a). The difficulty of monitoring the health and disease of beavers following translocation, either due to the difficulty of finding sick or dead wild animals (Wobeser, 2007) or the challenges of trapping free-living beavers (Campbell-Palmer et al., 2015a) suggests other cases of yersiniosis in beavers may have been missed.

As all translocations are associated with stress (Dickens et al., 2010), and stress precipitates reduced immunocompetence, and YE and YP are ubiquitous at the source and destination, translocated beavers will be predisposed to yersiniosis. Therefore, YE and YE should be considered as carrier hazards for the translocation of Eurasian beavers.

Risk Assessment

Release assessment

Both YE and YP are psychrophilic, able to survive and multiply at low temperatures (2-5°C), and capable of surviving for up to 20 days in water and 540 days in soil (Najdenski, 2012). Beavers at the source site will be exposed and infected primarily via the faeco-oral route via contaminated food or water. The likelihood of beavers being exposed to YE and YP at the source site(s) is estimated to be high because these bacteria are known to be ubiquitous and persistent for prolonged periods in the environment. In addition, sympatric species such as rodents and waterfowl are probable reservoirs (ibid). If exposed, there is a high likelihood that beavers will be infected because beavers are known to be susceptible to infection.

Exposure assessment

Mammals, including beavers, at the destination will be exposed to YE and YP through the faeco-oral route. Carriers of YE and YP are known to shed these bacteria for prolonged periods (Najdenski, 2012) and because YE and YP may survive for prolonged periods in the environment, there is a high probability of direct exposure at the destination site(s). Many mammal species are susceptible to infection and therefore there is a high likelihood that mammals at the destination will be infected.

There is a high likelihood that mammals at the destination will maintain and disseminate these agents at the destination site(s) by shedding infectious Yersinia bacteria in their faeces. In addition to faeco-oral transmission, venereal and transplacental routes are possible.

Consequence assessment

The clinical presentation of disease in mammals caused by both YE and YP may be similar (ibid.). Where YE is associated with acute disease, the signs are fulminating septicaemia and enteritis, leading to death within one to three days (ibid.). Chronic disease typically features necrotising enteritis resulting in weight loss, anorexia and lethargy amongst other clinical signs (ibid.).

There is a high probability that one translocated beaver becomes infected. Since translocated beavers will be under stress there is a high likelihood that they will be affected by yersiniosis (acute, subacute or chronic disease) as illustrated by reports of disease following translocation (Nolet et al., 1997). As Yersinia species are psychrophilic, there may also be recrudescence of latent infections during the winter months due to the stresses of cold and hunger, resulting in disease. Therefore, yersiniosis may occur weeks or months following translocation. There is a high probability of biological and economic consequences through failure of the translocation. However, since YE and YP are ubiquitous, the long term environmental and biological consequences are negligible.

Risk estimation

There is a high likelihood that released beavers will be exposed to, and infected with, YE or YP. The likelihood of exposure, infection and dissemination at the destination is high. There is a high probability that the stress of translocation may precipitate disease in infected beavers and lead to the failure of the translocation. The overall risk of disease in translocated beavers and failure of the translocation from YE- and YP-associated disease is therefore HIGH.

Risk Management

Risk evaluation

Based on the risk assessment above, preventative measures should be employed to reduce the risks from YE and YP.

Risk management options

Measures to reduce the stress from translocation are important. For example, efforts should be made to minimise stress from capture, transport and, in particular, reduce the need for repeated handling and the duration of transit. Consideration should also be given to the timing of releases, avoiding winter months when lower temperatures and food shortages may increase the risk from stressor-associated disease.

Diagnosis is usually by isolation of bacteria from faeces, throat swabs, mesenteric lymph nodes, peritoneal fluid or blood, with faecal culture the usual method in practice. However, this method is regarded as unreliable as positive cultures may only be achieved in the first two weeks of illness. As a consequence, cases of infection with Yersinia species may not always be detected.

Appendix 6 Disease Risk Analysis for the Carrier Hazard gram-negative enteric bacteria

Justification of hazard status

Gram negative enteric bacteria are found as part of the normal commensal flora in the digestive tracts of mammalian species; however, they may, under certain circumstances, act as opportunistic pathogens to cause intestinal and extra-intestinal disease (Kang et al., 2018). Two families are of concern: Enterobacteriaceae and Epsilonproteobacteria. Some genera such as Yersinia species, evaluated elsewhere in this report, Salmonella species, Shigella species and species such as Escherichia coli are considered to be important zoonoses, associated with severe morbidity and mortality (ibid.). Other genera of interest are: Klebsiella, Enterobacter, Citrobacter, Proteus, Serratia, Campylobacter and Helicobacter. Disease in the host animal may occur when gram negative bacteria either overgrow within the gastrointestinal tract or colonise a new body compartment (Melter and Castelhano, 2019). Survival of gram-negative enteric bacteria in the environment may be prolonged and up to several months for some species (Kramer et al., 2006) with direct or indirect infection of new hosts via the faecal-oral route or, occasionally, via mucous membranes (Gaffuri, 2012).

Numerous species, serotypes and serovars of varying pathogenicity and host specificity exist within each genus and E coli is additionally characterised by differing pathotypes expressing different virulence factors such as EPEC (enteropathogenic E. coli) and ETEC (enterotoxigenic E. coli) (Kang et al., 2018) of which VTEC O157 is considered to be the most common cause of foodborne illness in humans (FSA, 2020). The role of free-living animals in maintaining reservoirs of gram-negative enteric species pathogenic to humans and livestock is unclear. Simpson (2008) reviewed wildlife cases of E. coli O157 infections in wildlife and concluded that free-living wild animals do not play a significant role in epidemiology. Similarly, Healing and Greenwood (1981) found that rodents living near a poultry farm in Dorset were reservoirs of some Campylobacter species but not Salmonella species detected in poultry on the same farm and proposed that rodents were not important reservoirs for Campylobacter and Salmonella species. However, Meerburg and Kijlstra (2007) reviewed several studies of Campylobacter and Salmonella species infections of small rodents and concluded that, in agricultural environments, rodents may maintain or amplify reservoirs of Campylobacter and Salmonella species infection.

Sub-clinical carriage of Salmonella species appears to be common in free-living wild animals (Gaffuri, 2012). Salmonella species, including some found in humans and/or livestock, have been reported in badgers (Meles meles) and red foxes with no macroscopic or microscopic lesions consistent with salmonellosis (Millan et al., 2004; Handeland et al., 2008; Chiari et al., 2014; Euden, 1990). However, salmonellosis has been reported in several species of free-living wild mammal and is most common between November and April in Europe (Gaffuri, 2012).

Chronic infection with Helicobacter species is usually asymptomatic in immunocompetent hosts (Whary and Fox, 2004) and disease occurs when host immunoregulation breaks down (Harbour and Sutton, 2008). In rodents, naturally acquired infections are common and persistent with prolonged shedding (Whary and Fox, 2004). Helicobacter species infections have been reported with no association between infection and clinical signs of disease, gross or microscopic, in free-living red foxes in Sweden, Slovenia and Turkey (Morner et al., 2008; Gruntar et al., 2020; Erginsoy et al., 2004) and in 60% (n=93/154) vertebrate species studied in a captive zoological collection over 10 years (Schrenzel et al., 2010).

E. coli is found as asymptomatic infections in the small and large intestines of many mammal species, with higher prevalence levels in carnivores compared to omnivores and herbivores for reasons that are not well understood (Speck, 2012a). Extra-intestinal disease in host animals usually results from translocation of normal intestinal flora rather than exogenous infection (ibid.). There are few reports of infection and disease associated with E. coli in free-living wild mammals, although VTEC O157 has been isolated from wild boar (Sus scrofa) in Sweden (Wahlstrom et al., 2003); rabbits (Oryctolagus cuniculus) in Great Britain (Simpson, 2008), and deer (Cervidae species)in Germany and Spain (Speck, 2012a), but factors such as stress and gut dysbiosis, for example as a result of a predominantly grain-based diet, can contribute to enteric overgrowth of E. coli and disease in domestic livestock (ibid.).

Infectious disease is a common diagnosis in free-living beavers. Infectious disease was associated with the death of 50% (n=22) of beavers following translocation from Germany to the Netherlands between 1988 and 1994 (Nolet et al., 1997) and 23.3% (n=60) beavers found dead in Germany and Austria between 1990 and 2003 (Steineck and Sieber, 2003); however, there may be uncertainty as to the causative agent.

Gram-negative bacteria have rarely been found in association with beaver deaths: one of the beavers examined by Steineck and Sieber (2003) was infected with an unspecified Salmonella species; S. enteriditis was identified in a co-infection in a Canadian beaver which died with streptococcosis at Berne Zoo (Dollinger et al., 1999); a wild-caught beaver from Norway (M08K33), which died during quarantine in the UK with severe enteritis and focal hepatic necrosis, was found to have an E. coli bacteraemia, although histopathology was reported to be suggestive of yersiniosis (Cranwell, 2009a) and Pilo et al. (2015) reported the death of a free-living beaver in Switzerland in 2013 in association with Klebsiella pneumoniae. In addition, two of three sub-adult beavers killed in road traffic collisions in Germany were infected with unspecified E. coli and Shigella species (Pratama et al., 2019), although it is not known whether the infections in these animals were associated with disease, and Laukova et al. (2014) identified Enterococcus species with potential virulence factors in pooled faecal samples from 12 free-living beavers in Poland.

Neither Salmonella species or Campylobacter species were found on culture of faecal samples from free-living beavers (n = 65) in Great Britain screened during survey work of populations in Knapdale and Tayside, Scotland or the River Otter, Devon (Campbell-Palmer et al., 2015b; Campbell-Palmer and Goodman, 2019; Goodman et al., 2014). In addition, 0/235 beavers examined by faecal culture for Salmonella species in Telemark,

Norway were positive (Rosell et al., 2001). However, in studies in humans, the numbers of enterobacteria shed in faeces declines over time with only low numbers detected in faecal samples from chronically infected people (Ethelberg et al., 2007) so it is possible that cases of infection with gram-negative enteric bacteria in beavers have been missed.

Beavers are herbivorous hindgut fermenters and are reliant for digestion on large colonies of cellulase-producing bacteria (Pratama et al., 2019). In other, better-studied, hindgut fermenters such as the rabbit, gut dysbiosis as a result of an inappropriate diet or other stressors leads to changes in intestinal motility and pH precipitating enterotoxaemia and overgrowth of some bacterial species such as E. coli (Oglesbee and Jenkins, 2012). Beavers may be susceptible to similar enteric diseases.

Given the evidence discussed above, gram-negative enterobacterial infection in beavers is probably asymptomatic in immunocompetent hosts but stressors may increase their susceptibility to the development of disease. As discussed elsewhere in this report, free-living beavers captured and translocated are known to be particularly susceptible to stressor-related disease and translocation is a known stressor (Dickens et al., 2010). Gram-negative enteric bacteria should therefore be considered as a carrier hazard for the translocation of beavers.

Risk Assessment

Release assessment

Beavers may be exposed to gram-negative enteric bacteria shed by other animals and in environmental reservoirs such as soil, water and on plant foodstuffs and infected by the oro-faecal route. In addition, they may be exposed to water-borne bacteria via mucous membranes. As most species of gram-negative bacteria have prolonged persistence in soil and water and are ubiquitous, and commensal in numerous animal species, the likelihood of a beaver being exposed to gram negative enteric bacteria and infected at the source site(s) is therefore high.

Exposure assessment

Translocated beavers with commensal gram-negative enteric species infections may shed bacteria in their faeces and contribute to environmental reservoirs of gram-negative bacterial species at the destination site(s). There is a medium likelihood that other beavers, humans and sympatric mammalian species at the destination site(s) will be exposed to and infected by gram negative enteric bacteria shed by beavers and a high likelihood that beavers and sympatric species infected at the destination site(s) will maintain and disseminate gram-negative enteric bacteria in their faeces. Since these bacteria are harboured by many free-living wild mammals, the release of beavers is unlikely to markedly affect the dissemination of gram-negative enteric bacteria and the prevalence and intensity in mammal populations.

Consequence assessment

There is a high likelihood of one translocated beaver being infected with gram-negative enteric bacteria.

Gram-negative enteric bacteria species are commensal in immunocompetent mammals. However, infected beavers stressed through handling, transport, and adjustment to release environments may be more susceptible to disease. The range of diseases caused by enteric bacteria is extensive but, in addition to enteritis, includes sepsis, pneumonia, organ necrosis and wound infections. There is a low likelihood of translocated beavers suffering from stressor-precipitated disease associated with gram-negative enteric bacteria. There is a very low likelihood of failure of the reintroduction and the associated economic and biological consequences because evidence noted above suggests that cases of disease are sporadic.

We are not aware of any reports of disease in humans or other species as a result of direct or indirect contact with beavers. In immunocompetent humans, infection with gram-negative enteric bacteria usually results in self-limiting enteric disease and the probability of severe biological or economic consequences is therefore very low.

Risk estimation

There is a high likelihood that beavers will be exposed to and infected by gram-negative enteric bacteria at the source site(s). The likelihood of exposure and infection at the destination is medium and the likelihood of dissemination is high. There is a low likelihood that the stress of translocation may precipitate disease in infected beavers and a very low likelihood of the failure of the translocation. There is a very low likelihood of biological and economic consequences as a result of disease in humans and livestock. The overall risk of disease due to gram negative enteric bacteria in translocated beavers is MEDIUM.

Disease Risk Management

Risk management options

Testing asymptomatic beavers for infection with gram-negative enteric bacteria is likely to be of limited value as these agents are normal commensal organisms and infected beavers may be healthy and not necessarily of risk to other beavers or mammals. However, post-mortem examination of any beaver found dead or electively euthanased on welfare grounds with appropriate culture and possibly sequencing of associated infectious agents is strongly recommended in order to improve our understanding of gram-negative enteric species harboured by beavers.

Appropriate measures to minimise stress during capture, handling and transport should be undertaken. In addition, appropriate dietary provision should be made during any period in captivity, with emphasis on the provision of suitable browse, ideally taken from the source site.

To reduce the risk of zoonotic diseases, routine hygienic precautions such as use of disposable gloves and hand washing should be employed. Gloves should be worn whenever handling animals, and during the cleaning and disinfection of all equipment and transport materials. Equipment such as transport crates should be cleaned with detergent and water and then disinfected with a suitable agent diluted according to the manufacturer's guidelines.

It may be important to conserve commensal parasites during translocation because it may be counterproductive to create a population of beavers at the release site without exposure and immunity to these parasites, should a non-immune population be subsequently exposed to them.

Appendix 7 Disease Risk Analysis for the Carrier Hazard Streptococcus castoreus

Streptococcus species are gram-positive cocci of worldwide distribution responsible for a wide range of suppurative conditions and abscess formation in host animals (Quinn et al., 2011). Most species are found as commensals in the upper respiratory or urogenital tract of the host and have poor survival in the environment (ibid). The genus comprises both highly host-adapted and tissue-trophic species of varying pathogenicity as well as more generalist organisms only capable of causing disease as opportunists (Speck, 2012b).

Justification of Hazard Status

A novel Streptococcus species was isolated by Lawson et al., (2005) from the carcass of a Eurasian beaver that had died in a wildlife park as a consequence of multiple bite wounds from conspecifics. Gene sequencing confirmed that the novel species was a beta-haemolytic group A Streptococcus species which exhibited more than 3% diversity from other, reference streptococcal species and was most closely related to, but phenotypically and phylogenetically distinct from, S. porcinus and S. iniae (ibid.). Lawson et al. (2005) named this novel bacterium S. castoreus sp. nov. S. castoreus was subsequently isolated from 44% of beavers (n=16) found dead in Germany and these beavers were co-infected with other gram-positive and gram-negative bacteria (Schulze et al., 2015). S. castoreus was cultured from rectal swabs from two of these 16 animals suggesting that it is part of the normal commensal enteric flora in Eurasian beavers (ibid.). Schulze et al. (2015) found that in four of seven cases S. castoreus was associated with suppurative lesions but a mixed bacterial flora was grown from all four suppurative lesions. The other bacteria grown are also associated with pus-forming lesions and therefore the pathogenicity of S. castoreus is unclear. A summary of the post-mortem findings is given at Table 3.

Table 3 Post-mortem findings in beavers infected with S. castoreus. (Source: Schulze et al., 2015)

Isolate Identifier	**Animal characteristics, localisation of *Streptococcus castoreus* isolation and significant diseases**			
	Sex, age, body condition	Isolated from	Accompanying bacterial flora	Significant concurrent diseases
10UCF103	Male, juvenile, emaciated	Abscessing gonarthritis	*Fusobacterium necrophorum, Prevotella* sp.	Alveolar echinococcosis, Tibia fracture
11UCF142	Male, adult, emaciated	Biting wound abscess	Species of the *Actinomycetaceae* family, *Fusobacterium necrophorum*	Metacarpal fracture
11UCF216	Male, adult, fair	Incised skin wound, internal organs	*Actinobacillus* sp., *Prevotella* sp.	Septicaemia following wound infection
12UCF3	Male, adult, good	Suppurative laryngitis	*Yersinia pseudotuberculosis*	Yersiniosis
12UCF17	Female, adult, good	Suppurative cloacitis	Coliform bacteria	Fatty heart muscle degeneration
12UCF33	Male, adult, fair	Normal cloaca	Coliform bacteria	Tularameia, Postrenal uraemia
12UCF94	Female, adult, emaciated	Normal cloaca	Coliform bacteria, *Staphylococcus aureus*	Endocarditis and septicaemia (*Staphylococcus aureus*)

Further evaluation between 2010 and 2017 by Mühldorfer et al. (2019) of 27 Streptococcus species isolates from 18 free-living Eurasian beavers, 17 from Germany, including the seven previously assessed by Schulze et al. (2015), one from the UK, and four captive Canadian beavers, confirmed that all isolates were S. Castoreus. Twelve of the 27 isolates were found in the respiratory or intestinal tract in otherwise apparently healthy beavers and so Mühldorfer et al. (2019) concluded that S. castoreus is a normal commensal organism in beavers but may, in common with other Streptococcus species, act as an opportunistic pathogen under certain circumstances. It should be noted that, as far as we understand, Mühldorfer et al. (2019) isolates were not grown in pure culture from a lesion in any of the 27 cases and therefore the pathogenicity of this bacterium is uncertain. Additionally, as S. castoreus has not been isolated from any other species, Mühldorfer et al. (2019) proposed that S. castoreus is a host-specific species.

Opportunistic pathogens are usually of low pathogenicity under normal circumstances but when host immunity is impaired, they may behave as conventional pathogens to cause disease in the host (Shanson, 1989). As translocation is a known stressor and stress may reduce host immunocompetence (Dickens et al., 2010), on the assumption that S. castoreus is an opportunistic pathogen, it should be considered a carrier hazard for the translocation of beavers.

Risk Assessment

Release assessment

Streptococcus species can be isolated from bodily fluids including nasal discharges, pus, milk and exudative infected tissues (Speck, 2012b). As Streptococcus species are of short-lived duration in the environment, and are commensal bacteria in the respiratory and intestinal tracts, beavers are exposed to, and infected by, S. castoreus bacteria harboured by conspecifics through maternal milk, mutual grooming and bite wounds. Beavers may also transfer infection through licking or chewing lesions (Schulze et al., 2015).

Exposure assessment

There is a high likelihood that S. castoreus will be transmitted between beavers during translocation, or at the destination site, by maternal suckling, mutual grooming or fighting. Other beavers translocated to the destination may already be infected.

Since S. castoreus appears to be host-specific and Streptococcus species do not survive well in the environment, the likelihood of exposure of, and dissemination to, other species at the destination site(s) is very low in the short term, but as a commensal infectious agent there is a high likelihood that it would be transmitted through the reintroduced population in the long term.

Consequence assessment

There is a high probability that at least one beaver is infected with S. castoreus when translocated because this bacterium is a component of the normal commensal flora.

On the assumption that S. castoreus is confirmed as an opportunistic pathogen, there is a high probability that if beavers are under stress and consequential immunodepression, from trauma during capture or transit, or respiratory disease, they will be predisposed to develop S. castoreus- associated disease. There is substantial evidence that beavers are prone to severe disease and even fatalities following minor injuries and, in addition, susceptible to stressors (Campbell-Palmer and Rosell, 2015) and therefore there is a high probability of stressor associated diseases in general. Mühldorfer et al. (2019) reported that S. castoreus was associated with a range of lesions from local suppurative inflammation to systemic infection, but not in pure culture as far as we understand, and therefore its pathogenicity remains unclear. A captive Canadian beaver died at Berne zoo as a result of streptococcosis (Streptococcus species not identified), although Salmonella enteriditis was also cultured (Dollinger et al., 1999).

There is therefore a high likelihood that the stress of translocation will lead to immunocompromise resulting in severe S. castoreus-associated disease in an injured or sick beaver. However, reports appear to show disease incidence is sporadic and therefore there is a very low likelihood of economic and biological consequences due to translocation failure. There is a negligible likelihood of biological or ecological consequences due to dissemination of S. castoreus at the destination because S. castoreus is a commensal infectious agent, and conservation of infection may be important to the future health of the reintroduced population.

Risk estimation

There is a high likelihood that beavers will be exposed to and infected with S. castoreus at the source site(s) and a high likelihood that other beavers will be exposed to and infected with S. castoreus at the destination but a very low likelihood of onward transmission to other species and dissemination at the destination site in the short term and a high likelihood in the long term. There is a high likelihood that translocation acts as a stressor on beavers and, given their known susceptibility to stress, there is a high likelihood of disease associated with S. castoreus. There is a very low likelihood of economic and biological consequences due to translocation failure. The overall risk from disease caused by S. castoreus is estimated to be QUALIFIED MEDIUM, on the assumption that S. castoreus is an opportunistic pathogen.

Risk Management

Risk management options

In addition to measures to minimise stress to beavers during capture and handling, care should be taken to avoid injuries, through careful planning and preparation of translocation methods, and to ensure that prompt veterinary attention is given to even apparently minor injuries where veterinary intervention is unlikely to cause further stress to the beaver(s). Particular attention should be taken to minimise the risk of fight injuries and bite wounds by avoiding mixing of non-related beavers and releasing beavers at low density into environments with ample opportunities for dispersal and territory establishment.

It may be important to conserve commensal parasites during translocation, because it may be counterproductive to create a population of beavers at the release site without exposure and immunity to these parasites, should a non-immune population be subsequently exposed to them.

Appendix 8 Disease Risk Analysis for the Carrier Hazard and Hazard for Domestic and Free-living Mammals in England Mycobacterium species

Mycobacteria are rod-shaped, non spore-forming acid-fast bacilli. About 200 species have been identified to date, many of which can infect a wide range of hosts, including humans, causing a range of clinical outcomes from latent and asymptomatic infection to active infection with severe disease (Larsen et al., 2020). Reactivation of latent infection may be more likely with increasing age and reduced immunocompetence (Gavier-Widen et al., 2012). Most are environmental, opportunistic pathogens, existing as saprophytes in soil and water (Percival and Williams, 2014). Two mycobacterial complexes are of particular interest: Mycobacterium tuberculosis (MTBC) and Mycobacterium avium (MAC). MTBC includes M. bovis, the most common cause of tuberculosis in domestic livestock and wildlife in the UK; M. tuberculosis, mainly found in humans; and M. microti. The principal species of interest in MAC are M. avium subsp. avium (MAA) and M. avium subsp. paratuberculosis (MAP), the causative agent of Johne's Disease in livestock (Percival and Williams, 2014).

Mycobacterium bovis

The primary host for M. bovis in the UK is cattle with uncertainty regarding the role of wildlife species, notably the European badger and deer, in maintaining the cycle of transmission (Gavier-Widen et al., 2012). Estimates of M. bovis prevalence in the European badger in the UK vary but may be as high as 24.2% (Allen et al., 2018). M. bovis has also been reported in a wide range of free-living wildlife hosts including rodents which are considered to be relatively resistant to disease following infection (Gavier-Widen et al., 2012). Delahay et al. (2007) cultured and spoligotyped 4,715 tissue samples from 32 wildlife species trapped or culled in south-west England in areas with high prevalence of M. bovis infection in cattle. Low levels of prevalence were found in 12 species tested (Table 4). These results were compared to gross pathological findings. No gross lesions were observed in culture-positive small mammals, grey squirrels (Sciurus carolinensis) and polecats (Mustela putorius). Delahay et al. (2007) concluded that species other than deer and badgers were therefore probably not a high risk to livestock. Comparison of M. bovis strains in a national park in Spain has similarly indicated that spill-back events from most species of wildlife to livestock are probably rare (Gortazar et al., 2011). These results show that the prevalence of M bovis in rodents, and therefore their susceptibility to infection, is probably very low.

Table 4 Prevalence of M. bovis infection in mammals, south-west England. (From Delahay et al., 2007)

Species name	Prevalence (%)	Number tested positive
Red fox (Vulpes vulpes)	3.17	24/756
Stoat (Mustela erminea)	3.85	3/78
Polecat (Mustela putorius)	4.17	1/24
Common shrew (Sorex araneus)	2.44	1/141
Yellow-necked mouse (Apodemus flavicollis)	2.78	1/36
Wood mouse (Apodemus sylvaticus)	0.006	2/333
Field vole (Microtus agrestis)	1.49	1/67
Grey squirrel (Sciurus carolinensis)	0.44	2/450
Roe deer (Capreolus capreolus)	1.02	9/885
Red deer (Cervus elaphus)	1.02	2/196
Fallow deer (Dama dama)	4.37	22/504
Muntjac deer (Muntiacus reevesi)	5.17	3/58

M. avium subsp. paratuberculosis

M. avium subsp. paratuberculosis (MAP) is predominantly associated with ruminant species but has been found in non-ruminants, in particular lagomorphs which probably serve as a reservoir of infection (Gavier-Widen et al., 2012). Annual surveillance of domestic livestock in Norway has found no new cases of MAP infection since 2014 (Kampen et al., 2019). However, MAP is reported by Tryland et al. (2004) to have been endemic in goat (Capra species) herds in western Norway prior to implementation of a vaccination programme from 1967 with prevalence in 1997 and 1998 in these areas of 12.2% in roe deer (Capreolus capreolus) (n=6/49) and 3.8% in red deer (Cervus elaphus) (n=14/371) suggesting historic spill-over into wildlife hosts. MAP is considered ubiquitous in Great Britain (APHA, 2020). A study of 591 animals from 18 non-ruminant wildlife

species in Scotland (Beard et al., 2009) isolated MAP by culture and PCR from 10 species (Table 5).

Table 5 Diagnosis of M. avium subsp. paratuberculosis in wildlife, Scotland. (From Beard et al., 2001)

Species	Tissue culture +/ve	Faeces culture +/ve	Histopathology +/ve
Red fox (Vulpes vulpes)	23/27	3/27	12/26
Stoat (Mustela erminea)	17/37	1/6	1/13
Weasel (Mustela nivalis)	2/4	N/A	2/4
Hare (Lepus europaeus)	1/6	0/3	0/4
Badger (Meles meles)	½	NA	0/1
Rat (Rattus norvegicus)	3/35	0/7	0/23
Wood mouse (Apodemus sylvaticus)	3/88	2/2	1/88
Carrion crow (Corvus corone)	36/60	4/12	1/60
Rook (Corvus corax)	3/53	1/1	0/53
Jackdaw (Corvus monedula)	1/38	NA	0/38

Where a positive diagnosis of MAP infection was made, histopathological signs were subtle or absent. Rats and mice, in particular, had minimal lesions (ibid.). However, MAP was cultured from the faeces of wood mice (Apodemus sylvaticus), suggesting rodents'

potential to act as a source of transmission of MAP to other species, either through predation/scavenging, or through faecal contamination of food sources.

M. avium subsp. avium

M. avium subsp. avium (MAA) is the recognised cause of avian tuberculosis, which is particularly prevalent in water-fowl, and detected in a wide range of captive and free-living mammals (Gavier-Widen et al., 2012). MAA has been isolated from brown rats and grey squirrels without visible lesions and is of low virulence in field voles and coypu (Granger, 1990). Humans are considered resistant to disease following infection unless immunocompromised and this may be true for other species where stress-induced morbidity has been reported in captive animals (ibid.). The main route of infection is faeco-oral, via the environment, and direct transmission between mammals is probably very rare (Thorel et al., 2001).

Other Mycobacteria species

M. microti is considered to be endemic in the UK with mice and voles the main reservoir hosts (Gavier-Widen et al., 2012). 21% (n=38/180) of field voles in Kielder were found to have grossly visible cutaneous or abdominal lesions on post-mortem examination (Cavanagh et al., 2002). M. microti spoligotypes were confirmed in 12/13 cutaneous lesions and 5/7 abdominal lesions but no confirmed cases were positive on urine or faecal spoligotyping, suggesting that shedding of M. microti bacilli is intermittent (ibid.). Cavanagh et al. (2002) also isolated M. microti from three bank voles and two wood mice. Cats that hunt small rodents are recognized as frequent spill-over hosts but infection has also been occasionally reported in other species such as the badger, Eurasian otter (Lutra lutra) and grey squirrel (Michelet et al., 2015).

Mycobacterium lepromatosis, M. leprae and M. lepraemurium are the cause of lepromatous leprosy in many species including red squirrels and humans (Meredith et al., 2014) and rats, mice and cats (Rojas-Espinosa and Lovic, 2001).

Hazard for Domestic and Free-Living Mammals in England- Justification of Hazard Status

Mycobacterial infections, in particular M. bovis and MAP, are a major cause of morbidity and economic loss in many species, particularly dairy cattle (Bos taurus). Large areas of Europe, including Norway, Sweden, Germany and Scotland, are considered free from M. bovis and stringent measures are underway in all European Union (EU) countries to eradicate reservoirs of infection (Visavet, 2020). However, the UK continues to be the most severely affected of European member states, accounting for more than half of the M. bovis test-positive dairy herds in the EU in 2018 (n=10,334/18,801) with prevalence over 10% (EFSA, 2020). If beavers infected with mycobacterial species currently the subject of a control programme in the UK are translocated, their translocation may affect control goals in England and therefore these mycobacteria are evaluated as a hazard, with an emphasis on Mycobacterium bovis.

Risk Assessment

Release assessment

Scotland and Norway, as outlined above, are considered free from M. bovis. The origin of most free-living beavers in Scotland is not known with certainty but includes Germany, also considered free from M. bovis. It is possible that historic, unauthorised releases of beavers in Scotland, England and Wales could have included beavers from captive collections or geographic regions which were exposed to M. bovis and with the potential to transmit M. bovis to con-specifics and offspring. Free-living beavers in England and Wales may have been exposed to M bovis from free-living wildlife or domestic cattle reservoirs since they were released. In England and Wales exposure will be more likely in areas with known infection in wildlife / cattle, being highest for beavers inhabiting areas in close proximity to dairy cattle or badgers.

Transmission of MTBC species is primarily aerogenous, and faeco-oral for MAC species, but a wide range of transmission routes, including bite-wounds, is possible for all species with the environment a key source of exposure due to the potential for prolonged survival of bacilli in water and soil. The environment, in particular water, is probably the main reservoir of MAA (Percival and Williams, 2014). By contrast, animal hosts are probably the primary reservoirs for the other Mycobacteria species of interest. Animals that do not develop granulomas following infection may, therefore, have low infection potential but Gavier-Widen et al. (2009) report that microscopic lesions are frequently detectable by histopathology in animals without visible granulomas and that these animals may still present a risk to other animals if predated, scavenged or inadvertently ingested via contaminated foodstuffs.

Beavers may be exposed to Mycobacteria species in water and soil and on plant materials. In addition, MAA and MAP probably replicate in soil and water, increasing the environmental reservoir of infectious bacilli (Percival and Williams, 2014). Mycobacteria species are capable of prolonged survival in the environment due to their hydrophobic, lipid-rich cell walls which enable them to withstand desiccation and ultra-violet light (Gavier-Widen et al., 2012).

Prevalence of infection with M bovis in rodents is very low as indicated above, and rodents appear to be less susceptible than other mammals. Therefore, the likelihood of M bovis infection in a translocated beaver is very low.

Beavers could be exposed to other Mycobacteria species such as M. microti and MAC species through accidental ingestion of contaminated plant material or water. MAA and M. microti are ubiquitous and MAP is widely distributed in Great Britain and may be present in wildlife reservoirs in Norway. Prevalences of M microti, and probably MAC, are higher in rodents than M bovis in rodents, and therefore there is a medium probability that translocated beavers are infected with M microti and MAC.

Exposure assessment

An infected beaver could shed Mycobacteria bacilli in saliva, urine or faeces, depending on the location of lesions, which could be either inhaled by other animals or ingested from the environment in contaminated soil, water or food items. In addition, animals could become infected by predating or scavenging an infected beaver or through bite wounds from an infected beaver. The likelihood of transmission to conspecifics depends on host density, distribution and behaviour (Gavier-Widen et al., 2012). For example, badgers tend to aggregate in underground setts, use communal latrines, move between family groups and fight frequently, increasing their risk from all routes of transmission: aerogenous, environmental, ingestion and bite-wounds (ibid.).

The likelihood of conspecific transmission among beavers is unknown but is likely to be low as beavers live in small family groups at low density (Gurnell et al., 2008) and rodents rarely experience extensive granuloma formation. As beavers inhabit aquatic environments there is potential for widespread dissemination of infectious bacilli within watercourses and in riparian margins to sympatric species. However as rodent species do not appear to be susceptible to severe disease following infection, shedding of bacilli is likely to be low and beavers are unlikely to act as a major source of mycobacteria, and increase the mycobacterial load, in the destination environment. There is a low likelihood that mammals at the destination will be exposed and infected with mycobacteria.

Many different mammalian species have been shown to be susceptible to infection with Mycobacteria species and bacilli are extremely persistent in the environment and so there is a high probability of dissemination.

Consequence assessment

There is a low likelihood of one translocated beaver being infected with mycobacteria.

Following infection with Mycobacteria species, a cell-mediated immune response may result in the formation of granulomas in organs and lymphatic tissue. Lympho-haematogenous dissemination and granuloma rupture facilitate the spread of infectious bacilli within the host and shedding, for example through nasal secretions, urine or faeces (Gavier-Widen et al., 2012). As a result, shedding is intermittent and may be related to the size and location of granulomas (ibid.). The location of mycobacteria lesions is thought to relate to the route of infection: aerogenous infection causing predominantly pulmonary lesions, ingestion causing primarily alimentary lesions and bites causing cutaneous lesions. However, as disease progresses, bacilli may spread by haematogenous distribution to multiple organs. (ibid.). Haematogenous dissemination of large numbers of mycobacterial bacilli simultaneously may result in miliary tuberculosis, a fast-developing spread of numerous, small white foci of infection. More typically, disease progress is slow, with growth and coalescence of large granulomas ultimately resulting in organ failure and death (ibid.). MAP infection of ruminants causes chronic enteritis and progressive weight loss (Beard et al., 2001) and has been associated with Crohn’s Disease in humans (Percival and Williams, 2014).

Infected animals and humans are variably susceptible to disease following infection with Mycobacteria species and even individuals from species normally resistant to disease may, under some circumstances, develop severe lesions (Gavier-Widen et al., 2012). However, in general, domestic mammals and humans are relatively resistant to MAA infection unless immunocompromised (ibid.) which may result in pulmonary lesions and/or lymphadenitis (Percival and Williams, 2014).

There are severe biological and economic costs as a result of mycobacterial disease in livestock and sympatric species, and humans, following infection. However, since Mycobacteria species remain widely distributed in reservoir hosts and the environment in England, rodents are not an important component of that reservoir, and that small numbers of beavers at low density will be released, the biological and economic consequences attributable to beaver translocation are likely to be negligible.

Risk estimation

The likelihood of M bovis infection in a translocated beaver is very low. There is a medium probability that translocated beavers are infected with M microti and MAC. There is a low likelihood of exposure of mammals at the destination and a high probability of dissemination to sympatric species at the destination site(s). The consequences to mammals in England from the translocation of beavers is negligible. The overall risk to mammals in England from beaver translocation is NEGLIGIBLE.

Disease Risk Management

Risk evaluation

Although the risk from mycobacteria to other mammals in England is considered negligible we consider option evaluation.

Risk management options

Testing for mycobacterial infection is unlikely to be rewarding. Isolation, culture and spoligotyping of Mycobacteria species is regarded as the gold standard method of diagnosis but cannot be effectively performed in the live animal as shedding of bacilli is intermittent and bacterial growth is slow, often up to 12 weeks and potentially six months for MAP, and requires specialist laboratory facilities (Gavier-Widen et al., 2012). Serological assays to detect antibodies may be used to test wildlife for M. bovis but sensitivity tends to be low and tests may only work reliably in animals with more severe disease (Chambers, 2009). In addition, validation of serological tests has not, as far as we are aware, been performed for beavers, while cross-reactivity with non-pathogenic environmental mycobacteria may also be an issue (Gavier-Widen et al., 2012).

The intradermal tuberculin test used in cattle could potentially be used in beavers for detection of M. bovis exposure but sensitivity is of variable reliability in wildlife species and a minimum of 72 hours is required before results can be assessed (Chambers, 2009). Enzyme immunoassays may offer the greatest promise but would require validation and

must be performed on fresh blood samples (ibid.) so may have only limited potential for use in beavers. BAL, chest radiographs and abdominal ultrasound could be used in the anesthetised animal to detect pulmonary infections and gross lesions but sensitivity and specificity are likely to be unacceptably low.

Given the M bovis free status of Norway and Scotland the beavers in these countries represent a good source population from the perspective of risk of mycobacterial disease in domestic and free-living mammals in England.

Carrier hazard- Justification of Hazard Status

A known case of MAA-associated disease in a beaver (Nolet et al., 1997) and reported prevalence of MAA in other rodent species suggest that beavers may be susceptible to infection following exposure to Mycobacteria species. Progress of disease following infection with Mycobacteria species depends on the ability of the host animal to mount a successful immunological response in order to control the multiplication rate of bacilli and so host immunocompetence may have a major effect on the degree of morbidity experienced (Gavier-Widen et al., 2012). As all translocations are associated with stress (Dickens et al., 2010), and stress precipitates reduced immunocompetence, translocated beavers will be predisposed to clinical disease following infection with Mycobacteria species which should therefore be considered as carrier hazards for the translocation of Eurasian beavers.

Risk Assessment

Release assessment

Transmission of MTBC species is primarily aerogenous, and faeco-oral for MAC species, but a wide range of transmission routes, including bite-wounds, is possible for all species with the environment a key source of exposure due to the potential for prolonged survival of bacilli in water and soil. The environment, in particular water, is probably the main reservoir of MAA (Percival and Williams, 2014). By contrast, animal hosts are probably the primary reservoirs for the other Mycobacteria species of interest. Animals that do not develop granulomas following infection may, therefore, have low infection potential but Gavier-Widen et al. (2009) report that microscopic lesions are frequently detectable by histopathology in animals without visible granulomas and that these animals may still present a risk to other animals if predated, scavenged or inadvertently ingested via contaminated foodstuffs.

Beavers may be exposed to Mycobacteria species in water and soil and on plant materials. In addition, MAA and MAP probably replicate in soil and water, increasing the environmental reservoir of infectious bacilli (Percival and Williams, 2014). Mycobacteria species are capable of prolonged survival in the environment due to their hydrophobic, lipid-rich cell walls which enable them to withstand desiccation and ultra-violet light (Gavier-Widen et al., 2012).

There is a medium likelihood that translocated beavers are infected with mycobacteria.

Exposure assessment

An infected beaver could shed Mycobacteria bacilli in saliva, urine or faeces, depending on the location of lesions, which could be either inhaled by other animals or ingested from the environment in contaminated soil, water or food items. In addition, animals could become infected by predating or scavenging an infected beaver or through bite wounds from an infected beaver. The likelihood of transmission to conspecifics depends on host density, distribution and behaviour (Gavier-Widen et al., 2012). For example, badgers tend to aggregate in underground setts, use communal latrines, move between family groups and fight frequently, increasing their risk from all routes of transmission: aerogenous, environmental, ingestion and bite-wounds (ibid.). The likelihood of conspecific transmission among beavers is unknown but is likely to be low as beavers live in small family groups at low density (Gurnell et al., 2008) and rodents rarely experience extensive granuloma formation.

As beavers inhabit aquatic environments there is potential for widespread dissemination of infectious bacilli within watercourses and in riparian margins to sympatric species. However as rodent species do not appear to be susceptible to severe disease following infection, shedding of bacilli is likely to be low and beavers are unlikely to act as a major source of mycobacteria, and increase the mycobacterial load, in the destination environment. There is a low likelihood that mammals at the destination will be exposed and infected with mycobacteria.

Many different mammalian species have been shown to be susceptible to infection with Mycobacteria species and bacilli are extremely persistent in the environment and so there is a high probability of dissemination at the destination site(s).

Consequence assessment

There is a low likelihood of one translocated beaver being infected with mycobacteria.

There has been one recorded case of MAA associated with mortality in a beaver which died just under two years following translocation to the Netherlands (Nolet et al., 2007). The susceptibility of beavers to infection with other Mycobacteria species is unknown but, given the widespread prevalence of mycobacterial infection in other rodent hosts, it should be assumed that beavers are similarly susceptible and could, under certain conditions, develop clinical disease following infection. Beavers in England and Scotland have been tested for disease associated with M. bovis by broncho-alveolar lavage (BAL) and/or chest radiographs (n = 20) and MAP infection by faecal microscopy (n = 70) with no positive results to date (Campbell-Palmer et al., 2015b; Campbell-Palmer and Girling, 2019). However, as diagnostic testing is not very sensitive (see below for further discussion of testing protocols), it is possible that cases of infection have been missed.

Following infection with Mycobacteria species, a cell-mediated immune response may result in the formation of granulomas in organs and lymphatic tissue. Lympho-haematogenous dissemination and granuloma rupture facilitate the spread of infectious bacilli within the host and shedding, for example through nasal secretions, urine or faeces

(Gavier-Widen et al., 2012). As a result, shedding is intermittent and may be related to the size and location of granulomas (ibid.). The location of mycobacteria lesions is thought to relate to the route of infection: aerogenous infection causing predominantly pulmonary lesions, ingestion causing primarily alimentary lesions and bites causing cutaneous lesions. However, as disease progresses, bacilli may spread by haematogenous distribution to multiple organs. (ibid.). Haematogenous dissemination of large numbers of mycobacterial bacilli simultaneously may result in miliary tuberculosis, a fast-developing spread of numerous, small white foci of infection. More typically, disease progress is slow, with growth and coalescence of large granulomas ultimately resulting in organ failure and death (ibid.).

Recrudescence of latent infection may be triggered by stress following translocation. In addition, beavers may be less resistant to infection and disease progress following exposure at the destination site(s). Infected beavers may therefore develop disseminated granulomas, resulting in organ failure, severe morbidity and death. As disease progress can be slow, these effects on individual beaver health may not be discernible for months or even years following translocation. Infected beavers experiencing severe disease may be more likely to shed bacilli and contribute to dissemination of Mycobacteria species at the destination site(s) through faeces, urine or saliva as well as constituting an infection risk to predators and scavengers after death. There is a low likelihood of disease in translocated beavers but the probability of failure of the translocation is negligible. The biological, environmental and economic consequences are negligible.

Risk estimation

There is a medium likelihood that a translocated beaver is exposed to and infected with mycobacteria. There is a low likelihood of exposure of mammals at the destination and a high likelihood of dissemination. There is a low likelihood of disease in translocated beavers. The overall risk is LOW.

Disease Risk Management

Risk evaluation

Preventative measures should be considered to reduce stress associated with translocation and to reduce the risk of exposure to and infection with Mycobacteria species.

Risk management options

In line with previous recommendations, efforts should be made to minimise stress to beavers during capture and transit and to reduce the level of handling and duration of time in transit and captivity to the lowest possible levels.

Consideration could be given to the use of BCG vaccination which has been shown be effective in wild boar, red deer and badgers against M. bovis (Balseiro et al., 2010) and, in humans, has been shown to protect against other Mycobacteria species (Zimmermann et

al., 2018). Additionally, release sites with reduced access for grazing livestock and low levels of waterfowl presence could be considered.

PCR and/or extended culture of tissues removed during post-mortem examination of beavers found dead before or after release is therefore recommended in order to improve understanding of mycobacterial infection and disease progression and characterisation in beavers.

Appendix 9 Disease Risk Analysis for the Carrier Hazard Stichorchis subtriquetrus

Justification of Hazard Status

Stichorchis subtriquetris, the beaver fluke, is a trematode of both Eurasian and Canadian beavers, not known to infect other species (Demkowska-Kutrzepa et al., 2016). Its life cycle involves infection of the intermediate host, aquatic snails of Bithinia, Planorbis and Lymnaea species (ibid.), and ingestion of metacercariae attached to aquatic plants by beavers (Vengust et al., 2009).

Parasite prevalence from post-mortem examination analysis of beavers, has been recorded at levels as high as 93.7% (n=45/48) in Poland (Demiaszkiewicz et al., 2014) and 100% (n=30/30) in Sweden (Ahlen, 2001). Such high levels of prevalence may be related to the limited genetic diversity of host animals following a near-extinction bottleneck (ibid.) and to a loss of parasite diversity following captive management and reintroduction (Drozdz et al., 2004). S. subtriquetrus ova were found by faecal examination in 70% (n=14/20) of free-living beavers examined alive or post-mortem on Tayside in Scotland, most of which are believed to have originated from, or descended from, Bavarian beavers (Campbell-Palmer et al., 2015b). However, this may be an underestimate of prevalence because S. subtriquetrus ova shedding is likely to be intermittent (ibid.). Crucially there has been a confirmed case of S. subtriquetrus infection in a British-born beaver from Tayside, confirming that the parasite is able to complete its life cycle through suitable intermediate hosts in Great Britain (Campbell-Palmer et al., 2013).

63% (n=10/16) of beavers imported from Norway for the Knapdale trial were found to be infected either pre- or post-release; none were treated with anthelmintics (Goodman et al., 2014). Parasite burdens are reported to be twice as heavy in young animals under two years old (n=11) compared to adults (n=34) (Demiaszkiewicz et al., 2014): Mean S. subtriquetrus intensity in young beavers was 201 trematodes (range 5-479) compared to mean intensity in adult beavers of 93 trematodes (range 2-893). This may indicate that immunocompetence to S. subtriquetrus infection is increased in the healthy adult animal.

Translocation is a known stressor (Dickens et al., 2010) and susceptibility to morbidity and mortality may be increased by stress. Therefore S. subtriquetrus should be considered as a carrier hazard for the translocation of beavers.

Risk Assessment

Release assessment

Beavers are infected through ingestion of metacercariae attached to aquatic plants which form part of the beaver's diet. These metacercariae complete their life cycle to adult trematodes in the host. (Vengust et al., 2009). Trematodes are typically found in the caecum and with decreasing frequency in the colon and small intestine, and rarely in the

stomach of beavers (Sikorowski et al., 2016). Ova are shed in beaver faeces into water and are consumed by the intermediate aquatic snail host. As beavers live in family groups, there is a high likelihood that an infected beaver could disseminate S. subtriquetrus to other beavers, via the intermediate host, in the same habitat which will ingest metacercariae while foraging. Infection appears to be seasonal with highest burdens in the autumn (ibid., Drozdz et al., 2004). As S. subtriquetrus adult infestation is prevalent in beavers in both Great Britain and Norway, there is a very high probability of an infected beaver being released.

Exposure assessment

As the parasite has been shown to complete its lifecycle through intermediate hosts in Great Britain, there are likely to be infectious metacercariae present at release sites which will be ingested by beavers when they eat aquatic plants. As a result, there is a high probability that beavers at the destination site(s) will be exposed to and infected by S. subtriquetrus. There is a high likelihood of dissemination as a result of animals with S. subtriquetrus being released because the lifecycle of the parasite can be completed in Great Britain and beavers will be in relatively high-density family groups.

Consequence assessment

There is a high likelihood of a translocated beaver being infected with S. subtriquetrus. Infection is normally asymptomatic (Sager et al., 2005). However, heavy burdens are associated with parasite presence outside the caecum where they may cause clinical signs (Demiaszkiewicz et al., 2014). In histopathological examination of three infected beavers, Niemeic et al. (2016) reported that parasite presence was associated in the large intestine with chronic inflammation and Cirovic et al. (2009) reported that in an earlier study, Romashov and Safonov (1965), burdens greater than 150 trematodes were observed in association with chronic inflammation and vomiting, diarrhoea, weakness, anorexia, constipation and anaemia but did not confirm whether this was an isolated case, nor have we been able to verify the source.

Immunocompetent and healthy beavers would be expected to tolerate low levels of infection with S. subtriquetrus. However, beavers undergoing handling, transport, and adjustment to release environments, and therefore stressed, may be more susceptible to disease and experience morbidity or mortality. Three beavers (M08K22, M08K29, M08K31) died in captivity in association with S. subtriquetrus infection and in one of these, M08K29, the pathologist attributed focal ulceration and haemorrhage in the large intestine and poor body condition to the parasite burden (Deuchande, 2009; Howie, 2009; Collins, 2009). There is a low likelihood of a high proportion of translocated beavers suffering from stressor-initiated trematode-associated-disease and a failure of the reintroduction and the associated economic and biological consequences.

Risk estimation

There is a high likelihood of beavers being exposed to S. subtriquetrus and a very high likelihood of an infected beaver being released. There is a high likelihood of exposure and

dissemination of the parasite at the release site. There is a low probability that the stress of translocation may precipitate disease in a high proportion of translocated infected beavers and lead to failure of the reintroduction. The overall risk from disease caused by S. subtriquetrus is therefore MEDIUM.

Disease Risk Management

Risk evaluation

Based on the risk assessment above, preventative measures should be employed to reduce the risks from S. subtriquetrus as a stress hazard.

Risk management options

Measures to reduce the stress from translocation are important. For example, efforts should be made to minimise stress from capture, transport and, in particular, repeated handling and to reduce transit times. Consideration should also be given to the timing of releases, avoiding winter months in the event that the autumn burden of S. subtriquetrus might be at its highest.

Appendix 10 Disease Risk Analysis for the Source Hazard Echinococcus multilocularis

Echinococcus multilocularis is a tapeworm (cestode) of, primarily, the red fox which can cause morbidity and mortality in intermediate hosts (Barlow et al., 2011). It is endemic in many parts of Europe but is not currently present in Great Britain.

The lifecycle of the tapeworm in Europe involves two hosts (see Figure 1): a definitive, or primary, canid host, including the red fox, the raccoon dog, grey wolf (Canis lupus), golden jackal (Canis aureus) and Arctic fox (Vulpes lagopus). Pet dogs can also be infected as a definitive host, with increasing prevalence in endemic areas (Karamon et al., 2016). Domestic cats and wild cats (Felis silvestris) can be infected but are probably less significant in the transmission cycle because mature adult cestode development and the potential for egg shedding is less likely than in canids (Deplazes et al., 2017; Avcioglu et al., 2018; Knapp et al., 2018). Infection in the definitive host is usually asymptomatic (Davidson et al., 2012). The prepatent period in canids is about 4-5 weeks following infection and then adult tapeworms survive for about 100 days, potentially producing eggs every day (Toth et al., 2010).

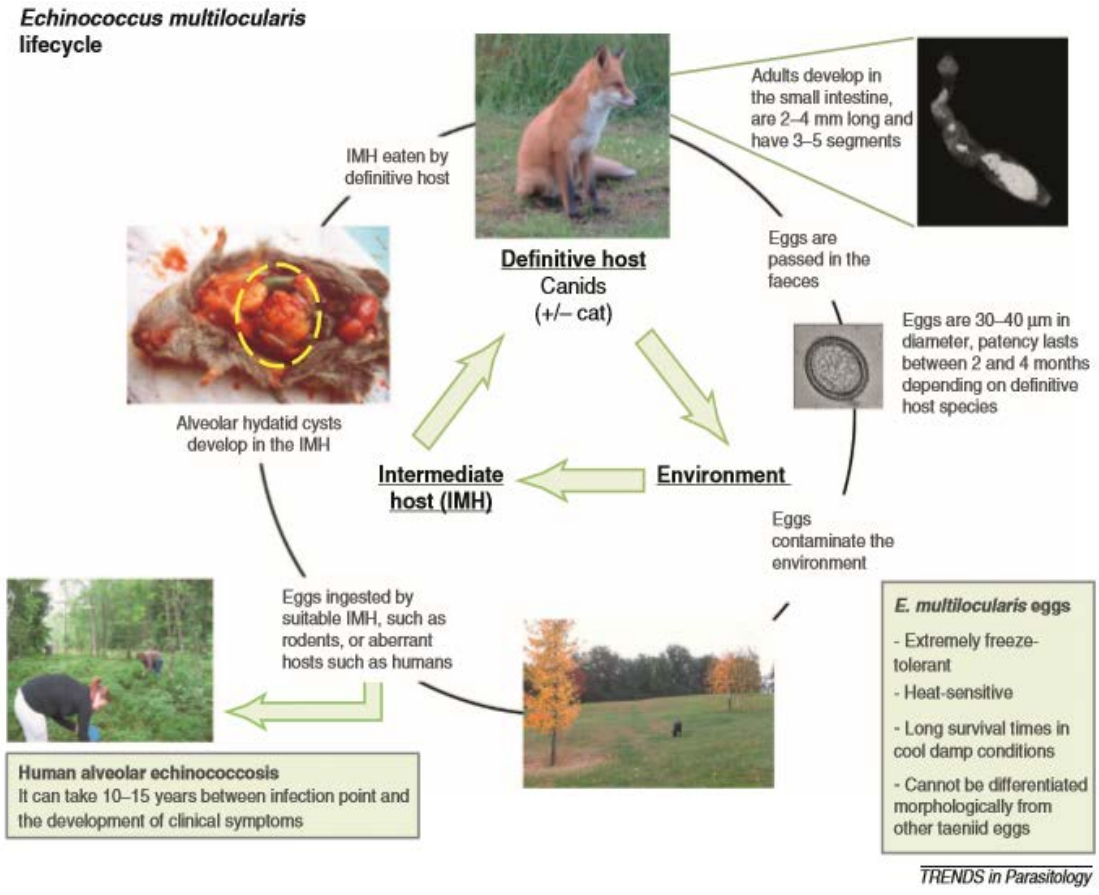

Figure 2 The transmission cycle of Echinococcus multilocularis (Source: Davidson et al., 2012)

Intermediate hosts in Europe have been shown in metastudies by Oksanen et al. (2016) and Takeuchi-Storm et al. (2015) to be primarily Cricetidae species (voles) and the muskrat with a distribution of prevalence in most countries similar to that in the definitive host, the red fox, albeit at lower levels of prevalence. However, the role of the muskrat in transmission is still not well understood (Deplazes et al., 2017). The coypu and murids may, in addition, contribute to the transmission cycle in areas with medium to high prevalence in foxes (Oksanen et al., 2016). Infection has also been reported in the European brown hare (Chaignat et al., 2015). The main arvicoline hosts in Europe are the common vole (Microtus arvalis) and water vole with the bank vole and Apodemus species of less importance (Miller et al., 2016). Takeuchi-Storm et al. (2015) proposed that this variation may be a consequence of habitat preference, with the bank vole and Apodemus species preferring wooded environments with reduced predator-prey encounters. However, experimental studies by Woolsey et al. (2016) demonstrated variations in intermediate host susceptibility, suggesting that the transmission capability of the common vole and field vole is high; that the bank vole has limited potential and that the house mouse probably plays no significant role in transmission. In Sweden, where the common vole is not found, the field vole is believed to act as the main intermediate host (Miller et al., 2017). Unusually, dogs may be infected as both definitive and intermediate hosts (Romig et al., 2017).

E. multilocularis ova are shed in the faeces of infected definitive hosts and ingested in food or water by intermediate hosts. These ova develop in the intermediate host to oncospheres which pass through the intestinal wall and via the bloodstream to organs, primarily the liver, but also, occasionally, the lungs and brain where they develop into encysted larvae (metacestodes) which proliferate by lateral budding into surrounding tissues (EFSA, 2019). The cysts act in the same way as space-occupying neoplasms with the severity of disease in the intermediate host depending on the location and number of cysts (Davidson et al., 2012). The parasite lifecycle is completed when the intermediate host is predated or scavenged and the protoscolices are ingested (ibid.).

E. multilocularis ova are persistent in the environment, particularly in cool and damp conditions (Veit et al., 1995). Veit et al. (1995) tested the effect of seasonal conditions in south-west Germany and demonstrated that, in the field, E. multilocularis ova may be viable for up to 240 days in autumn conditions and 78 days in summer. Additionally, ova stored in vitro in phosphate buffered saline at 4ºC were viable for at least 478 days (ibid.). It is not known how long cysts in the intermediate host remain infectious after the host's death. It is likely to be influenced by environmental factors but is considered to be seven to ten days (Roberts, 2012).

Justification of Hazard Status

Surveillance of infection levels in the definitive host, the red fox, is the primary method of assessing distribution and prevalence levels across Europe. Prevalence in Europe is believed to be increasing, particularly in central Europe, following implementation of rabies vaccination of free-living foxes which has led to an increase in the number and density of foxes (Cirovic et al., 2012). From four countries known to be endemic in the 1980s, E. multilocularis is now found in 24 countries in Europe, with prevalence in foxes reported to

be as high as 50% (EFSA, 2019). Studies in Germany since 1995 suggest a prevalence level in foxes in Bavaria of 40.4 to 55.5% (numbers tested not reported), the highest of any region in Germany (Deplazes et al., 2017). However, even within low prevalence or non-endemic regions there may be islands of infection as genetic analysis of strains suggest that E. multilocularis may have been circulating undetected in some areas for several years (Davidson et al., 2012).

E. multilocularis was first detected in Denmark, in 2000, in a fox hit by a car on the outskirts of Copenhagen (Wahlstrom et al., 2015). As a result, surveillance in Scandinavia was increased and, in 2011, the first case in a red fox was found in Sweden, 80 km from the Norwegian border (ibid.). There is some uncertainty as to whether E. multilocularis spread into Sweden via wildlife dispersal or pet dog movements but it is now believed that the latter route is more likely (Toth et al., 2010). Since 2011, prevalences in red foxes in Sweden have been detected at levels between 0.1 and 0.9%, with burdens in individual foxes of up to 1235 tapeworms (Wahlstrom et al., 2015). Knowledge of the habitat use and migration behaviour of foxes in Sweden is limited but, given the 1600 km shared border with Norway, the risk of E. multilocularis being introduced to Norway via infected wildlife is considered high (EFSA, 2019).

However, Echinococcus multilocularis has not been detected in mainland Norway or the UK using the EFSA threshold of <1% prevalence at the 95% confidence level to date. In 2019, faecal samples from approximately 540 culled foxes were tested in Norway by PCR for E. multilocularis DNA. All were negative (Inger Sofie Hamnes, Norwegian Veterinary Institute, pers. comm). Nevertheless, Davidson et al., (2013) reported that E. multilocularis is possibly present in Norway already but at prevalence levels below the detection level of the surveillance programme. Robertson et al. (2012), reporting on the views of the Norwegian Scientific Committee for Food Safety, have suggested that E. multilocularis would probably not be detected on first introduction as up to 1200 foxes could theoretically become infected before the first case was detected based on the 1% prevalence threshold and population estimates of between 70,000 and 120,000 foxes in Norway.

The risk of E. multilocularis being introduced to Norway via pet dog movements as a result of poor worming compliance, infrequent border checks and the risk from the illegal pet trade is also considered high (Davidson and Robertson, 2012; Davidson et al., 2012). In addition, owners may be given incorrect advice on appropriate anthelmintic treatment prior to bringing dogs into Norway: in a phone survey of 90 veterinary practices across Europe in 2011, only 10 gave correct and complete advice on the required treatment (Davidson and Robertson, 2012). In 2009, prior to proposed changes in import requirements for pet dogs entering the UK from other EU countries, Torgerson and Craig (2009) predicted that, without compulsory praziquantel treatment, there was a 98% chance for every 10,000 dogs making short trips from the UK to Germany that one would be infected with E. multilocularis on return to the UK. The current requirement is that dogs entering the UK from other countries, with the exception of Norway, Finland, Malta and the Republic of Ireland, must receive appropriate tapeworm treatment between 24 and 120 hours (one to five days) prior to entry and again 28 days after entry (DEFRA 2020). The levels of compliance and stringency of border checks is unknown.

E. multilocularis may also be spread by wild canids to potential intermediate hosts in captivity. In 2005 a Barbary macaque (Macaca sylvanus), recently imported from southern Germany, died in a zoological collection in the UK and was found on post-mortem examination to be infected with E. multilocularis (Boufana et al., 2012). The colony which the macaque had been translocated from was in a park from which foxes were rigorously excluded and it was concluded that the source of infection was contaminated foliage (ibid.). Boufana et al. (2012) reported that free roaming red foxes in zoological gardens in Switzerland have been implicated as the source of infections of captive primates in Switzerland. Additionally, a captive-born coypu in a wildlife park in France died in 2011 followed by several ring-tailed lemurs (Lemur catta) from echinococcosis, showing the risks posed to captive animals by free-living foxes even in fenced enclosures (Umhang et al., 2016). However, captive intermediate hosts are unlikely to perpetuate the transmission cycle as there is little risk that their carcasses could be scavenged after death.

Reports of infections of beavers suggest the beaver has potential to act as a competent intermediate host for E. multilocularis transmission: E. multilocularis infestation has been confirmed in free-living beavers in Switzerland (Janovsky et al., 2002), Serbia (Cirovic et al., 2012) and Austria (Posautz et al., 2015). Additionally, Gottstein et al. (2014) reported that beavers exhibit only limited humoral response to infection, which may suggest that they are particularly susceptible. Following the death associated with E. multilocularis infection of a captive beaver in England, previously wild-caught in Bavaria, the prevalence of E. multilocularis in beavers in Bavaria has been estimated to be between 2.5% and 5% (Barlow et al., 2011). However, this estimate is based on hunters' visual assessment of culled beaver livers and not on formal testing so the true prevalence may be higher. Because beavers are intermediate hosts they cannot transmit E. multilocularis to other beavers or intermediate hosts, directly or indirectly via the environment (Roberts, 2012). It is not known how long beavers can survive following infection with E. multilocularis. The case reported by Barlow et al. (2011) was of a beaver found dead in England, presumed to be as a result of E multilocularis associated disease, three and a half years after it had been imported. A female beaver, recently imported to England from Bavaria, was euthanased following a positive serological test for E. multilocularis in 2017 (Britton and Barlow, 2019). The cases reported from Serbia and Switzerland (Cirovic et al.; Janovsky et al.) were of beavers that had died in road traffic accidents. Infection with E. multilocularis may have contributed to morbidity in these animals but was not considered to be the cause of death.

Beavers for translocation may be free-living animals sourced from either Norway or Great Britain. As Echinococcus multilocularis may now be present in Norway, albeit at low prevalence levels, and beavers are known to be susceptible to infection, translocation from Norway should be considered to present a potential source hazard. Free-living beavers in Great Britain are of uncertain origin. As discussed previously, some are known to have escaped from captive facilities and others may have been deliberately released. The limited genetic testing that has taken place to date has indicated that at least some of the free-living beavers in Great Britain are of Bavarian origin i.e. from an area known to be endemic for E. multilocularis. As there is no reliable method of screening for E. multilocularis infection in intermediate hosts, there is a possibility that beavers were

infected prior to translocation to Great Britain and could present a source hazard to species at the destination site(s). If an infected beaver had been predated or died and been scavenged by a potential definitive host, the possibility of low-level prevalence of Echinococcus multilocularis in potential source areas in Great Britain cannot be ruled out. As a result, free-living beavers from both Great Britain and Norway should be considered to present a potential source hazard.

Risk Assessment

Release assessment

Beavers are exposed through ingestion of ova in food or water, which are resistant in the environment. The likelihood of exposure of beavers in Norway is low because the prevalence of E multilocularis in infected definitive hosts in Norway is very low (Davidson et al. 2013). The likelihood of exposure of free-living beavers in Great Britain is very low because (i) although adult beavers may originate from geographic areas with infection (for example, Bavaria), they do not transmit infection to the next generation and (ii) E. multilocularis has not been detected in the fox population in Great Britain. In addition, the prevalence of E. multilocularis in beavers in Bavaria, an endemic area with reported prevalence in foxes of approximately 50%, was estimated at 2.5 to 5% in 2011 i.e. beavers exhibit prevalence at substantially lower levels than in the definitive host population. Given the absence of a barrier between Sweden and Norway, the presence of E. multilocularis in red foxes in Sweden, the possible presence of E. multilocularis in Norway without detection, the large population of red foxes in Norway, the likelihood of a Norwegian beaver being exposed and infected is higher than a beaver in Great Britain. Infection occurs when the oncospheres pass through the intestinal wall and therefore, once exposed, there is a high likelihood of infection.

Exposure assessment

Infected released beavers may die and be consumed by potential definitive hosts. There is a high density of foxes throughout England and therefore the likelihood of ingestion by a fox is high. Infection of foxes occurs when they ingest the protoscolices in the beaver intermediate host. Infected foxes will excrete ova in their faeces and these ova may be ingested by beavers and other intermediate hosts such as voles. There is a high density of intermediate hosts in England. There is a high likelihood of infection of definitive and intermediate hosts at the destination. Dissemination will occur as the life cycle of the parasite repeats and there is therefore a high likelihood of dissemination.

Humans are intermediate hosts, and fieldworkers, particularly those working at the release location, could be exposed through contact with excreted ova in the environment, in the same way as other intermediate hosts above.

Consequence assessment

There is a very low likelihood of one beaver being infected at the release site.

Beavers are intermediate hosts and the effect on their health depends on the location and number of cysts (Davidson et al., 2012). There is a negligible likelihood of disease in beavers and of biological and economic consequence to the reintroduction programme.

Humans are intermediate hosts and chronic, severe disease occurs as a result of cyst formation which is potentially fatal (WHO, 2020). The consequences of infection in humans are therefore severe. It is assumed that if E. multilocularis were to enter Great Britain it would be very difficult to eradicate due to the high numbers and densities of intermediate and definitive hosts. If the human infection rate were similar to Germany and France, where E. multilocularis is endemic, that could equate to 10 to 20 cases per year (DEFRA, 2014). There is therefore a high likelihood of economic costs through the diagnosis, treatment, public health awareness, and other medical costs associated with the detection of disease in humans.

Risk estimation

There is a low probability that Norwegian free-living beavers will be exposed, a very low probability that free-living beavers in Great Britain will be exposed and a high risk of infection with E. multilocularis at both these source sites. There is a high likelihood of exposure and infection of definitive and intermediate hosts at the destination and a high likelihood of dissemination. There is a negligible likelihood of disease in beavers and biological or economic costs to the reintroduction programme. The consequence of disease in humans is severe. There is a high likelihood of economic costs from surveillance and monitoring of the human population plus public awareness campaigns. The risk from the translocation of Norwegian beavers is higher than for free-living beavers from Great Britain. The overall risk is HIGH.

Risk Management

Risk evaluation

Preventative measures should be considered for any free-living beavers being translocated within Great Britain or from Norway.

Risk management options

Ante-mortem diagnosis in the intermediate host is considered challenging and in humans is usually based on mixed modalities combining imaging with serology (Campbell-Palmer et al., 2015a). Campbell-Palmer et al. (2015a) trialed the effectiveness of combined laparoscopy and ultrasonography under general anaesthesia in the field in screening beavers for echinococcosis and achieved reported sensitivity and specificity of 100% (n=45) though the authors acknowledged that the protocol may not be effective in picking up small lesions in early infections. A serological immunoblotting technique has reported sensitivity of 85% and specificity of 100% (Gottstein et al., 2014; Gottstein et al., 2019) but is not suitable for field use as results are not immediately available (Campbell-Palmer et al., 2015a). Blood sampling could be performed on a conscious beaver with restraint and without the need for general anaesthesia.

There will be advantages in using free-living beavers proven to have been born in Great Britain to reduce the risk from Echinoccocus multilocularis.

Treatment for intermediate hosts is limited, and often unsuccessful, requiring surgical resection and prolonged treatment with benzimidazoles (Wen et al., 2019).

Appendix 11 Disease Risk Analysis for the Source Hazard Trichinella species

Trichinella species are parasitic nematodes, currently comprising nine species and 4 genotypes with variations in host and geographic preferences, and a major historic cause of zoonotic infections and economic losses in Europe (Pozio, 2020). The nematode is unusual in that it undergoes a complete life cycle, from larva to adult to larva, in a single host animal (Pozio et al., 2019) but requires a second host to perpetuate its life cycle (Figure 3). There were 66 confirmed cases of trichinellosis in humans in Europe in 2018, compared to 324 in 2014, of which cases in Bulgaria and Romania accounted for 83% (n=55/66), and major efforts continue in Europe to reduce and eradicate Trichinella species from domestic livestock (EFSA, 2019).

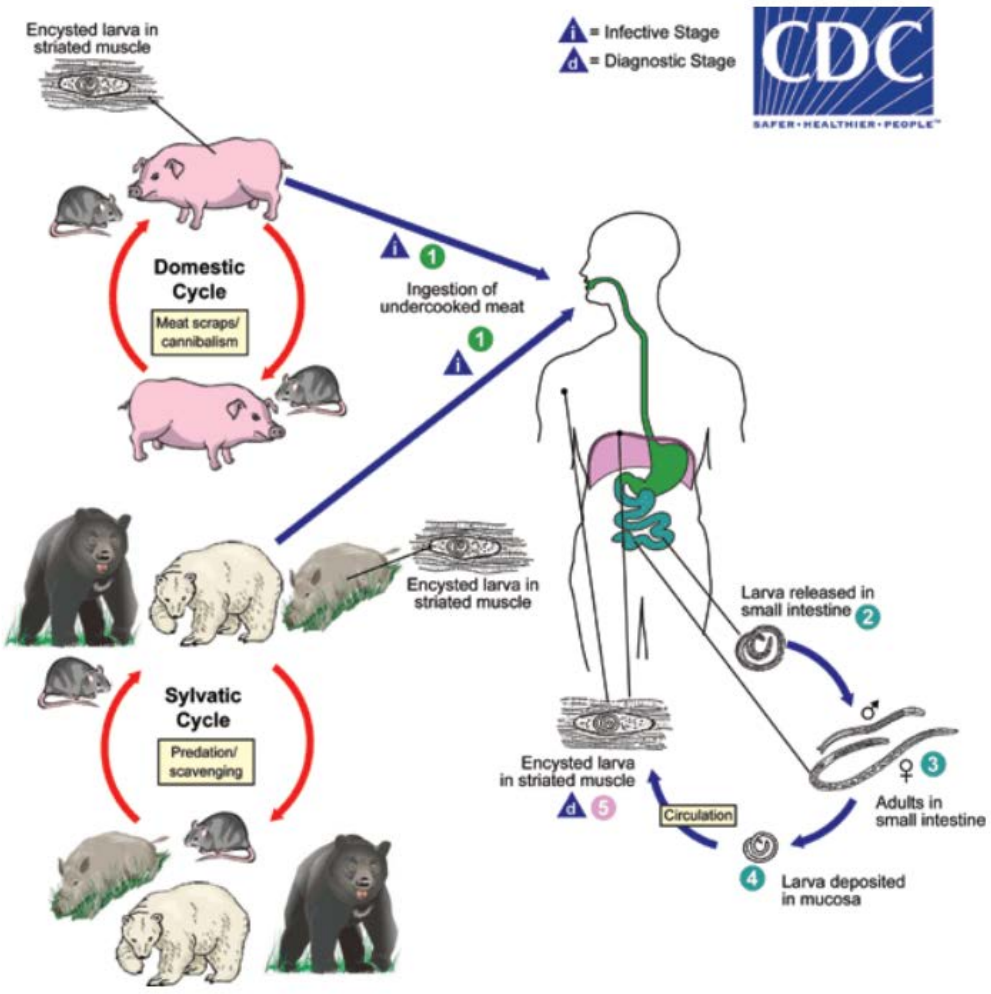

Figure 3 The life cycle of Trichinella species (Source: CDC, 2020)

Trichinella species have a broad host range and infections have been reported in over 150 mammalian species, across 12 orders, as well as in birds and reptiles (Pozio, 2019). However, humans are probably the only mammals to experience clinical disease,

trichinellosis; host animals ingesting large numbers of infective larvae have not been reported to exhibit symptoms (Gottstein et al., 2009). Trichinellosis is a disease of varying severity in humans, usually as a result of eating undercooked or raw pork products containing Trichinella species larvae from both domestic pigs and wild boar (ibid.). Vertical transmission has also been demonstrated experimentally in ferrets, guinea pigs (Cavia porcellus) and mice but not in foxes or pigs (Webster and Kapel, 2005). The highest proportion of Trichinella species infections in humans are of Trichinella spiralis, but infections with other Trichinella species, including T. britovi, T. nativa and T. pseudospiralis, have also been reported (Bronstein and Lukashev, 2018; Ranque et al., 2000).

In the domestic environment, pigs are infected when management and welfare standards are low, for example, by scavenging infected carcasses and through tail biting (Pozio, 2000). There was a notable resurgence of infections in Eastern Europe following the break-up of the former Soviet Union and the resultant increase in small-scale farming with reduced veterinary supervision (ibid.). The infectious agent in pigs is usually T. spiralis which also exists in a sylvanian cycle in Europe in areas where it has been eliminated from domestic livestock (ibid.). Klun et al. (2019) investigated Trichinella species infections of eight species of wildlife in Serbia over a 20 year period from 1994 and found that nearly half of all infected animals (n=14/29) were infected with T. spiralis.

In addition, there are sylvanian cycles of other Trichinella species, of which T. britovi is the most prevalent (Pozio, et al., 2009), with geographic distribution governed by ambient temperature. T. nativa is typically the prevalent species in arctic and subarctic regions, including Norway and Sweden, and T. britovi in temperate regions, including Germany and Sweden (Pozio, 2000). However, there is considerable overlap in the geothermal divisions: Chmurszynska et al. (2013) found infection with T. nativa in three red foxes in Germany, 1200 km south of the perceived boundary between T. nativa and T. britova. The authors concluded that, as it was unlikely that the animals had migrated such a considerable distance, sylvanian cycles of T. nativa may be maintained in temperate regions.

T. pseudospiralis is the only Trichinella species known to infect both birds and mammals and is rarely reported in wildlife: 1.6% (n=63/3925) of isolates in European wildlife were confirmed as T. pseudospiralis between 2007 and 2014 (Pozio, 2016a). However, the prevalence of T. pseudospiralis in wildlife may be underestimated due to the limited sampling of birds compared to mammals (Learmount et al., 2015). In addition, unlike the other Trichinella species of interest in Europe, encysted T. pseudospiralis larvae lack a surrounding collagen capsule, making visual diagnosis using trichinoscopy almost impossible historically (Pozio, 2016). Although the environmental survival of T. pseudospiralis is poor compared to other Trichinella species, its broad host range, and bird migration and dispersal, may perpetuate sylvanian transmission cycles and geographic range expansion (ibid.). The perpetuation of sylvanian cycles of all Trichinella species is facilitated in areas where hunters leave animal carcasses for other animals to scavenge (Pozio, 2009).

There is occasional spillback and spillover between the domestic and sylvanian cycles, probably facilitated by foxes, rats and domestic cats, particularly when pigs are housed outdoors or are fed hunters' scraps (Pozio, 2019). However, Kapel (2001) demonstrated experimentally that wild boar are not particularly susceptible to infection with T. nativa, with rapid declines in antibody levels shown to be associated with the disappearance of larvae from muscle tissues and it is believed that domestic pigs are similarly resistant to infection with T. nativa . Additionally, infections of T. britovi in swine are reported to be short-lived, with larvae surviving for less than one year in pig muscle, but reports of occasional infections of swine in the Baltic states suggest that animals immunosuppressed by stress and hunger or concurrent infection may, on occasions, be susceptible to infection with T. nativa (Pozio, 2019). T. pseudospiralis has been rarely reported in domestic swine in Europe (Pozio, 2016a).

Justification of Hazard Status

After several years of declining prevalence of Trichinella species infection in wildlife in the European Union (EU), small increases were reported in 2018 (EFSA, 2019). However, surveillance is not standardised across member states and not all member states submit reports. A number of wildlife species are screened for Trichinella species infection, primarily the red fox and wild boar, with prevalences in 2018 respectively 1.6% (n=108/6612) and 0.09% (n=1,293/1,465,482) across 14 member states (ibid.). Infections were also reported in a further 10 species, with highest prevalences in the Eurasian lynx (Lynx lynx), wolf and raccoon dog (ibid.). As the population levels of these three species are low when compared to the red fox, they are not currently considered to be a significant reservoir of infection in the sylvanian cycles but this may change with increasing population numbers and distribution of these species and the European jackal (Canis aureus morioticus) (Pozio, 2019).

The United Kingdom is currently considered to be free of Trichinella species with 6,976,629 farmed pigs, 581 wild boar, 360 red foxes and 2,771 horses screened negative in 2018 (EFSA, 2019). The last reported wildlife case of T. spiralis infection in Great Britain was of a red fox from Truro, Cornwall sampled in 1957 (Oldham and Beresford-Jones, 1957) although more recent cases of a single fox infected with T. spiralis were reported in 2007 and 2009 in Northern Ireland (Learmount et al., 2015). In 2013, T. pseudospiralis was identified by artificial digestion and PCR in a red fox found dead following a road traffic collision near Bristol (ibid.). As this was an isolated case (n= 1/6806 red foxes sampled between 1999 and 2013 in Great Britain), Learmount et al. (2015) concluded that the prevalence of T. pseudospiralis in Great Britain is extremely low and the associated risk negligible.

As transmission is reliant on ingestion of animal carcases infested with larvae, infections are found primarily in carnivorous or omnivorous animals. However, infection of herbivorous animals, including horses, is also reported (EFSA, 2019). Grzybek et al. (2019) screened three free-living populations of bank voles at three intervals between 2002 and 2010 in Poland for Trichinella species antibodies and found an average prevalence of infection with unspecified Trichinella species of 1.37% (n=656). Infection

probably occurs as herbivores inadvertently ingest larvae while foraging for food near carcasses, consume carrion or from cannibalism (ibid.).

Infections with T. britovi and T. spiralis have been reported in beavers: 1/182 beavers killed by hunters in Latvia between 2010 and 2014 was positive for T. britovi with 148 larvae identified in a muscle tissue sample of approximately 25g (Seglina et al., 2015); a single T. spiralis larva was found in a tissue sample from one of 69 beavers hunted in Poland in 2018 (Rozycki et al., 2020); a young woman was admitted to hospital in Russia in 2017 with acute abdominal pain and fever, later diagnosed as trichinellosis, following consumption of beaver meat two days earlier (Bronstein and Lukashev, 2018). There have been no reports, as far as we are aware, of infection with Trichinella species in beavers in Norway or Sweden. However, T. nativa larvae were found in 4.6% (n=393) of culled red foxes and T. britovi larvae in one fox (of 393 sampled) in Norway between 1994 and 2005 (Davidson et al., 2006) and, according to the database of the International Trichinella Reference Centre in Rome, T. spiralis has been found in foxes in Sweden (Pozio, 2019). As beavers may share territory with red foxes and have been shown to be susceptible to infection with Trichinella species, Trichinella species should be considered as a source hazard as a result of the translocation of beavers from Norway.

Risk Assessment

Release assessment

Beavers may be infected through accidental ingestion of Trichinella species larvae from carcasses of sympatric species. In addition, it has been speculated that, in common with other herbivores, beavers may, on occasions, intentionally consume animals as the liver parasite, Capillaria hepatica, and fish parasite, Paragonimus westermani, have been rarely detected in beavers (Bronstein and Lukashev, 2019). Following ingestion, larvae penetrate the intestinal mucosa where they complete their development to adulthood (Gottstein et al., 2009). Adult nematodes mate and, five days after infection, release larvae which migrate via blood and lymphatic vessels to striated muscle tissue where they complete their development to the infective stage and then enter a dormant state until the host animal is predated or dies (ibid.). As the first stage of the lifecycle is completed quickly, larvae successfully evade the host's immune system but adult nematodes are expelled and no further reproduction takes place in the host unless further infective larvae are ingested (ibid.). As a result, an animal ingesting only low numbers of larvae is likely to have only low infectivity potential.

Release assessment for free-living beavers translocated from Norway. Since T. nativa and T. britovi have been found in red foxes in Norway, and T. spiralis in red foxes in neighbouring Sweden, and infections, of very low prevalence, with T. britovi and T. spiralis have been reported in beavers in other countries, there is a very low probability that beavers translocated from Norway could be infected with low levels of Trichinella species larvae.

Release assessment for free-living beavers translocated from Great Britain. As both T. nativa and T. britovi have been found in red foxes in Norway and Germany, infections with T. britovi and T. spiralis have been reported in beavers, there is a very low probability that beavers previously imported into the UK from these countries were infected with Trichinella species prior to capture. However, since beavers have been imported, with the exception of an isolated case of T. pseudospiralis infection in a red fox in the Bristol region in 2013, infection has not been detected in the red fox population in Britain. There is therefore a very low likelihood that an adult beaver, previously imported from an area with endemic Trichinella species infection, translocated to England will be infected.

Vertical transmission from parent to foetus has been demonstrated in rodents so there is a very low probability that an infected female beaver could have transmitted Trichinella species infection to its offspring. However, the maximum larval burden in offspring from experimentally infected guinea pigs was 60 larvae and, in mice, six larvae (Webster and Kapel, 2005) so the infective burden, if any, is likely to be very low. There is therefore a very low likelihood that a beaver born to an infected dam, previously imported from an area with endemic Trichinella species infection, translocated to England will be infected.

As the number of free-living beavers in Great Britain is low and some carcasses have been retrieved for post-mortem examination, there is a very low probability that a previously imported, infected, free-living beaver in Great Britain has been predated or scavenged, thereby infecting a sympatric carnivore(s) in Great Britain. There is a negligible likelihood that Trichinella species larvae from a carnivore infected in this way have been ingested by a sympatric beaver as there has only been one isolated case of Trichinella infection in a red fox in Great Britain and this was in an area not known to be inhabited by beavers. Additionally, the prevalence in beavers has been shown to be low even in an area with high prevalence in an endemic sylvanian cycle (Grzybek et al., 2019; Bakasejevs et al., 2012).

There is therefore a very low likelihood that a free-living beaver in Great Britain, translocated to England, is infected with Trichinella species.

Exposure assessment

There is a high likelihood that a sympatric carnivore or omnivore is infected by predating an infected beaver. In addition, there is a high likelihood that a sympatric carnivore or omnivore would be infected by scavenging the carcass of an infected beaver as the larvae of most Trichinella species are encapsulated in muscle tissues which facilitates prolonged survival in the environment following the death of the host animal (Pozio, 2000). The duration of larval survival is greatest between 0 and -20ºC and at higher humidity levels, with T. nativa shown to remain infective after five years of freezing and T. britovi after just less than one year (Pozio, 2019). Larvae survive longer in frozen carnivore carcasses than in swine and rodents for reasons which are not well understood (Pozio, 2016b). In addition, Davidson et al. (2008) demonstrated that T. nativa is highly tolerant to repeated freezing and thawing with larval survival after seven events comparable to unthawed larvae.

Encapsulated larvae can also survive up to four months after muscle tissue has decayed and so may constitute a source of environmental infection to herbivorous animals (Pozio, 2000). There is therefore a very low likelihood that herbivores such as deer and horses, as well as other beavers, at the destination site(s) are infected through accidental ingestion of infective larvae on plant matter or through deliberate scavenging of infected carcasses.

The establishment of sylvanian Trichinella species cycles in Europe is facilitated by hunter activity and the survival of encapsulated Trichinella species larvae in carcasses is temperature and humidity dependent with optimum survival between 0 and -2ºC. As the average winter temperature low in England is 0.9ºC (Met Office, 2020) and sport hunting is less common than in Europe, there is a lower likelihood of Trichinella species establishing in sylvanian cycles in Great Britain compared with the same cycles on the continent. The probability of dissemination of Trichinella species through the establishment of a sylvanian cycle is therefore very low.

40% of domestic pigs are kept outdoors in Great Britain (ADHB, 2020). A pig could be infected with Trichinella species if it scavenged the carcass of an infected animal. Small rodents act as vectors between sylvanian and domestic cycles in Europe and a domestic pig could be infected if it scavenged an infected rodent. However, the likelihood of dissemination through the domestic pig population is very low as pigs are not routinely fed hunters' scraps in the UK and are kept in fenced enclosures. In addition, both T. nativa and T. britovi appear to have short survival times in swine. The probability of dissemination through the domestic cycle is very low.

There is a very low likelihood that humans are infected by eating undercooked meat from an infected animal if Trichinella species enters either the domestic or sylvatic cycles through one of the mechanisms above.

It is interesting to note that beaver hunting, both licensed and unlicensed, is widespread across Europe and beaver meat is considered healthy and a great delicacy in Eastern Europe (Bronstein and Lukashev, 2018). There is therefore a very low probability at release sites of illegal hunting of free-living beavers for consumption.

Consequence assessment

There is a very low likelihood of one translocated beaver being infected.

Following ingestion of Trichinella species larvae in raw or undercooked meat, disease in humans may range from asymptomatic to more severe illness including fever and gastroenteritis as larvae migrate through the intestinal mucosa. In severe cases, encephalitis and secondary infections may occur (Davidson et al., 2009) and one third of human cases may require hospitalisation (Pozio, 2019). The severity of disease in humans is believed to be dependent on the infective dose ingested and may be more severe with T. spiralis than with other Trichinella species (Gottstein et al., 2009). The lowest dose associated with disease in humans is not known but is believed to be over 100 larvae (ibid.). Ingestion of more than 1000 larvae is believed to be associated with severe

symptoms in humans (Davidson et al., 2009). There is a high likelihood of severe disease in humans.

The economic impact of trichinellosis in countries where the parasite is endemic in domestic pigs is considerable due to the cost of control systems in abattoirs which was estimated at 3USD per pig in the EU in 2000, checks on wildlife, the commercial value of wasted carcasses, and medical costs associated with treating human infections (Pozio, 2000). The consequence of Great Britain losing its Trichinella-free status is therefore high. The biological and economic consequences of disease in humans is high.

Evidence noted above indicates humans are the only animals which seem to experience clinical signs following infection with Trichinella species. There is therefore a negligible likelihood of clinical disease in infected beavers and a negligible likelihood of translocation failure as a result of Trichinella species infection of beavers.

Risk estimation

There is therefore a very low likelihood that a free-living beaver in Great Britain or Norway, translocated to England, is infected with Trichinella species. However, the likelihood is lower for beavers from Great Britain because infection from previously imported beavers has not been detected in the red fox population in Britain. There is a high likelihood of exposure and infection of sympatric carnivores and omnivores at the destination site(s) and a very low likelihood of exposure and infection of herbivores. The likelihood of onward transmission and dissemination into a sylvatic and/or domestic cycle of infection is very low. There is a very low likelihood of exposure and infection of the human population following dissemination into the domestic or sylvatic life cycles. There is a negligible likelihood of translocation failure and biological and economic consequences from that failure. There is a high likelihood of severe disease in humans and of severe economic and biological consequences as a result of disease in humans and domestic livestock. The overall risk is MEDIUM.

Risk Management

Risk evaluation

Steps should be taken to minimise the risks to humans and livestock from the source hazard Trichinella species.

Risk Management Options

Detection of immature Trichinella species larvae in carcasses by muscle digestion is the gold standard of diagnosis but is time-consuming and costly (Davidson et al., 2009). Serology, in combination with western blot for crude larval antigen, demonstrates comparable sensitivity but may not be a reliable method of diagnosis: seroconversion to detectable levels in animals with low levels of infection may take up to seven weeks and some animals, for example horses, do not appear to seroconvert despite high larval burdens (ibid.). Additionally, haemolysis or contamination of field samples may

significantly reduce the sensitivity and specificity of tests (ibid.). Efficacy of serological testing has not, as far as we are aware, been demonstrated in beavers but testing is unlikely to be sufficiently sensitive as the prevalence of Trichinella species larvae in beavers is low, even in endemic areas.

Sourcing beavers from Great Britain, particularly those proven to have been born in the UK, is more likely to be effective in minimising the risk of translocating a beaver infected with Trichinella species.

Post-mortem examination of translocated beavers and sympatric species is strongly recommended to assess for entry of Trichinella species into the UK. Additionally, farmers and hunters at the destination sites should be reminded of the importance of appropriate carcass removal and disposal following pest control.

Appendix 12 Disease Risk analysis for the Carrier and Population Hazard Toxoplasma gondii

Toxoplasma gondii, of the phylum Apicomplexa, is an obligate intracellular protozoan which is ubiquitous worldwide (Herrmann et al., 2013; Tenter et al., 2000). The parasite has an indirect life cycle; the sexual phase occurs only in felids, but the asexual phase is possible in almost any mammalian intermediate host (Herrmann et al., 2013). In felids, the infectious phase of toxoplasma is the sporozoite, which occurs in oocysts. Toxoplasma gondii has two forms in intermediate hosts: tachyzoites and bradyzoites (found in tissue cysts). The initial acute period of infection occurs when an intermediate host ingests sporozoites from an oocyst, or bradyzoites from a tissue cyst. These then convert to tachyzoites within the intestinal epithelium of the intermediate host and begin to rapidly replicate by asexual reproduction. These tachyzoites spread throughout the body via the bloodstream, leading to systemic infection. At this stage, in most cases the host immune response leads to clearance before clinical signs develop (Suzuki et al., 1988). However, tachyzoites can convert to dormant bradyzoites within tissue cysts as an immune evasion mechanism. Tissue cysts form more often in muscular and neural tissue such as the brain, eye and cardiac muscle, but can also be found in the lungs, liver and kidneys (Hill et al., 2005). During periods of host immunocompromise, tissue cysts can rupture, and bradyzoites can recrudesce to become tachyzoites again. This can lead to acute toxoplasmosis (Shen et al., 2016; Skariah et al., 2010).

Carrier Hazard - Justification of Hazard Status

Exposure of American beavers to T. gondii has been reported in several studies. A serological survey was undertaken across several free-living mammals in Missouri, USA, in which 14 American beavers were sampled. One beaver had a positive antibody titer and T. gondii was later isolated from this animal. Several other rodents tested positive using serology, including one woodland white-footed mouse (Peromyscus leucopus), seven muskrats and two grey squirrels. T. gondii was also isolated from one of these two grey squirrels (Smith and Frenkel, 1995). Furthermore, a T. gondii seroprevalence of approximately 10% (n=6/62) was reported in a population of American beavers in Massachusetts, USA (Jordan et al., 2005). American beavers are also susceptible to disease associated with T. gondii. A five month old free-living beaver found orphaned in Connecticut, USA, died of severe systemic toxoplasmosis, confirmed using immunohistochemistry, after spending 14 weeks at a rehabilitation facility (Forzán and Frasca, 2004). It is unknown whether this animal was exposed before or after admission to this facility, but numerous cysts in the cerebral and cerebellar tissue containing bradyzoites suggest that the infection may have been chronic, and acute infection may have occurred after immunosuppression and reactivation of dormant disease. This provides further concern about the impact of captivity and stress on free-living beavers with chronic toxoplasma exposure.

Exposure of Eurasian beavers to T. gondii has also been reported. Six free living adult beavers found dead around the River Havel, Germany, between 2006 and 2011 were sampled for T. gondii using PCR. Two animals tested positive; one of these had histopathological evidence of tissue cysts in the brain along with a moderate to severe inflammatory response which suggested toxoplasma- associated encephalitis as the cause of death in this animal (Herrmann et al., 2013).

T. gondii has been shown to be present in Norway. A seroprevalence of 10.9% (n=3907) was found in pregnant women in a survey undertaken in 1992 (Jenum et al., 1998). Another study into prevalence in free-living Norwegian cervids showed a seroprevalence of 33.9% (n= 258) in roe deer, 12.8% (n=270) in moose (Alces alces), 7.7% (n= 44) in red deer and 1% (n = 87) reindeer (Rangifer tarandus) (Vikøren et al., 2004). More recent data suggests that T. gondii is currently prevalent across Europe. Information provided to the European Food Safety Authority and European Centre for Disease Prevention and Control (EFSA and ECDPC) by countries including Norway and the UK in 2017 showed seroprevalence across Europe to be between 13 and 30% in small ruminants. A prevalence of 10.5% was reported in cattle, although no data was provided by Norway, and seven cases of congenital toxoplasmosis were described in the UK (European Food Safety Authority and European Centre for Disease Prevention and Control, 2018). As translocation is a known stressor (Dickens et al., 2010) and given the prospect of exposure and chronic infection with T. gondii in Eurasian beavers from Norway or the UK, it is possible that translocation of beavers could lead to acute toxoplasmosis as a result of resurgence of chronic disease under stressful conditions. Therefore, Toxoplasma gondii should be considered as a carrier hazard for the translocation of beavers.

Risk Assessment

Release assessment

The most likely route of exposure to T. gondii for beavers is direct ingestion of sporulated oocysts shed into the environment by infected felids, for example in drinking water or on vegetation. Toxoplasma oocysts have been reported to be able to survive for between 1.5 and 4.5 years in soil and fresh water environments and in sea water for several months (Aramini et al., 1999; Bowie et al., 1997; Dubey, 1998; Frenkel et al., 1975; Jordan et al., 2005; Lindsay et al., 2003; Tenter et al., 2000. Prolonged survival in fresh-water environments suggests that aquatic mammals, such as beavers, may be at particular risk of exposure (Herrmann et al., 2013) and there is a medium probability of exposure of all free-living beavers. Vertical transmission is also possible (Parameswaran et al., 2009) and has been shown experimentally in other rodents such as house mice and field mice (Owen and Trees, 1998) suggesting it may also be possible in beavers.

Considering the ubiquity of the parasite across Europe, its ability to survive for long periods of time in aquatic environments, and the previous detection of infected Eurasian beavers, the probability of beavers being infected with T. gondii at the source is estimated to be medium. There is a medium likelihood of beavers being chronically infected with T. gondii when translocated.

Exposure assessment

Infected beavers will carry the protozoa to the destination but will not contribute to the burden of T. gondii sporozoites in the environment at the release site as only felids shed T. gondii sporozoites in faeces. However, infected beavers could represent a source of infection for species which prey on rodents such as red foxes (Pavey et al., 2008) or scavenger species. There is therefore a low likelihood of exposure of beavers and other mammals at the reintroduction site.

The reintroduction itself is predicted to have little influence on the host-parasite dynamics at the destination site since T. gondii is already prevalent in the environment across Europe. The likelihood of dissemination at the destination site because of beaver reintroductions is negligible.

Consequence assessment

The probability that at least one beaver is chronically infected with T. gondii at the time of translocation is medium. There is a medium likelihood that the conditions of translocation will lead to an alteration in host-parasite dynamics resulting in immunocompromise and recrudescence of chronic toxoplasmosis leading to acute disease. In cases of acute clinical disease, consequences are likely to be severe for the individual with a high likelihood of death. There is a medium probability of biological and economic consequences as a result of T. gondii recrudescence under conditions of translocation stress due to failure of the reintroduction program. Since Toxoplasma gondii is widespread in the environment the likelihood of environmental and ecological consequences at the destination site is negligible.

Risk estimation

There is a medium likelihood of beavers being exposed to Toxoplasma gondii at the source site and a medium likelihood of beavers being chronically infected when translocated. The likelihood of exposure and infection of free-living species which prey on rodents is estimated to be low, and negligible in all other free-living species at the destination site. Dissemination of Toxoplasma gondii at the destination is likely to be negligible. There is a medium likelihood of at least one translocated beaver being infected and developing disease and a high likelihood of severe consequences for these individuals. There is a negligible likelihood of substantial ecological consequences at the destination, but medium likelihood of negative biological and economic consequences as a result of translocation failure in the case of recrudescence of disease under stressful conditions. The overall risk is estimated to be MEDIUM.

Risk Management

Risk evaluation

Based on the risk assessment above, management methods should be employed to reduce the risk of T. gondii to translocated beavers.

Risk management options

Disease risk management methods to reduce stress in the translocated population of beavers through good husbandry and management methods are recommended.

Serological testing for T. gondii specific IgG antibodies may be a useful tool to gauge exposure level of the population (Q. Liu et al., 2015), however it is unlikely that a high percentage of positive results will impact the decision to translocate this population since there are no records of post-release disease outbreaks of toxoplasmosis in any reintroduced mammal.

Diagnostics for this disease should be considered as part of the post release health surveillance protocol to help inform future decision making regarding this parasite.

Population Hazard - Justification of Hazard Status

Toxoplasma gondii has already been evaluated as a carrier hazard and the risk considered to be medium. The risk to translocated beavers from road traffic collisions (RTCs) has also been evaluated as a population hazard and considered to be medium. Here we analyse how chronic disease associated with Toxoplasma gondii will affect the risk from road traffic collision and/or predation to the beaver reintroduction.

Latent infection with T. gondii is known to induce behavioural changes in intermediate hosts as a result of predilection to neural tissue. This is thought to be an evolutionary mechanism of transmission to feline definitive hosts by increasing the likelihood of predation of the intermediate host (Havlícek et al., 2001). In humans, there is evidence to suggest that infection with T. gondii leads to slower reaction times (Havlícek et al., 2001) and, as a result, can increase the risk of the host being involved in road traffic collisions (Flegr et al., 2002; Galván-Ramírez et al., 2013; Gohardehi et al., 2018; Kocazeybek et al., 2009; Stepanova et al., 2017; Yereli et al., 2006).

There is further evidence to suggest that this may also be the case in other mammals. An Australian study by Hollings et al. (2013) found a higher seroprevalence of T. gondii in road-killed Tasmanian pademelons (Thylogale billardierii) (31%, n=16) than in culled individuals (11%, n=212). However, the small sample size of road killed animals compared to culled necessitates results to be interpreted with caution. Of particular interest to the beaver reintroduction are the apparent behavioural changes exhibited in rodents as a result of T. gondii infection. Berdoy (2000) found that brown rats experimentally infected with T. gondii did not exhibit normal predator avoidance when compared to controls. Although the study focused specifically on olfactory queues and avoidance of predator scent, it could be true that avoidance of other dangerous situations, such as road traffic, could also be affected if innate fear is reduced. However, others have suggested that the behavioural effects of T. gondii on an intermediate rodent host are likely to be relative to the dose of stimulus and are more likely to be specific to avoidance of feline urine (Vyas et al., 2007).

Positive serology for T. gondii has been significantly associated with low neophobia (fear of novel objects) in brown rats (Webster et al., 1994). As well as advantageously affecting the parasite by increasing susceptibility to predation by definitive hosts, Webster et al. (1994) suggest that this could lead to an increased risk of trapping and poisoning of infected rats. In addition, rats may be less likely to avoid road traffic. However, causation cannot be established from this observational study and further research is required to deduce whether T. gondii infection reduces neophobia. Moreover, the effects of T. gondii on rodent behaviour are widely disputed; a study into six infected mice found no alterations in cognitive function, anxiety levels, social behaviour or motivation to explore novel objects when compared to controls, although the small sample size reduces the reliability of these results (Gulinello et al., 2010).

Risk Assessment

Exposure assessment

Our analysis of T. gondii as a carrier hazard estimated a medium likelihood of beavers being infected when translocated. Our analysis of RTCs as a population hazard estimated a medium risk to translocated beavers. Although evidence is somewhat conflicting, the neurological/behavioural effects of T. gondii reported in other rodents as a result of the formation of tissue cysts in the brain, as well as increased risk of RTCs implied in other species, suggests that an increased risk of RTCs cannot be ruled out.

There is therefore a medium likelihood of reintroduced beavers suffering from RTCs as a result of chronic toxoplasmosis. There is also a medium probability of reintroduced beavers suffering from predation as a result of chronic toxoplasmosis.

Consequence assessment

Research suggests that chronic infection with T. gondii may cause behavioural changes that increase susceptibility to RTCs or predation which decrease survival. The probability of these events occurring in an individual beaver chronically infected with T. gondii is estimated to be medium. The probability of severe consequences in the in the case of predation or RTC is high, as mortality rates as a result of these events are likely to be high. There is a low likelihood of significant biological and economic consequences due to failure of the reintroduction program as a result of multiple deaths from chronic toxoplasmosis.

Risk estimation

There is a medium likelihood of road traffic collision in reintroduced beavers and a medium likelihood of at least one beaver being chronically infected with T. gondii when translocated. The likelihood of reintroduced beavers suffering from road traffic collision or predation as a consequence of chronic toxoplasmosis is estimated to be medium. The likelihood of severe consequences, including death, in individuals involved in these events is high. The probability of significant biological and economic consequences as a result of

RTCs or predation following chronic T. gondii infection is low. The overall risk from chronic toxoplasmosis is estimated to be MEDIUM.

Risk Management

Risk evaluation

Based on the risk assessment, preventative measures should be employed to reduce the risks of RTCs and predation from chronic toxoplasmosis.

Risk management options

Mitigation measures against RTCs have been discussed in the individual RTC DRA and also apply to RTCs resulting from chronic toxoplasmosis. This includes taking care when choosing the release site for reintroduced beavers, as well as adding warning signs to stretches of road considered a risk, to encourage safe driving.

Appendix 13 Disease Risk Analysis for the Unclassified Hazard Giardia duodenalis

Giardia species are enteric protozoan parasites with marked differences in host specificity, geographic range and host preferences (Mateo et al., 2017). Controversy over nomenclature and species identification has historically hindered investigation into the role of wildlife in the epidemiology of these parasites but is being resolved by the recent application of DNA-based molecular tools which can be used to confirm the identify of species and sub-types, and to differentiate between patent infection and the passage of non-infective oocysts (Thompson and Ash, 2019). Giardia duodenalis (syn. lamblia syn. intestinalis) is the only Giardia species found in humans (Ryan and Caccio, 2013). It is regarded as a species complex comprising at least eight assemblages, A to H, with each assemblage probably representing a distinct species due to the degree of genetic divergence (Thompson and Ash, 2019). A and B, the only assemblages known to infect humans, also infect the largest range of host species, including some domestic livestock, companion animals and wildlife (Horton et al., 2018) and it is proposed that reservoirs may be bi-directional i.e. humans may act as a reservoir of infection to animals and vice versa (Ryan and Caccio, 2013). Recognition of further genetic variation within each assemblage has led to the classification of sub-assemblages, for example, AI, AII, of closely-related isolates (Ryan and Caccio, 2013). It is not known how host-specific sub-assemblages are and it is proposed that minor nucleotide variations between isolates may reduce the potential for inter-specific transmission (Van Keulen et al., 2002).

G. duodenalis assemblage B has a higher prevalence than assemblage A in humans worldwide (Feng and Xiao, 2017) and this pattern has been observed in analysis of faecal samples from 150 human patients in the UK (Minetti et al., 2015) (67% prevalence of assemblage B, and 31% prevalence of assemblage A (all sub-assemblage AII)). However, assemblage B is reported to cause more severe symptoms in human patients than assemblage A and the higher prevalence of assemblage B may therefore be a consequence of reporting bias (ibid.). In addition, mixed infections may be under-reported in both humans and animals as PCR testing may only identify the most abundant isolate; this may also lead to missed diagnoses of isolates of relevance in some studies (Ryan and Caccio, 2013).

Transmission of Giardia species is faeco-oral by ingestion of infective cysts and trophozoites and may be direct or, more commonly, indirect via contaminated water sources or food. (ibid.). Cysts are immediately infectious following excretion and may survive several months in the environment with an infective dose of as few as 10 oocysts (ibid.). Survival of cysts increases with decreases in temperature and a small number of cysts can survive a single freeze-thaw episode (USEPA, 1999). Infection with Giardia duodenalis species is often asymptomatic and, as a consequence, they are regarded by some authors as commensal parasites (Polack and Adjou, 2020). Clinical disease, giardiasis, also known as Beaver Fever in North America, may be acute or chronic and is characterised by diarrhoea, abdominal pain, nausea and weight loss (ibid.). Variations in

individual susceptibility to disease following infection are poorly understood but, in humans, prevalence of disease is known to decrease with age (ECDP, 2019).

Justification of Hazard Status

Both Canadian and Eurasian beavers have been implicated as the source of infections in humans and domestic animals (Tsui et al., 2018; Paziewska et al., 2007; Sroka et al., 2015). Historic reports based on the presence of beaver colonies upstream from drinking and recreational water sources and experimental inoculation of humans with Giardia species isolated from Canadian beavers (Davies and Hibler, 1979) have been supported by whole gene sequencing (WGS) which has demonstrated clustering of assemblage A and B isolates in Canadian beavers, humans and domestic animals, supportive of intraspecific transmission (Tsui et al., 2018).

We are not aware of similar studies in Eurasian beavers; however, Eurasian beavers have been shown to be susceptible to infection with Giardia species. Paziewska et al. (2007) isolated Giardia species from 7.7% (n=4/52) of faecal samples from captive (n=30) and wild (n=22) beavers in a study in Poland. Additionally, PCR and sequencing have been used to identify G. duodenalis assemblages A and B in water close to beaver lodges: Sroka et al. (2015) analysed 79 water samples from 14 known beaver habitats in north-east Poland. 48.1% of these water samples tested positive by PCR for the presence of Giardia species DNA (n=38). 11 samples were successfully genotyped and identified as G. duodenalis assemblage A (n=3) and G. duodenalis assemblage B (n=8). In addition, the density of Giardia cysts significantly declined with increasing distance from the beavers' lodges suggesting that beavers rather than other animals were the source of the cysts.

No Giardia cysts or trophozoites were found by faecal microscopy during testing of beavers from the River Otter Beaver Trial (n=0/43), Tayside, Scotland (n=0/22) (Campbell-Palmer et al. 2015a; Campbell-Palmer and Girling, 2019) or Knapdale (n=0/19) by PCR (Goodman et al., 2012). However, microscopy is not a particularly sensitive method of detection of Giardia species (Fayer et al., 2006) and shedding of cysts is sporadic (Horton et al., 2018) so it is possible that cases of infection with G. duodenalis in free-living beavers have been missed. Prior to and following the introduction of beavers to Knapdale, water courses were monitored for the presence of Giardia cysts (Mackie, 2014). Giardia species were identified at one site prior to release of the beavers by microscopy and, following release, were again found at this site at similar levels but at no new sites. However, neither the species nor the source of the original contamination was identified so it is possible that the beavers were not susceptible to the Giardia species or assemblages at the site.

Robertson and Gjerde (2001) detected Giardia species in 29% (n=28/147) of water courses tested between 1998 and 1999 in Norway using immunofluorescence microscopy. These were not genotyped and no association was noted between the presence of beavers at a site and water contamination. In addition, no infected beavers were found in Norway (n=0/241), or beavers imported from Norway for the Knapdale trial (n=0/19), using an immunoassay to detect Giardia antigen in faeces (Rosell et al. 2001; Goodman 2014).

It has therefore been proposed that beavers may not be a true reservoir for G. duodenalis but may act to maintain and amplify an environmental reservoir once infected (Monzingo and Hibler, 2007). In a previous assessment following the Knapdale trial, Boden and Auty (2015) concluded that existing sources of contamination such as humans and other animals were likely to be greater contributors to the overall number of Giardia cysts shed into the environment than beavers but that beavers were likely to make a small additional contribution to the environmental reservoir of Giardia species.

Given the potential for Eurasian beavers to amplify environmental reservoirs once infected, thereby increasing the infection potential to humans and livestock and sympatric species, G. duodenalis should be considered as a hazard for humans and livestock following the translocation of beavers.

Risk Assessment

Exposure assessment

Beavers living in areas where water courses have been contaminated by faeces from infected humans or domestic animals, for example cattle, may ingest Giardia cysts in water or on plant material. As G. duodenalis assemblages A and B can infect, and replicate in, a wide range of species, including beavers, the infective dose is low and cysts survive for prolonged periods in cool water, there is a low likelihood that translocated beavers may be exposed to and infected by G. duodenalis assemblages A or B.

Following ingestion, trophozoites are released from the cyst in the duodenum where they undergo repeated mitotic division and formation of infectious cysts which are shed in faeces (Ryan and Caccio, 2013). As beavers are coprophagic they are likely to repeatedly re-infect themselves and to increase the number of infectious cysts shed in their faeces into water surrounding their lodges (Monzingo and Hibler, 2007). Conspecific, sympatric species and humans and domestic animals drinking or accidentally ingesting water while swimming downstream will be exposed to infection and there is a high likelihood of exposure and infection. In slow moving water, cysts quickly fall to the bottom of the water course but may spread widely in faster-moving water (ibid.).

As Giardia cysts have prolonged survival in water and are fairly resistant to chemical treatments (Tsui et al., 2015) the likelihood of dissemination to other susceptible species close to beaver habitat or, at some distance in moderate to fast moving watercourses, is high.

Consequence assessment

No cases of giardiasis have been reported in beavers so it is likely that, in common with many other species, otherwise healthy animals do not experience clinical disease following infection. In humans, age, immunocompetence and gut flora determine susceptibility to disease development (Horton et al., 2018) and the same may be true of other species. Young calves, puppies and kittens infected with G. duodenalis may experience acute diarrhoea, ill-thrift and even death (Feng and Xiao, 2011). Feng and Xiao

(2011) report several studies in farm animals demonstrating decreased weight gain and reduced feed efficiency with associated economic loss as a result of giardiasis. There is a very low likelihood of a disease outbreak in humans or domestic animals, and associated economic effects, as a result of an increased load of Giardia species at the destination.

Risk estimation

There is a low likelihood that beavers will be exposed to and infected with G. duodenalis A or B, a high likelihood of exposure and infection of sympatric species at the destination and a high likelihood of dissemination to other species in close proximity to beaver lodges or at greater distances in areas of fast moving water at the destination site(s). There is a very low likelihood of a disease outbreak in humans and domestic animals. The change in risk at the destination site(s) as a result of beaver translocations is likely to be very low. The overall risk is VERY LOW.

Risk Management

Risk management options

Public health advice, particularly warning of the risks of swimming close to beaver lodges, and regular water testing is likely to prove more valuable in management of the risks. Release sites should, ideally, be chosen in consultation with relevant water authorities or private water supply owners, particularly given the likely long-term potential for beavers to disperse away from release sites. Consultation with local landowners and recommendations to fence grazing areas to prevent livestock defecating into water edges may also be advisable in order to reduce the likelihood of infection to beavers.

Appendix 14 Disease Risk Analysis for the Unclassified Hazard Cryptosporidium parvum

Cryptosporidium species are ubiquitous enteric protozoan parasites that can infect a broad spectrum of vertebrate hosts causing a range of clinical disease from asymptomatic to acute or chronic diarrhoeal disease (Mateo et al. 2017). Infection in healthy humans is usually self-limiting and declines in prevalence with increasing age (ECDPC, 2019b) but disease can be severe in young mammals, especially if malnourished, and persistent in immunodeficient adults (Laurent, 2019). Transmission is primarily faeco-oral, either directly or indirectly via the environment in water and food, and respiratory infection via nasal secretions is also reported (Thompson et al., 2005). Oocysts have been shown experimentally to remain viable in river water for almost six months with prolonged survival in faeces (Robertson et al., 1992). Water-borne oocysts are resistant to chemical treatment, including chlorine (Chalmers et al., 2019), and ingestion of fewer than 10 oocysts may lead to infection (Ryan et al., 2014).

At least 38 species of Cryptosporidia have been identified to date, most of which are host-specific (Feng et al., 2018). Genotyping, usually using the Gp60 gene, has facilitated understanding of Cryptosporidium species epidemiology and transmission between species and the environment (Chalmers et al., 2019). At least 20 Cryptosporidium species and genotypes have been identified in humans but not all may be true infections as it is often hard to differentiate patent infections with replicating parasites from the mechanical transmission of ingested oocysts (Feng et al., 2018). Humans are commonly infected by C. parvum or C. hominis with C. ubiquitum regarded as an important emerging zoonosis because of its wide geographic distribution and host range (Mateo et al., 2017).

C. hominis is usually regarded as host-specific to humans but is increasingly reported in animals. however most animal infections with C. hominis are probably spill-over events from human reservoirs (Feng et al., 2018). To date, eight host-adapted sub-families of C. ubiquitum have been identified (Feng et al., 2018). In the USA, humans are predominantly infected with rodent sub-types XIIb to XIId but in the UK zoonotic infection is predominantly from ruminant-adapted sub-type XIIa (ibid.). The broad host range of rodent-adapted C. ubiquitum sub-types may indicate a sylvanian transmission cycle with occasional spill-over to humans (Tan et al., 2016).

C. parvum is the most important zoonotic Cryptosporidium species and also the most common cause of cryptosporidial disease in young calves (Brook et al., 2009). Currently nearly 20 sub-types of C. parvum are recognised of which the most prevalent, IIa and IId, are adapted to animals and IIc adapted to humans (Feng et al., 2018). Of these, IIaA15G2R1 is the dominant IIa subtype in calves and lambs and is also commonly reported in humans (ibid.). In addition to ruminants, IIa has been reported in a wide range of species including wild trout (Salmo trutta) in northwest Spain (n=47/613) (Couso-Perez et al., 2019) and rats in Malaysia (n=9/12) (Tan et al. 2019) although cats and dogs do not appear to be susceptible to infection (Thompson et al., 2005). Historic reports of high levels of C. parvum prevalence in wild rodents by Sturdee et al. (2003) and Bajer et al.

(2002) may have been overstated due to reliance on diagnosis by morphology alone prior to the advent of molecular genetic tools and the potential for cross-reactivity between C. parvum and newly identified Cryptosporidium species in voles (Horcickova et al., 2019).

Chalmers et al. (2019) analysed outbreaks of human infections with Cryptosporidium species between 2009 and 2017 in England and Wales and found that 56% (n=82/178) were caused by contact with recreational waters and 42% (n=74/178) were as a result of animal contact. Of outbreaks where the causative species was identified, 53% were found to be C. parvum (n=69/131) and 46% (n=60/131) C. hominis. Using gp60 subtyping, Chalmers et al. (2019) identified that animal contact-based outbreaks predominated in the first half of the year, when incidence in calves and lambs also peaks, and were all caused by C parvum. Identical subtypes were isolated from lambs in 12 outbreaks and from calves in 2 (ibid.). The predominant subtype (IIaA15G2R1) was also previously isolated from faecal samples from calves on 14/41 farms in a study in Cheshire in 2004 (Brook et al., 2009). C hominis was not isolated from any animals at locations associated with recreational water outbreaks in the study by Chalmers et al. (2019). Following the outbreak of foot and mouth disease in the UK in 2001, and the extensive culling of ruminant livestock and limits on human and animal movements, reported human cases of cryptosporidiosis caused by C. parvum were only 35% (n=338/977) of the previous year's level (Smerdon et al., 2003) further suggesting that ruminants are a major reservoir of zoonotic C. parvum isolates.

Justification of Hazard Status

There has been only limited testing of beavers for infection with Cryptosporidium species and so susceptibility and reservoir potential is poorly understood in the species. Paziewska et al. (2007) analysed faecal samples from 52 wild caught and farmed Eurasian beavers in Poland using an immunofluorescence assay (MeriFluor IFA) for Cryptosporidium antigen. 19.2% (n=10/52) samples were positive with statistically insignificant differences between prevalence and abundance in wild and farmed beavers which Paziewska et al. (2007) proposed as an indication of autogenous rather than environmental infection. The test used in this study is specific for C. parvum but is reported to also cross-react with C. muris and C. meleagridis (Y. Craig, pers. comm). Sroka et al. (2015) tested 79 water samples from 14 water courses close to beaver habitats between 2010-14 in Poland. 45.6% (n=36/79) of water samples were positive for Cryptosporidium species by immunomagnetic separation which is not specific for C. parvum. There was no statistical difference in the prevalence of oocysts at different distances from the beaver lodge, unlike for Giardia duodenalis, also tested in this study, for which prevalences were significantly higher the closer to the lodge the water was sampled, so the authors were unable to conclude that beavers were the source of the water contamination.

Human cases of Cryptosporidium infection in Norway are reported to be the 4th highest in Europe and to be increasing rapidly, with a 50% increase in 2017 (n=379/255), the last year for which figures are available (ECDPC, 2019b). Cryptosporidium oocysts are regularly isolated from surface water in Norway (Rosell et al., 2001) but were not detected in limited testing of 241 free-living Norwegian beavers in Telemark, Norway between 1997

and 1999 using a microplate immunoassay for Cryptosporidium species antigen (ibid.). Human outbreaks in 2009 and 2012 in Norway have been associated with sub-type IIaA19G1R1, shown to have been caused by contact with infected lambs and kids (Lange et al., 2013). Beavers imported from Norway (n=19) for the Knapdale project in Scotland in 2008 all tested negative for Cryptosporidium infection (Goodman et al., 2012).

The UK reported the highest number of Cryptosporidium species infections in humans (n=5052) of any reporting country in Europe in 2017 (ECDPC, 2019b) with nearly half of cases resulting from animal contact (Chalmers et al., 2019). Testing of free-living beavers by microscopy as part of the River Otter Beaver Trial (n=43) did not identify any infected beavers (Campbell Palmer and Girling 2019); however a single adult male (n=1/22), shot in Tayside, Scotland (Campbell-Palmer et al., 2015b) and a predated kit, recovered in Knapdale (Mackie et al., 2014) were found to be infected although the Cryptosporidium species was not identified in either case. Testing for Cryptosporidium species infection by microscopy is not regarded as sensitive and it is estimated that about 50% of all cases are missed by this method (Nichols et al., 2006). In addition, oocyst shedding may be intermittent (Ryan et al., 2016) so it is possible that further infected beavers have been missed. In addition, it has been proposed that beavers can amplify and contribute to the environmental reservoir of Giardia duodenalis, even if not a primary reservoir (Monzingo and Hibler, 2007), and there may similarly be potential for beavers to amplify environmental burdens following infection with zoonotic Cryptosporidium parvum sub-types. Prior to and following the release of beavers for the Knapdale trial, water courses were monitored for the presence of Cryptosporidium species oocysts (Mackie et al., 2014). 4/6 sites in Knapdale were found to contain Cryptosporidium species oocysts of unknown species prior to the release of beavers but following release of the beavers, Cryptosporidium oocysts were only recovered from one of the four sites (ibid.). However, this may indicate that beavers were not susceptible to infection with the particular Cryptosporidium species detected. As beavers have been shown by other authors to be susceptible to unidentified Cryptosporidium species which may include sub-types that are infectious to livestock and humans, C. parvum should be considered as a hazard for humans and livestock following the translocation of beavers from Norway and within Great Britain.

Risk Assessment

Release assessment

Contamination of watercourses by faeces from infected humans and other animals may be sporadic with oocysts remaining infectious for several months following excretion. Beavers sourced from, or released into, contaminated areas may ingest Cryptosporidium oocysts in water or on plant material. As the infective dose is low, oocysts can survive for prolonged periods and C. parvum sub-types IIa and IId can infect, and replicate in, a wide range of species which may include beavers, there is a very low likelihood that translocated beavers may be exposed to and infected by sub-types of Cryptosporidium species infectious to humans and other animals. Following ingestion or inhalation of sporulated oocysts by a suitable host, the oocyst excysts and its 4 sporozoites rapidly invade

epithelial cells and undergo asexual proliferation, ultimately resulting in the formation of large numbers of thick-walled oocysts which are released in either faeces or nasal secretions (Thompson et al., 2005).

Exposure assessment

Infected beavers will excrete large numbers of oocysts in their faeces into water courses close to their lodges. In addition, as beavers are coprophagic they are likely to repeatedly reinfect themselves and to increase the number of infectious oocysts shed in their faeces into water surrounding their lodges (Monzingo and Hibler, 2007). Conspecifics, sympatric species and humans and domestic animals drinking or accidentally ingesting water will be exposed to infection. As Cryptosporidium oocysts have prolonged survival in water and are resistant to chlorine treatment there is a high likelihood of exposure and infection of people and domestic animals. There is a high likelihood of dissemination to other susceptible species, even at some distance from beaver lodges due to the prolonged survival in water.

Consequence assessment

There is a very low likelihood that a translocated beaver will be infected with Cryptosporidium species.

No cases of cryptosporidiosis disease have been reported in beavers so it is likely that, in common with many other species, otherwise healthy adult animals do not experience long-lasting clinical disease following infection. However, immunocompromised hosts may develop more severe clinical signs or recurrent and chronic infections and young calves, lambs and kids may die from dehydration and cardiovascular collapse (Thompson et al., 2005). The economic cost to farmers as a result of impaired weight gain and the cost of treatment may be significant (ibid.). There is a medium likelihood of sporadic disease in humans and domestic animals and economic effects from public health control, hospital treatment and veterinary treatment.

In a previous assessment following the Knapdale trial, Boden and Auty (2015) concluded that other, existing sources of contamination such as humans and other animals are likely to be greater contributors to the overall number of shed into the environment than beavers. It seems likely that beavers may have potential to contribute to and amplify the environmental burden of infectious Cryptosporidium species oocysts but are likely to cause only a very low increase in the overall burden.

Risk estimation

There is a very low likelihood that beavers will be exposed to and infected with Cryptosporidium parvum sub-types II a or d, a high risk of exposure and infection of beavers, sympatric animals, humans and domestic animals at the destination, and a high risk of dissemination to other species at the destination site(s). There is a medium likelihood of sporadic disease in humans and domestic animals at the destination. The

change in risk at the destination site(s) as a result of beaver translocations is likely to be very low. The overall risk is VERY LOW.

Disease Risk Management

Risk management options

Public health advice, particularly warning of the risks of swimming close to beaver lodges, and regular water testing may prove valuable in management of the risks. Release sites should, ideally, be chosen in consultation with relevant water authorities or private water supply owners, particularly given the likely long-term potential for beavers to disperse away from release sites. Fencing to prevent livestock defecating into water edges may also be advisable in order to reduce the likelihood of infection of beavers and transmission from beavers to domestic animals.

Appendix 15 Disease Risk Analysis for the Carrier Hazard Eimeria species

Justification of Hazard Status

Coccidia are a subclass of protozoan parasites within the phylum Apicomplexa, further divided into four orders including Eucoccidiorida. There are two suborders within Eucoccidiorid, the second being Eimeriorina, which contains several genera of coccidian parasites known to cause disease in vertebrates.

Eimeria sprehni oocysts have been reported several times as a post-mortem finding in beavers. Demiaszkiewicz et al. (2014) undertook parasitological examinations of 48 free-living Eurasian beaver carcasses found between April 2011 and November 2012 in Poland. In one young beaver, oocysts of E. sprehni were detected in faeces. A low burden of Eimeria species oocysts were detected in the faeces of one live-trapped Eurasian beaver in Tayside as part of health screening of this population between 2013 and 2019. The beaver was a juvenile and in good body condition with no signs of associated disease. No analysis was undertaken to determine the species of Eimeria (Campbell-Palmer et al., submitted).

E. sprehni has also been detected in free-living North American beavers. A survey was undertaken in Kansas, USA, during the trapping season of 1991, and 63 beaver carcasses were analysed to determine their endoparasite fauna. 25% of beavers (n=16) were infected with E. sprehni, and a further 5% (n=3) were infected with E. causeyi. One of these animals had a mixed infection with both species (Mckown et al., 1995). Two early reports of coccidia in C. canadensis exist. Morley (1934) found coccidia oocysts in the faeces of one beaver from Pennsylvania (cited by McKown et al., 1995) and, in the same year, Yakimoff (1934) described a case of E. sprehni from a captive North American beaver (cited by McKown et al., 1995). These reports provide evidence that coccidian parasites can be present in beavers, although associated disease has not been reported.

The lack of disease associated with these coccidian infections in beavers concurs with general consensus that these parasites are non-pathogenic in rodents in the absence of underlying disease (Chapman et al., 2013; Schmidt, 1995). However, there are several reports which present evidence that some coccidian parasites can lead to disease in rodent species. In guinea pigs, infection with E. caviae can lead to severe disease and death. Clinical signs include watery or haemorrhagic diarrhoea, anorexia and a poor quality coat (Brabb et al., 2012; Ellis and Wright, 1961). Gross pathological lesions associated with this gastrointestinal parasite include thickening of the colon and petechial hemorrhages alongside white plaques on the colonic mucosa (Schmidt, 1995). Stress has been attributed to increased virulence of this parasite; a group of 12 laboratory guinea pigs died after exhibiting clinical signs of diarrhoea, and the cause of death was attributed to E. caviae after lifecycle stages were found within the colonic mucosa on histopathological examination. It is thought that disease was triggered after the guinea pigs were exposed to

stress including transport, injection and introduction to new surroundings (Ellis and Wright, 1961).

Another Eimeria species, E. falciformis, has been suggested to cause diarrhoea and catarrhal enteritis in European mice when heavy infection occurs (Whary et al., 2015). Mice have been shown to be susceptible to disease from E. falciformis in a laboratory setting. In a study by Mesfin et al. (1997), groups of mice were infected orally with different numbers of oocysts to determine if parasite burdens lead to increased disease severity. It was found that mortality rates increased as the infective dose increased. The highest mortality rates were seen in mice infected with over 20,000 oocysts (30.8%, n=20), although this mortality rate was not significantly different to mice infected with 5,000 oocysts (27.3%, n=29). No mortalities occurred in the 105 mice infected with 500 oocysts, but disease including diarrhoea, depression, anorexia and weight loss occurred in all experimental groups and histopathology determined that E. falciformis was associated with the disease (Mesfin et al., 1977). Although this study was undertaken in a laboratory setting, it provides indication that rodents can suffer disease and death as a result of infection with coccidian parasites under certain conditions, and severity may increase with exposure dose. Although the validity is reduced by the laboratory setting, the increased stress experienced by animals in this environment may have impacted upon the severity of results, which may be replicated when undertaking conservation interventions such as translocations. Indeed, stress has been attributed as a cause for increased virulence of coccidian parasites in host species. It is widely understood and accepted that stress can lead to immunocompromise (Dhabhar and McEwen, 1997; Dickens et al., 2010; Glaser and Kiecolt-Glaser, 2005) and stress has been suggested to be an inevitable component of animal translocations, which can occur at multiple stages including capture, transport and captivity (Dickens et al., 2009, 2010; Teixeira et al., 2006).

Coccidiosis was suggested to be a common cause of death in red squirrels in the UK after a post-mortem survey was undertaken (Keymer, 1983). This finding was further supported by reports of mortality associated with coccidiosis in red and grey squirrels in the UK (Tittensor, 1975, 1977) and red squirrels in Finland (Lampio, 1967). However, it is difficult to conclude that coccidiosis was the cause of death of squirrels in these studies as results were not confirmed histopathologically and relied instead on findings of oocysts within the intestines. Pathogenicity of E. sciurorum has been confirmed experimentally (Pellérdy, 1974), but never in free-living animals. It is likely that stress, infective dose and underlying disease initiate increased virulence of the parasite.

It is known that beavers carry certain coccidian parasites within their intestines, and that rodents can suffer from disease as a result of coccidiosis, particularly under conditions of stress and/or high infective doses. Therefore, since translocation is likely to act as a stressor to the beavers, and there is the possibility that beavers will be exposed to infective doses coccidiosis could occur.

Risk Assessment

Release assessment

Eimeria species have a direct life cycle. Infected hosts shed unsporulated oocysts in faeces which sporulate in the environment, if conditions are favourable, and become infective. When a new host ingests these oocysts, they migrate to epithelial cells, most often of the intestinal mucosa, where they develop (McDonald and Shirley, 2009; Norton and Chard, 1983). In order to become infected, a beaver must ingest unsporulated oocysts from the environment. Coccidian parasites show a high degree of host specificity, particularly within the Eimeria genus (Chapman et al., 2013; Ellis and Wright, 1961), and can persist for long periods of time in the environment, particularly soil (Lassen et al., 2013).

There have been no reports of Eimeria species detection in beavers from Norway, although sporadic cases have been described in beavers across the world, both free living and captive, including in Scotland. There is a low likelihood of beavers being chronically infected with Eimeria species when translocated.

Exposure assessment

Infected beavers will carry the protozoa to the destination and may contribute to the environmental reservoir of these parasites through faecal shedding. Therefore, there is a high likelihood of exposure of other beavers at the destination especially because the small population will be at relatively high density immediately after translocation.

Since Eimeria species are host specific, this is unlikely to contribute to infection in other rodent species at the destination site. The reintroduction itself is predicted to have a low impact on the host-parasite dynamics at the destination site since Eimeria species are likely to be prevalent in the environment across Europe. Therefore, the likelihood of dissemination at the destination site because of beaver reintroduction is negligible.

Consequence assessment

The probability that at least one beaver is infected with Eimeria species at the time of translocation is low.

There is a medium likelihood that the conditions of translocation will, as a stressor, lead to immunocompromise and a change in host-parasite dynamics resulting in coccidiosis in translocated beavers. There is a medium likelihood that the conditions of translocation may expose beavers to a higher burden of parasites than would occur naturally, leading to disease.

In cases of acute clinical disease, there is a low likelihood of severe disease in the individual and a low likelihood of death. There is a low probability of economic consequences as a result of coccidiosis in translocated beavers leading to the failure of the translocation. There is a low likelihood of biological, environmental and economic consequences at the destination as a result of failure of the translocation. The likelihood of

ecological consequences at the destination site is negligible because Eimeria species are already present in the UK.

Risk estimation

There is a low probability of beavers being exposed and infected with Eimeria species at the source site. There is a high likelihood of exposure at the destination but a negligible likelihood of dissemination. There is a medium likelihood that infected beavers will develop disease as a result of translocation and a low likelihood of biological, economic and environmental consequences through failure of the translocation. Overall, the risk is estimated to be LOW.

Disease Risk Management

Risk evaluation

Since the risk is estimated to be higher than negligible, mitigation methods should be implemented.

Risk management options

Stress reduction and good captive management throughout the translocation process are key in reducing the probability of disease associated with coccidiosis in beavers. In addition, hygiene to reduce environmental burdens of coccidia oocysts will be beneficial.

Diagnostics for coccidiosis should be part of the post release health surveillance protocol to help inform future decision making regarding this parasite.

Appendix 16 Disease Risk analysis for the Carrier Hazard Emmonsia crescens

Justification for Hazard Status

Emmonsia species are saprophytic fungi which can infect a broad range of mammalian hosts, including occasionally domestic animals and humans, leading to adiaspiromycosis, a respiratory disease of variable severity (Danesi et al., 2020). The disease is considered to be one, primarily, of burrowing animals, in particular small rodents and mustelids (ibid.). The two Emmonsia species of concern are Emmonsia crescens, (syn. Emmonsia parva var. crescens) and E. parva, recently reclassified as Blastomyces parvum. The two are differentiated primarily on microscopic evaluation of adiaspore size and morphology with B. parvum characterised by thin-walled uninucleate adiaspores of 10 to 40µm and E. crescens by multinucleate adiaspores up to 400 µm in diameter (ibid.). B. parvum has a narrow host and geographic range and is very rarely found in Europe (Borman et al., 2018). The only reported case of B. parvum in Europe is from a red fox in Czechoslovakia in 1975, based on adiaspore appearance prior to the availability of PCR for confirmatory diagnosis (Otcenasek et al., 1975).

Infections with E. crescens occur when saphrophytic conidia are inadvertently inhaled from the environment, such as soil or nesting materials (Borman et al., 2018). The conidia do not replicate in the lungs, instead enlarging in size to form microscopically visible, dormant adiaspores (ibid.). In immunocompetent hosts, granulomata form around the adiaspores and may compress small airways, leading to asymptomatic infection or respiratory disease (ibid.) although granulomata without adiaspores may also be observed (Harrington et al., 2012). The severity of disease is believed to be related to the number of spores inhaled (Dolka et al., 2017). Heavy infections, typically in animals that burrow where exposure risk may be higher, are associated with poor body condition, emaciation and occasional mortality (Borman et al., 2009). The lifecycle of the parasite is completed when the host animal dies and spores are released to the environment as the carcass decays where they sporulate on mycelia in decaying plant material (Simpson et al., 2016).

E. crescens infection has been diagnosed in a broad range of wildlife species in Great Britain and Norway. Borman et al. (2009) reported that almost 1/3 (n=27/94) animals found dead in Great Britain and submitted to the Wildlife Veterinary Investigation Centre, Truro between 2003 and 2005 were positive for E. crescens infection on either microscopy or histopathology (Table 1). When both microscopy and histopathology were used together for diagnosis, recorded prevalence was higher at 43% (n=9/21) (ibid.). Borman et al. (2009) noted that true prevalence of infection may be even higher as low burdens could have been missed as only a small portion of lung tissue was selected for evaluation. E. crescens infection has been reported in Great Britain in the American mink, water vole, European rabbit; red squirrel and European mole (Talpa europaea) (Harrington et al., 2012; Chantrey et al., 2006; Hughes and Borman, 2018; Simpson et al., 2013; Simpson et al., 2019).

Of 562 mammals from 16 species culled for evaluation in Norway in 1959, 40% (n=4/10) of voles (Microtus species) and 1/1 water vole were positive for E. crescens (Table 6) and infection was reported in museum specimens of two wood mice and six bank voles from a sample of unspecified size (Table 7) (Jellison and Vinson, 1961). High prevalences of infection with E. crescens in otherwise healthy animals have also been reported in Europe in the muskrat: 22.3% (n=46/206) of muskrats culled in Sweden (Macieira, 2019) and 8/8 culled in Czechoslovakia (Otcenasek et al., 1974).

Table 6 Prevalence of E. crescens in British wildlife 2003-5. (Source: Borman et al., 2009)

Mammalian species	Prevalence (%)				
	Immature		Adults		Total
	M	F	M	F	
Lutra lutra (Otter)	4/8 (50)	4/8 (50)	4/19 (21.1)	6/20 (30)	18/55 (32.7)
Mustela nivalis (Weasel)	-	0/3	2/5 (40)	0/2	2/10 (20)
Mustela erminea (Stoat)	-	-	1/4 (25)	1/3 (33.3)	2/7 (28.6)
Mustela vison (Mink)	-	-	0/1	0/2	0/3
Vulpes vulpes (red Fox)	-	1/3 (33.3)	0/1	0/3	1/7 (14.3)
Martes martes (Pine marten)	-	1/1 (100)	0/1	-	1/2 (50)
Talpa europea (Mole)	-	-	-	1/3 (33.3)	1/3 (33.3)
Mus sp. (mice)	-	0/1	1/1 (100)	1/2 (50)	-
Rattus norvegicus (Rat)	-	-	1/2 (50)	-	1/2 (50)
Mustela furo (Ferret)	-	-	0/1	-	0/1 (0)
Sorex sp. (Shrews)	-	-	-	0/2	0/2
Total	4/8 (50)	6/15 (40)	8/35 (22.9)	9/36 (25)	27/94 (28.7)

Table 7 Prevalence of E. crescens in Norwegian wildlife 1959. (Source: Jellison and Vinson, 1961)

Hosts	Number examined	Number infected
Mus Musculus, house mouse	239	0
Apodemus spp., wood mice	102	0
Sus sp., domestic pig	60	0
Sorex sp., shrew	40	0
Rattus norvegicus, rat	27	0

Hosts	Number examined	Number infected
Clethrionomys sp., red-backed mouse	22	0
Mustela vison, mink	22	0
Vulpes sp., fox	14	0
Microtus sp., vole	10	4
Felis catus, domestic cat	9	0
Lepus sp., rabbit	6	0
Sciurus sp., squirrel	4	0
Mustela sp., weasel	4	0
Arvicola terrestris, water vole	1	1
Lemmus sp., lemming	1	0
Meles meles, badger	1	0
Total	562	5

Borman et al. (2009) reported that E. crescens infection burdens in most animals were low (≤2 adiaspores/cm3 of lung tissue) and unlikely to have impaired physical health; however several animals (three otters, one weasel and one mole) had higher infection burdens (range 3-8 adiaspores/cm3 of lung tissue) with significant areas of lung parenchyma in the weasel infiltrated by granulomata likely to have caused severe respiratory disease. It has been proposed that some species, for example otters and wombats, may be more susceptible to disease following infection than others (Danesi et al., 2020). It is also suggested that immunocompromised animals may be more susceptible to disease: a previously healthy water vole died in captivity one month after capture with widespread adiaspiromycosis and was found on post-mortem examination to be severely emaciated and co-infected with another, unidentified fungus (Chantrey et al., 2006). Large scale die-offs of moles co-infected with Emmonsia species and other parasites are also reported (Simpson et al., 2016).

Infections with E. crescens have been rarely reported in free-living beavers. Morner et al. (1999) observed macroscopically visual lung lesions, consistent with adiaspiromycosis, with thick-walled adiaspores ranging between 100µm and 200µm noted on histopathology in both the lungs and mediastinal lymph nodes in a beaver shot in 1998 in northern Sweden which was in normal body condition with no signs of clinical disease. However, Morner et al. (1999) noted that no signs of infection had been noted in 110 previously culled beavers in Sweden. Eight percent of beavers (n=25) culled in Poland were found to be infected with Emmonsia species on histopathology and thick-walled adiaspores ranging between 163.4µm and 437.1µm (Dolka et al., 2017). One beaver had severe lesions with extensive granulomata, interstitial inflammation and emphysema, and was in poor physical

condition (ibid.). In both studies, the causative agent was assumed to be E. crescens based on the size and morphology of adiaspores.

As Emmonsia crescens is likely to be an ubiquitous organism in the environment, and translocation is a known stressor which may reduce immunocompetence (Dickens et al., 2010), E. crescens should be considered a carrier hazard for the translocation of beavers.

Risk Assessment

Release assessment

As Emmonsia crescens is widely present in Norway and Great Britain, there is a medium likelihood that beavers at the source site(s) could be exposed to E. crescens in the soil, on bark or in lodges, or from decaying carcasses of sympatric infected animals such as otters and muskrats which are reported to frequently share lodges with beavers, particularly in winter (Janiszewski et al., 2014). In addition, viable adiaspores have been isolated from the digestive tracts of rodents and carnivores that prey on small mammals suggesting that, even if the host is predated, adiaspores may still be returned to the environment and infection reservoir (Borman et al., 2018). If exposed to E. crescens conidia, there is a low likelihood that beavers could be infected by inhaling conidia as beavers are known to be susceptible to infection. There is a low likelihood that at least one translocated beaver could be infected with E. crescens at the source site(s).

Exposure assessment

Beavers translocated to the destination may already be infected with E crescens. As E. crescens does not replicate in mammalian hosts, the environmental burden of infective E. crescens conidia will not be increased as a result of live, infected beavers arriving at the destination. Spores may be released from beavers which die and decompose. As a wide range of mammalian species are susceptible to infection, there is a low likelihood that other beavers and sympatric species will be infected.

There is a very low likelihood that spores released from translocated beavers which subsequently die increase the environmental burden of infective conidia and disseminate infection amongst sympatric mammals including beavers.

Consequence assessment

There is a low likelihood that one beaver will be infected with E crescens.

Infection with E. crescens in most mammals is asymptomatic unless the host is immunocompromised, for example by stress, starvation, hunger or concomitant disease (Chantrey et al., 2006; Simpson et al., 2016). Adiaspiromycosis is characterised by compromised respiratory function, loss of body condition and increased susceptibility to secondary infection. Diseased hosts may also be more susceptible to predation if they are weak and slow-moving. There is a low likelihood that at least one beaver will be infected and since translocated beavers will be under stress (Dickens et al. 2010) there is a high

likelihood that infected beavers will be susceptible to adiaspiromycosis. As disease progression may be slow, clinical disease may not be apparent until weeks or months following translocation.

There is a medium likelihood of economic and biological consequences through failure of the translocation but the long-term environmental consequences are likely to be negligible.

Risk estimation

There is a medium likelihood that beavers will be exposed to and a low likelihood that they will be infected with E. crescens. The likelihood of exposure and infection at the destination site(s) is low and there is a very low likelihood of dissemination. There is a high probability that the stress associated with translocation may precipitate disease in infected beavers. The overall risk is MEDIUM.

Disease Risk Management

Risk management options

The gold standard of diagnosis is histopathological examination of biopsy or necropsy tissues with confirmatory PCR, with no reliable method of testing for infection in the live animal (Borman et al., 2018). Adiaspiromycosis should be considered in the differential diagnosis if sick beavers are found and examined post-translocation. Post-mortem examination of beavers dying following translocation, and of sympatric mammals at the destination is essential to monitor the effects of the translocation on health. Measures to reduce the level of stress from translocation are important. For example, efforts should be made to minimise the stress associated with capture, transport and, in particular, to reduce repeated handling, loading and unloading events, and the duration of transit.

Appendix 17 Disease Risk Analysis for the Population Hazard Road Traffic Collisions

Justification for Hazard Status

Road traffic collisions (RTCs) have been reported as a cause of death of beavers across Europe. Stefen (2018) analysed 1137 post-mortem reports of Eurasian beavers in Eastern Germany dating from 1941 to 2009 and found RTCs to account for the highest number of deaths (25.7%, n=292). Train collisions also caused 1.3% (n=15) of deaths. Others have similarly reported that RTCs are responsible for as high as 50-86.5% of beaver deaths in Germany (Pokorny et al., 2014., Muller 2014 cited by Grubešić et al., (2015)).

RTCs have been suggested to be the main cause of beaver mortalities in Croatia (Sager et al., 2005). Another study carried out across Croatia and Serbia found that 33% (n=50) of beaver carcasses analysed had been involved in traffic accidents (Grubešić et al., 2015). Furthermore, a report from France suggests that of 46 beavers found dead in the Haute-Savoie region, 37% (n=17) died as a result of RTCs (Estève, 1988). After translocation to the Netherlands, four beavers were killed by traffic in the Biesbosch (Nolet et al., 1997). Along the Elbe, three beavers were found to have been killed in RTCs in a study by Hinze (1950), and a further 10 in a study by Piechocki (1977). Two free-living beaver carcasses submitted and analysed after the Tayside beaver reintroduction in Scotland had injuries consistent with road traffic collisions (Campbell-Palmer et al., 2015b), as did a beaver carcass submitted to a veterinary practice in (Brazier et al., 2020, p91). Post-mortem examinations revealed road traffic collisions to be the cause of death in five out of six beavers found dead in the north western suburbs of Berlin, Germany, between 2006 and 2011 (Herrmann et al., 2013).

Risk Assessment

Exposure assessment

Many factors are likely to contribute to the exposure of beavers to traffic and therefore RTCs. Studies have indicated that elements such as traffic volume and roadside vegetation cover are associated with higher roadside mortality and mammals are more frequently affected by RTCs than birds or reptiles (Taylor and Goldingay, 2010). Moreover, road width has appeared as a broadly important predictor of mammalian road mortality (Barthelmess, 2014), as has landscape interconnectivity (Grilo et al., 2011).

It is possible that beaver dispersal at the release site will be high, leading to an increased probability of exposure to roads and thus RTCs. Following reintroduction into the Loire, France, post-release monitoring over a ten-year period identified 13 beavers reproducing in an area 200km upstream of the release site, demonstrating that substantial movement is possible in this species. That being said, beavers at release sites surrounded by urban areas did not undergo the same range expansion (MacDonald et al., 1995). Once settled,

beavers are also thought to travel up to 1.5 km into adjacent territories (Campbell et al., 2005).

Traffic densities at the release site are likely to impact upon incidence of beaver RTCs. In the areas surrounding the river Tay (Perth and Kinross, plus Angus), where two beavers were found dead due to RTCs, the road and traffic density is relatively low. In Perth and Kinross there are 124 major roads and 12 minor roads, with 1604.4 million vehicles travelling on these roads in 2018. In Angus, there are 59 major roads, nine minor roads and 715 million vehicles travelled in 2018. In Devon, where one free-living beaver mortality was reported as a result of an RTC, road and traffic density is higher despite Devon being comparable in size to Perth and Kinross/Angus. There are 246 major roads in Devon, 200 minor roads and 5441.8 million vehicles travelled in 2018 (Road Traffic Statistics - Department for Transport, 2018). These findings suggest that free-living beavers are at risk from RTCs in many areas of Great Britain with road numbers and traffic densities in a similar range in their release area. Notwithstanding, it has been suggested that the natural behaviours of beavers make them less likely to cross-roads than other mammals (A. C. L. Jones et al., 2012), and so lower number of roads would seemingly reduce their exposure to RTCs.

Minor roads have been suggested to have a greater impact on mortality than major roads in some mammalian species, such as badgers (which display territorial behaviour patterns similar to that of beavers), particularly if there is a high number of these roads (Taylor and Goldingay, 2010; van Langevelde et al., 2009). Therefore, the traffic densities and road size at the release site of these beavers is likely to impact survival, even if the roads are small and traffic density low.

We estimate a medium likelihood that reintroduced beavers will be exposed to a vehicle collision at the release site.

Consequence assessment

There is a medium likelihood that at least one reintroduced beaver will be hit by a vehicle. We found no reports of beavers surviving RTCs and therefore conclude that there is a high likelihood that death of the beaver will result. Economic impacts of a failed reintroduction would be considerable, however given the general success of the Scottish reintroductions despite two RTCs occurring, as well as numerous other successful reintroduction programs across Europe in which RTCs have occurred, there is a low probability that sporadic RTCs will lead to sufficient population losses to lead to reintroduction failure.

Risk estimation

We estimate a medium likelihood that beavers will be exposed to a vehicle collision at the release site. The likelihood of death if a beaver is involved in an RTC is high. However, there is a low probability that sporadic RTCs will lead to ecological and economic consequences through failure of the reintroduction program. The overall risk is estimated to be MEDIUM.

Disease Risk Management

Risk evaluation

Mitigation methods should be employed to reduce the risk of reintroduced beavers being involved in RTCs.

Risk management options

Traffic density, road size and road interconnectivity should be considered before choosing the release site, and ideally areas with low traffic density and smaller numbers of roads should be chosen to reduce the risk from RTCs. Warning signs and fencing could be placed along stretches of road which are considered to be at risk from beaver RTCs to encourage careful driving (A. C. L. Jones et al., 2012).

Appendix 18 Disease risk analysis for the Population Hazard Persecution

Justification for Hazard Status

The Eurasian beaver is persecuted throughout its range, including through snaring, shooting, hunting and malicious poisoning, and particularly when perceived negatively by local communities. Beavers are important keystone species which undertake landscape modification which benefits numerous other species within the ecosystem (Janiszewski et al., 2014). That being said, the impacts beavers have to local hydrology and fish stocks as a result of this landscape modification have been perceived negatively by local landowners and angling interests in the past, for example when the Scottish beaver reintroduction trial was proposed (Halley and Rosell, 2002; Scottish National Heritage, 1998).

Although there is substantial evidence confirming the positive effects of beaver populations to ecosystem health and other species populations, there have been conflicting reports on their impacts on fisheries. It was concluded by Scottish National Heritage, in response to concerns voiced about beaver reintroduction, that there may eventually be some areas of conflict between beavers and fishery interests depending upon the management of the beavers (Scottish National Heritage, 1998). Reduced fish stocks downstream of beaver dams have been reported in countries such as Lithuania (Kesminas et al., 2013; Virbickas et al., 2015), although a meta-analysis undertaken by Kemp et al. (2012) reported that the majority of experts found beaver populations to have an overall positive impact on fish populations in European and North American fisheries. North American beavers are a different species, although their ecological impact is likely to be comparable to the Eurasian beaver. It is likely that the impact of beavers is highly dependent on specific environmental components and management, and therefore the possibility of negative impacts to local communities after the reintroduction cannot be ruled out.

Pathological findings on free-living beavers following reintroduction into Tayside, Scotland found four animals examined suffered gunshot wounds (Campbell-Palmer et al., 2015b), which clearly indicates that this population was persecuted. It is unclear whether these authors undertook toxicological testing to detect malicious poisoning (or poisoning through misuse) and therefore, it is possible that further persecution has gone unreported. A survey-based study in Eastern Poland found beavers to be one of four species most frequently blamed for reducing yield at commercial fisheries. 21.2% of fish farms (n=29) reported serious, intolerable losses to fish stocks, and a further 46% (n=63) reported tolerable losses. Moreover, despite their protected status in Poland, and notwithstanding the provision of government compensation for losses attributed to beavers, persecution and culling still occurred (Kloskowski, 2011).

Furthermore, in a study by Stefen (2019), 1137 records of beavers found dead in Germany between 1941 and 2009 were analysed. Each case was attributed a probable cause of death and, overall, 41.5% (n=472) of deaths were directly related to anthropogenic

impacts. Suspected intoxication accounted for 1.8% (n=21) of deaths, metal traps 0.9% (n=10) and shooting 3.5% (n=40), indicating the potential for population losses to Eurasian beavers as a result of persecution. Others have reported beaver shootings across Europe, including a further two cases in Germany (MacDonald et al., 1995). Licenses to undertake lethal control were granted after the Tayside beaver population grew to an unmanageable level, however beaver shootings have occurred 'outside of licences' in Scotland/Wales (Roisin Campbell-Palmer, pers. comm, 7 May 2020). Some traps likely to be targeting beavers have also been noted in the UK, but the target species cannot be proven (Roisin Campbell-Palmer, pers. comm, 7May 2020).

Historically, Eurasian beavers have been hunted for their coats and castoreum, a urine-based secretion used for scent marking which was considered to have medicinal properties. This persecution is thought to still occur in parts of their range, including Mongolia (Batbold et al., 2017). Nonetheless, there is little likelihood of a fur/castoreum market being re-established within the UK and therefore hunting pressures are not likely to affect these populations in the same manner as conflict-related persecution (Scottish National Heritage, 1998).

Risk Assessment

Exposure assessment

There are several opportunities for human-wildlife conflict to occur as a result of the beaver reintroduction. Fisheries, angling and farming interests are widespread in England, and therefore there is a medium likelihood of exposure to persecution through shooting and poisoning. Between April 2018 and March 2019, 955,310 fishing licences were granted in the UK (Environmental Agency, 2020). Between 2017 and 2018, 1,191,142 fishing licences were granted across England; highest numbers were in Yorkshire (120,961), Staffordshire, Warwickshire and West Midlands (109,798) and Kent and South London (106,741), showing that angling interests are extensive across England (Environment Agency, 2019).

Attitudes towards beavers are mixed across Europe. A telephone survey of pond fisheries in Eastern Poland found a general negative attitude towards beavers as a 'nuisance species' (Kloskowski, 2011). Before the Knapdale beaver reintroduction was undertaken, attitudes towards the scheme from local residents were largely positive, with 46% (n=680) of Argyll and Bute residents agreeing that a trial reintroduction of beavers should be undertaken and 21% (n=310) disagreeing. The remainder were indifferent.

Any potential conflict between wildlife and humans may result in persecution and therefore, we estimate a medium likelihood of sporadic cases of illegal persecution occurring amongst the reintroduced Eurasian beaver population, particularly given the perceived negative impacts of landscape modification on fisheries and farmland. There is a negligible probability that reintroduced beavers will be hunted for their fur or castoreum.

Consequence assessment

The probability of one beaver being persecuted is high. The consequence could range from severe injury to death. Judging by the infrequent shootings which occurred to beavers during reintroduction in Scotland, cases of persecution are likely to be sporadic. However, the small population size of reintroduced beavers may be significantly affected by even low numbers of persecutions. Notwithstanding, within the Tayside population, the sporadic shootings did not lead to significant population effects. Therefore, there is a low likelihood of a negative impact on the population of reintroduced beavers and a failure of the reintroduction with resultant biological and economic consequences. There is a negligible impact of environmental consequences.

Risk estimation

There is a medium likelihood of the reintroduced population of beavers being exposed to persecution. There is a high likelihood of severe consequences, such as death, from the persecution of one individual. There is a low probability of economic and biological impacts of a failed reintroduction. The overall risk is estimated to be MEDIUM.

Disease Risk Management

Risk evaluation

Preventative measures must be employed to reduce the risk and consequences of illegal persecution to reintroduced Eurasian beavers.

Risk Management Options

It is imperative to educate local communities about the reintroduction program and the benefits of reintroducing Eurasian beavers to the local area.

Eurasian beavers should be closely monitored, and detailed pathological examinations performed on any carcasses found using pre-determined protocols. Testing should include toxicology to identify cases of accidental/non-targeted/malicious poisoning so that, if necessary, mitigation can be implemented.

It would be an advantage to give Eurasian beaver population protected species status in England, as has been granted by the Scottish government to those beavers reintroduced into Scotland. Furthermore, licences to alter beaver habitats which result in negative impacts to adjacent agricultural land could be authorised to certain individuals to try to minimise conflict as far as possible. Dam removal or modification has been suggested to mimic natural dam failures which have no significant impact on populations and rarely cause problems to beavers (Jones et al., 2012).

Appendix 19 Disease risk analysis for the Population Hazard Captivity During Translocation

Justification for Hazard Status

It is inevitable that, during the process of translocation of beavers from the source to the destination, a period of captivity will be necessary, as is true for all translocations. Initially, beavers must be trapped, followed by transportation and, depending on quarantine recommendations, a period held in captivity. There are numerous reports of disease in captive beavers, some of which, evidence shows, have resulted from inappropriate husbandry measures and other stressors, and several of these cases have occurred as a result of beaver translocations. Here we consider these cases collectively as a hazard described as ‘captivity during translocation’. We have used some evidence from reports in Canadian beavers because the behaviour of this species and Eurasian beavers in captivity has similarities.

Between 1994 and 1999, 277 Canadian beavers were captured using Hancock traps and snares throughout Wyoming, USA, for the purpose of translocation. Fifteen Canadian beavers (5.4%) died during trapping and 13 (4.7%) died during transport to the release site (McKinstry and Anderson, 2002). Trapping mortality resulted from either predation whilst trapped or entanglement in snares. Diagnoses in those animals which died during transport were unclear. One further case of mortality while trapping using a Hancock trap has been reported in New York, USA (Rosell and Kvinlaug, 1998).

Several authors have postulated that a period of time in captivity may reduce the fitness of translocated beavers post-release. For example, 34 beavers in the Wyoming translocation (McKinstry and Anderson, 2002), of 114 fitted with radio transmitters, died within 180 days of release as a result of predation. Although beavers in England may not face the scale of predators Canadian beavers contend with in Wyoming (black bears, coyotes and grizzly bears all contributed to mortality), it is possible that reduced fitness of the beavers as a result of transport resulted in increased predation risk. Translocated beavers may also be more vulnerable to persecution and road traffic collisions: these hazards were responsible for the deaths of 5% (n=14) of released Canadian beavers, a threat too for beavers released into England (McKinstry and Anderson, 2002).

During trapping and health screening of free-living Eurasian beavers on the river Tay in Scotland, no trap related mortality was observed in the 17 animals caught, however mild trap-related morbidity was detected in an unspecified number of beavers (Campbell-Palmer et al., 2015a). Unusual incisor wear was noted in one individual, thought to have occurred as a result of the beaver biting the metal trap in an attempt to escape. The tooth root was not exposed, and the injury was not believed to be causing pain or feeding problems. Minor abrasions to the oral cavity, nose and forepaws were also noted in some of the 17 beavers (number not specified), likely to have resulted, again, from escape

attempts from the traps. Elevated creatine kinase levels, a sign of muscular disease, were present in six individuals, hypothesised to be to be due to increased activity levels from attempting to escape from the traps (Campbell-Palmer et al., 2015a).

Throughout the reintroduction of Eurasian beavers from Norway into Knapdale, Scotland, 20% (n=6) of beavers died during the statutory six-month quarantine period in captivity, despite being housed in purpose-built facilities. Severe parasitism and/or infection contributed to the death of four individuals, and no diagnosis was made with the other two beavers. Goodman et al. (2012) considered that stress related immunocompromise contributed to the deaths. In general, the beavers' health was compromised during the quarantine period, with most animals losing body weight and "body condition" (Jones and Campbell-Palmer, 2014). Two further animals died following release, one of which was an adult male in poor body condition (Goodman et al., 2012). It was suggested that this male most likely did not feed due to a failure to cope with the stress of the translocation and environmental changes (Harrington et al., 2010).

A reintroduction program of Eurasian beavers into Hungary was undertaken between 1996 and 2008. Two beavers died during the period of transport and captivity, but no diagnosis was made. Moreover, one further beaver was found dead within one month of release, but a post-mortem examination was not undertaken due to autolysis. Another individual died as a result of intraspecific aggression at the release site (Bajomi, 2011).

Captivity related morbidity was reported when undertaking the Knapdale beaver reintroduction. Similarly to the case reported by Campbell-Palmer et al. (2015a), abnormal tooth wear resulted in postponement of release in a male beaver. Pulp exposure of the upper right incisor and inflammation of the upper lip was reported (Goodman et al., 2012), which may have resulted from escape attempts as reported in Tayside. Other reports of dental disease of captive beavers have appeared in the literature. Inadequate wear due to inappropriate feeding resulted in malocclusion of the incisors in a three year old captive Canadian beaver in Cheong-ju Zoo, South Korea (Kim et al., 2005a; Kim et al., 2005b). A four year old captive Canadian beaver from National Zoological Gardens of Pretoria, South Africa presented with an infected lesion to the right upper lip, resulting from overgrowth of the mandibular incisor. This tooth had elongated due to loss of the upper incisor. Since the beaver arrived at the zoo in this condition, it was unknown how the incisor was lost (Steenkamp et al., 2009). In these cases in South Korea and South Africa, the beavers were held in captivity for longer periods of time than would be expected to be necessary during a translocation and the implications for translocations should therefore be drawn carefully.

Trauma from aggressive interactions between beavers, and self-harm by individual beavers, have been reported. Five Eurasian beavers became trapped within a lodge as a result of extreme frosts in Mongolia (Saveljev et al., 2016). It is unclear how long the animals were trapped for, but on release by local residents, the authors' concluded that all five beavers had evidence of tail trauma consistent with self-cannibalism. It was hypothesized that this trauma had resulted from the severe stress of the captive environment (Saveljev et al., 2016). A Eurasian beaver kit held in a captive collection in

England was found in its enclosure with multiple wounds caused by intraspecific aggression (O'Brien et al., 2018). Treatment of this case took several months, and complications arose, including abscessation of some wounds and proprioceptive deficits, although the beaver did recover (ibid.). Although this case occurred in a captive collection in which the beavers had been in captivity longer than would be expected to occur during a translocation, it is not possible to rule out this aggression occurring under conditions of stress associated with translocation. Intraspecific aggression has been reported in the wild for beavers (Stefen, 2018), and a recent study by Mayer and colleagues demonstrated an inverse density-dependent territorial behaviour pattern in Eurasian beavers; at lower population densities, intraspecific aggression appeared to increase (Mayer et al., 2020). Resource competition in excessively large groups has also been noted to lead to aggression in free-living Eurasian beavers (Kitchener, 2001).

It is possible that in a captive setting, aggression is heightened due to stress and inappropriate husbandry conditions, such as lack of space. Post-mortem examination reports from beavers in Scotland provide evidence of aggression in captivity. An adult female held in a captive collection in Scotland was found on post-mortem examination to have died from blunt trauma. This individual was housed with a male, and it is possible that the death was a result of intraspecific aggression (Brownlow, 2011). Moreover, as previously mentioned, one beaver died as a result of intraspecific aggression after reintroduction into Hungary; two animals were released together, and one inflicted lethal injuries upon the other. This aggression was thought to have resulted from the stress of translocation and release into a new environment (Bajomi, 2011).

Other cases of wounds are reported for captive Eurasian beavers, most likely as a result of inappropriate housing facilities. Injuries and abrasions to the tails and plantar surfaces of feet were found on post-mortem examination of five beavers which died whilst in quarantine as part of the Knapdale reintroduction (Cranwell 2009a, 2009b, Collins 2009, Howie 2009, Deuchande 2009). In one of these cases a severe tail wound progressed to osteomyelitis of the caudal vertebrae - it is unclear whether the original wound was caused by intraspecific aggression (Collins, 2009). Given the nature of the abrasions, lesions in these beavers are likely to have occurred due to unnatural substrate in captive enclosures, such as concrete. Inappropriate use of 'hot wire' fencing has resulted in mortalities of several beavers. The animals bit down on the wire, and their front teeth became locked behind it, trapping them (Campbell-Palmer, Schwab, et al., 2015). This highlights the importance of appropriate husbandry conditions for maximising reintroduction success.

The evidence outlined above indicates that captivity during translocation can result in diseases associated with trapping, stressors and immunosuppression, intraspecific aggression, and housing facilities and, therefore, captivity during translocation is considered a population hazard.

Risk Assessment

Exposure assessment

Beavers will be required to undergo a period of time in captivity as part of the reintroduction program, including trapping and transport. Therefore, there will be multiple opportunities for morbidity and mortality to occur as a result, either through wounds and abrasions resulting from inappropriate husbandry measures, stress related immunocompromise, or trauma as a result of aggression. There is a medium likelihood that translocated beavers will be exposed to this hazard, given the numerous previous reports of diseases associated with captivity described above. Beavers originating from a free-living environment may be more prone to stress-related diseases during translocation due to having no previous experience in a captive setting.

Consequence assessment

The probability of one translocated beaver suffering from morbidity or mortality as a result of captivity during translocation is medium.

The probability of beavers suffering from wounds caused by intraspecific aggression or self-trauma due to stress is medium. The likelihood of severe disease and death from wounds is high as even minor wounds and abrasions can lead to severe consequences in beavers in captivity as a result of infection (Campbell-Palmer and Rosell, 2015). The probability of beavers suffering from injury as a result of inappropriate enclosure conditions, for example inappropriate fencing or substrate, is medium. The probability of dental disease occurring during the period of time held in captivity is predicted to be low, as these diseases are likely to take several months to arise. The likelihood of severe dental diseases is low. The probability of disease and death occurring as a result of stress-related immunosuppression in captivity is high.

The probability of negative economic consequences occurring due to captivity during translocation is low, and there is a very low likelihood of failure of the reintroduction program due to this hazard. Several other reintroduction programs of Eurasian beavers have been successful despite numerous deaths occurring in captivity. The probability of environmental or ecological consequences as a result of captivity during translocation is negligible.

Risk estimation

There is a medium likelihood of the reintroduced population of beavers being exposed to the hazards of captivity during translocation. There is a medium or high likelihood that beavers will be exhibit disease (depending on the disease as indicated in the consequence assessment) as a result of captivity, and a medium likelihood of severe consequences, such as death, in the case of captivity-associated morbidity occurring. There is a low probability of economic and biological impacts of a failed reintroduction. The overall risk is estimated to be MEDIUM.

Risk Management

Risk evaluation

It is necessary to implement mitigation measures to reduce the risk from the hazard of captivity.

Risk management options

Duration in captivity should be minimized to reduce the propensity to develop stressor associated disease, dental disease, housing-related injury and aggression-associated injury. Stress reduction should be maximized through appropriate husbandry measures, such as good hygiene, appropriate nutrition, appropriate stocking densities and good enclosure design. Naturalistic substrates should be used wherever possible to reduce the risk of abrasion injuries.

Table 8 Potential hazards assumed to be of very low, if not negligible risk of disease in translocated beavers (Castor fiber) and destination populations and therefore a detailed disease risk analysis was not completed.

POTENTIAL HAZARD		Susceptibility to infection and/or disease		Other reasons for inclusion	Present in			Reference	Hazard Category
		Beaver	Other rodentia		UK	Scandinavia	central Europe		
Viral	Aujeszky's (Porcine herpes-virus1)	No reports, Likely*	Rats, mice		No	No	No	Ruiz-Fons, 2012	Not assigned
	Borna Disease Virus	No reports, Likely*	White-toothed shrew	Multi-host pathogen	Yes	Sweden	Germany, Switzerland, Austria	Weissenbock 2012a	Not assigned
	Cowpox virus	No reports, Likely*	Bank voles, wood mice		Yes	Norway		Hazel et al., 2000	Not assigned
	Encephalomyocarditis virus (EMCV)	No reports, Likely*	Brown rat, house mouse, wood mouse, bank vole, field vole		Yes	N/K	N/K	Backhans et al., 2013; Kaplan et al., 1980	Not assigned
	Parechovirus B (formerly Ljungan) virus	No reports, Likely*	Bank vole and many small rodents		Yes	Sweden, Finland, Denmark	Germany	Fevola, 2019; Fevola et al., 2020	Not assigned

POTENTIAL HAZARD		Susceptibility to infection and/or disease		Other reasons for inclusion	Present in			Reference	Hazard Category
		Beaver	Other rodentia		UK	Scandinavia	central Europe		
	Louping ill virus	No reports, Likely*	Bank vole, wood mouse		Yes	Norway		Kaplan et al., 1980	Not assigned
	Lymphocytic choriomeningitis virus	No reports, Likely*	House mouse, Apodemus sp.		Yes	N/K		Duh et al., 2014	Not assigned
	Omsk haemorrhagic fever virus	Unknown	Muskrats		No	No	Western Siberia	CDC, 2020	Not assigned
	Pneumonia virus of mice	Unknown	Bank vole, wood mouse	Multiple rodent spp.	Yes	N/K		Schoeb, 2000; Kaplan et al., 1980	Not assigned
	Rabies virus	No reports, Likely*		Multi-host pathogen	No	Yes (Svalbard)		WHO, 2018	Source
	Rotaviruses	No reports, Likely*		Multiple rodent spp.	Yes	Yes		Meredith, 2012	Carrier
	Sendai virus (Para-influenza virus 1)	Unknown	Bank vole, wood mouse, field vole		Yes	N/K		Kaplan et al., 1980	Not assigned
	Tahyna virus (Californian encephalitis)	No reports, Likely*	Rodents	YES	Yes	Yes		Bennett et al., 2011	Not assigned

POTENTIAL HAZARD		Susceptibility to infection and/or disease		Other reasons for inclusion	Present in			Reference	Hazard Category
		Beaver	Other rodentia		UK	Scandinavia	central Europe		
	Theiler's murine encephalomyelitis virus	Unknown	Bank vole, house mouse		Yes	N/K		Lipton et al., 2001	Not assigned
	Tick borne encephalitis virus	No reports, Likely*	Wood mouse, yell-necked mouse, bank vole		Yes	Yes		Michelitsch et al., 2019	Population
Bacterial	Aeromonas hydrophila	Yes		Fish; multi-host pathogen	Yes	Norway		PM report M08K25; Citterio et al., 2015	Not assigned
	Anaplasma phagocytophilum	No reports, Likely*	Bank vole, wood mouse, yellow-necked mouse	Multi-host pathogen	Yes	Norway		Chastagner et al., 2016; Birtles, 2012b	Not assigned
	Arcanobacterium pyogenes	Yes		Multi-host pathogen	Yes	Norway		Jost et al., 1999; M08K31 (Collins, 2009)	Not assigned

POTENTIAL HAZARD		Susceptibility to infection and/or disease		Other reasons for inclusion	Present in			Reference	Hazard Category
		Beaver	Other rodentia		UK	Scandinavia	central Europe		
	Bartonella sp.	No reports, Likely*		Multi-host pathogen	Yes	Norway		Birtles, 2012c	Carrier
	Borrelia burgdorferi	No reports, Likely*		Multi-host pathogen	Yes	Norway		Ytrehus & Vikoren, 2012	Not assigned
	Brucella sp.	No reports, Likely*	Bank vole, Apodemus sp.		Yes	Norway		Hammeri et al., 2015	Not assigned
	Campylobacter sp.	No reports, Likely*	Brown rat, yellow-necked mouse, house mouse		Yes	Norway		Backhans et al., 2013	Not assigned
	Chlamydia sp.	Unknown	Mice, hamsters		Yes	Norway		Speck and Duff, 2012c	Not assigned
	Clostridia sp.	No reports, Likely*		Multi-host pathogen	Yes	Norway		Neimanis and Speck, 2012; Simpson et al., 2008; Krijger et al. 2019	Carrier

POTENTIAL HAZARD		Susceptibility to infection and/or disease		Other reasons for inclusion	Present in			Reference	Hazard Category
		Beaver	Other rodentia		UK	Scandinavia	central Europe		
	Coxiella burnetii	No reports, Likely*	Bank vole, wood mouse, field mouse	Multi-host pathogen	Yes	No		Meredith et al., 2015a; Ruiz-Fons, 2012	Not assigned
	Erysipelothrix rhusiothipiae	No reports, Likely*		Multi-host pathogen	Yes	Norway		Wang et al., 2010	Carrier
	Lawsonia intracellularis	No reports, Likely*	Yellow-necked mouse, house mouse	Multi-host pathogen	Yes	Norway		Weissenbock, 2012b	Carrier
	Listeria monocytogenes	No reports, Likely*		Multi-host pathogen	Yes	Yes		Ferroglio, 2012a	Carrier
	Micrococcus sp.	Yes			Yes	N/K		Cullen, 2003	Carrier
	Mycoplasma sp.	No reports, Likely*	Common vole, bank vole		Yes	Norway		Pawelczyk et al., 2004; Bajer et al., 2001	Carrier
	Pasteurella sp.	No reports, Likely*	Coypu, brown rats		Yes	Norway		Ferroglio, 2012b	Carrier
	Pseudomonas sp.	Yes			Yes	Norway		Cullen, 2003	Not assigned

POTENTIAL HAZARD		Susceptibility to infection and/or disease		Other reasons for inclusion	Present in			Reference	Hazard Category
		Beaver	Other rodentia		UK	Scandinavia	central Europe		
	Rickettsia sp. incl. Anaplasma phagocytophilum	No reports, Likely*	Bank vole		Yes	Norway		Birtles, 2012b	Not assigned
	Staphylococcus sp.	Yes	Bank vole		Yes	Norway		Speck, 2012b; Cullen, 2003	Not assigned
	Yersinia frederikensii	Yes		Multi-host pathogen	Yes	N/K		Campbell-Palmer, 2018; Healing and Greenwood, 1991	Carrier
Fungal	Candida albicans	Yes		Multi-host pathogen	Yes	Norway		Saez, 1976	Carrier
	Dermatophyte sp. (incl. Trichophyton mentagrophyes)	No reports, Likely*	Water vole, field vole, field mouse	Multi-host pathogen	Yes	Norway		Pesterev and Stadukhin, 1987	Carrier
	Enterocytozoon sp.	No reports, Likely*	Bank vole, house mouse, yellow-necked mouse	Multi-host pathogen	No	N/K		Perec-Matysiak et al., 2015	Carrier
	Histoplasma sp.	Unknown	Brown rat, house mouse	Multi-host pathogen	No	N/K		Emmons, 1950	Not assigned

POTENTIAL HAZARD		Susceptibility to infection and/or disease		Other reasons for inclusion	Present in			Reference	Hazard Category
		Beaver	Other rodentia		UK	Scandinavia	central Europe		
Protozoal	Babesia sp.	No reports, Likely*	Water vole, bank vole, yellow-necked mouse	Multi-host pathogen	Yes	Norway		Gelling et al., 2012; Beck et al., 2011	Not assigned
	Encephalitozoon cuniculi	No reports, Likely*	Bank vole, field vole, wood mouse	Red fox	Yes	Norway		Meredith et al., 2015	Carrier
	Entamoeba sp.	No reports, Likely*	Water vole		Yes	Norway		Gelling et al., 2012; Cox, 1987	Carrier
	Hepatozoon sp.	No reports, LIKELY*	Bank vole, field vole, common vole, yellow-necked mouse		Yes	Norway		Laakonen et al., 2001	Not assigned
	Neospora caninum	No reports, Likely*	Water vole, common vole	Multi-host pathogen	Yes	Norway		Fuehrer et al., 2010	Carrier
	Sarcocystis sp. incl. Frenkelia sp.	Yes			Yes	Norway		Cranwell, 2009; Fichet-Calvet et al., 2014	Carrier

POTENTIAL HAZARD		Susceptibility to infection and/or disease		Other reasons for inclusion	Present in			Reference	Hazard Category
		Beaver	Other rodentia		UK	Scandinavia	central Europe		
	Trypanosoma sp.	Unknown		Eurasian Badger	Yes	Norway		Ideozu et al., 2015	Not assigned
	Capillaria hepatica	Yes			Yes	Norway		Fuehrer, 2014	Carrier
	Fasciola hepatica	Yes			Yes	Norway		Shimalov and Shimalov, 2000	Not assigned
	Hymenolepsis sp.	No reports, Likely*	Water vole		Yes	Norway		Gelling et al., 2012	Not assigned
	Taenia sp.	Yes			Yes	N/K		Campbell-Palmer et al., 2015c	Carrier
	Travassosius rufus	Yes			Yes	Yes		Drozdz et al., 2000; Goodman et al., 2014	Carrier
Ectoparasite	Demodex sp.	Yes			Yes	Norway		Izdebska et al., 2016	Carrier
	Ixodes sp.	Yes			Yes	Norway		Haitlinger, 1991, Wodecka and Stotarczak, 2016	Carrier

POTENTIAL HAZARD		Susceptibility to infection and/or disease		Other reasons for inclusion	Present in			Reference	Hazard Category
		Beaver	Other rodentia		UK	Scandinavia	central Europe		
	Mites incl. Schizocarpus sp.	Yes			No	Sweden	SW	Ahlen, 2001; Haitlinger, 1991	Carrier
	Platypsyllus castoris (incl. Leptinillus sp.)	Yes			Yes	Sweden		Duff et al., 2013	Carrier
Non-Infectious	Environmental pollutants	Yes			Yes	N/K		Gizejeweska et al., 2015	Population
	Mortality as a result of general anaesthesia	Yes			N/A	N/A		Swain et al., 1998; Campbell-Palmer et al., 2015a	Not assigned

(*): Because of the paucity of data available on both infectious and non-infectious hazards in free-living beavers, a qualitative judgement of susceptibility to some hazards, based on expert opinion, was used when it could not otherwise be supported by evidence in the scientific literature. Beavers were considered to be “likely susceptible” to those parasites isolated in Rodentia species but also to those multi-host parasites known to infect many other mammalian families and orders. Similarly, non-infectious hazards known to be associated with morbidity and mortality in other mammals were considered ‘likely susceptible’.

Appendix 20 Hazards assumed to be of very low, if not negligible risk of disease in translocated beavers and destination populations and therefore a detailed disease risk analysis was not completed

VIRUSES

- Borna Disease Virus causes severe neurological disease, mainly in horses and sheep but with sporadic cases in several other species (Weissenbock, 2012a). The main host is reported to be the bicoloured white-toothed shrew (Crocidura leucodon) but birds may also act as a reservoir (ibid.). It has not been reported in beavers but has been found in several species in Germany and Sweden.
- Cowpox virus is an orthopoxvirus endemic in European free-living small rodents, in particular voles, regarded as the natural reservoir, which can infect many species including humans (Hazel et al., 2000). There are no reports of infection in beavers but, given the ubiquity of cowpox virus in sympatric species, beavers may be exposed to infection at source or destination sites.
- Encephalomyocarditis virus (EMCV) is a small non-enveloped single-strand virus associated with encephalitis and myocarditis in a number of species, including humans. Pathogenesis appears to be strain and host-specific. It has not been reported in beavers but is found in sympatric rodent species (Kaplan et al., 1980; Backhans et al., 2013).
- Lymphocytic choriomeningitis virus (LCMV) is an arenavirus found in the house mouse but also isolated from other free-living rodents and associated with neurological disease in humans (Duh et al., 2014). It has not been reported in beavers.
- Louping ill virus is a tick-borne flavivirus associated with disease and, occasionally, acute mortality in sheep, red grouse and humans. It has been isolated from wood mice and bank voles in Great Britain (Kaplan et al., 1980) and cervids in Norway (Gao et al., 1993) but has not been reported in beavers.
- Omsk haemorrhagic fever virus is a tick-borne flavivirus carried by a wide range of aquatic rodents, including the water vole and non-native muskrat, in western Siberia, and the cause of haemorrhagic fever and encephalitis in humans (CDC, 2020). It has not been reported in beavers and its narrow geographical distribution suggests that the risk from disease in translocated beavers from this virus is currently negligible.
- Parechovirus B, formerly known as Ljungan virus, has been widely reported in small rodents and is believed to be associated with disease in humans (Fevola, 2020).

There do not appear to be host-specific isolates (ibid.) and so infection of beavers from sympatric species is possible.

- Pneumonia virus of mice is a paramyxovirus known to infect a wide range of rodents and lagomorphs. It has not been reported in beavers but is unlikely to cause disease in immunocompetent hosts.
- Porcine herpesvirus 1 (Aujeszky's Disease virus/Pseudorabies virus) is an alphaherpesvirus associated with rapid onset and usually fatal disease in dead-end hosts, including rats, mice and lagomorphs (Ruiz-Fons, 2012). Wild boar are the primary reservoir in parts of north-east Germany but it has not been reported in beavers, and is not currently in Norway or the UK.
- Rabies lyssavirus causes acute and fatal encephalitis in all mammals and has been eradicated from most of Europe following vaccination of the primary host, red foxes (WHO, 2018). Rabies lyssavirus remains present in focal areas of Eastern Europe. As mammals, beavers are susceptible to infection with rabies virus. Rabies lyssavirus is not present in the UK or Norway, although sporadic cases are found on the island of Svalbard as a result of migrating animals from mainland Russia. As Svalbard is approximately 2000km from mainland Norway there is considered to be limited likelihood of transmission to humans or animals in Norway.
- Rotavirus infection and associated enteritis has been reported in free-living squirrels, mice and rats (Meredith, 2012). No reports have been found in free-living beavers. Immune status is important in determining the severity of disease (ibid.) so immunocompromised animals may be expected to experience severe morbidity.
- Sendai virus (Parainfluenza 1) is found in a wide range of free-living small rodents (Kaplan et al., 1980), including those sympatric with beavers. It is not known if beavers are susceptible to infection.
- Tahyna virus (Californian encephalitis) is endemic throughout Europe where its main reservoir is the mosquito vector, amplified by a broad range of mammalian hosts, and which causes encephalitis in humans (Bennett et al., 2011). It is not known if rodents, including beavers, are susceptible to infection.
- Theiler's murine encephalomyelitis virus has been reported in free-living rodent species (Kaplan et al., 1980). It is not known if beavers are susceptible to infection but pathogenicity is likely to be low in immunocompetent hosts.
- Tick-borne encephalitis virus is one of the main arboriviruses of Eurasia, which is adapted to a broad range of vertebrate host species and, primarily, transmitted via hard ticks (Michelitsch et al., 2019). Small mammals are considered to be the main reservoirs of infection and have been shown to act as hosts for co-feeding ticks (Cull et al., 2017) with wild cervids acting as the main reservoir of the tick vector (ibid.). There are no reports of infected beavers but as they share habitat with reservoir species, and can be infected by the vector, they may be susceptible to infection. TBEV has recently been shown to be present in England, in Thetford Forest, East Anglia, and the Hampshire/Dorset border (Holding et al., 2019; Holding et al., 2020). The virus sequences are closest to previously isolated TBEV strains from Norway and the Netherlands respectively and are believed to have been introduced by migratory birds (ibid.). As a result, translocation of an infected beaver does not constitute a source hazard but may, if beavers are found to be susceptible

to disease following infection, constitute a population hazard and merit further assessment in the future.

BACTERIA

- Aeromonas hydrophila is an aquatic gram-negative bacterium of amphibians and fish responsible for skin infections and gastroenteritis and occasional systemic disease in other hosts. It has been found as a suspected opportunist pathogen in a beaver M08K25 associated with fatal myocarditis.
- Anaplasma phagocytophilum, a tick-borne rickettsial parasite, is a multi-host pathogen for which infection has been reported in many domestic and wild animals (Birtles, 2012b). It is the causative agent of tick-borne fever (TBF) in domestic ruminants and zoonotic disease in humans. Infections have been reported in the bank vole and other rodents (ibid.) which may act as asymptomatic reservoirs. It is not known if beavers are susceptible to infection.
- Arcanobacterium pyogenes is a commensal bacterium of the upper respiratory and genital tracts and opportunistic pathogen of many domestic animals associated with a wide range of suppurative infections (Jost et al., 1999). It was isolated from a beaver M08K31 which died in quarantine following a tail injury (Collins, 1999).
- Bartonella species are Gram negative bacteria exploiting a wide range of mammalian species, including humans, domestic animals and wildlife, as reservoir hosts. Bartonella species are generally species specific, causing chronic but asymptomatic infections in their hosts (Birtles, 2012c). No reports of infection of beaver with Bartonella species have been found but 51% (n=93/183) of water voles were positive for Bartonella species in a study by Oliver et. al. (2009).
- Borrelia burgdorferi is a bacterium responsible for a tick-borne disease, Lyme borreliosis. Its life cycle is maintained by hard ticks in the genus Ixodes and a wide spectrum of mammalian, avian and reptilian hosts (Ytrehus and Vikøren, 2012). B. burgdorferi generally establishes persistent infections with minimal harm to its natural hosts, with clinical disease usually developing only in aberrant hosts such as humans and domestic animals (ibid.). Beavers may be susceptible to infection as they may harbour the vector.
- Brucella species are facultative intracellular pathogens responsible for disease and economic losses in domestic animals and multi-organ disease in humans (Hammeri et al., 2015). Brucella species have been isolated from bank voles and Apodemus species in Europe which may act as a reservoir of infection for other species (ibid.). There are no reports of infection of beavers, but they may be susceptible to infection as they are sympatric with other hosts.
- Clostridia species are obligate anaerobic bacteria that form spores to survive adverse environmental conditions. They are widely distributed in soil, water, decaying organic matter and on mucosal surfaces or within digestive tracts of humans and animals. They produce toxins which are responsible for their pathogenicity (Neimanis and Speck, 2012). Clostridium botulinum is the most significant and widely reported species which, in the wild predominantly affects birds, particularly waterfowl, but mammals are also susceptible. Botulism in wildlife occurs following the ingestion of preformed toxin. Clostridium piliforme is the

causative agent of Tyzzer's disease, an acute disease most commonly seen in laboratory animals and commercially bred rabbits but that has also been described in free-ranging mammals, including in a wild Eurasian otter (Lutra lutra) cub on the isle of Harris, Scotland (Simpson et al., 2008). Zoonotic strains of C. difficile have been found in small rodents, including the muskrat, in the Netherlands (Krijger et al., 2019). No reports have been found in beavers.

- Coxiella burnetii is a worldwide distributed bacterium, responsible for Q fever, a disease affecting humans and animals. Infection is usually subclinical but can produce acute disease in animals (abortion in farmed ruminants) (Ruiz-Fons, 2012). Virtually all animals are considered able to harbour C. burnetii. Seroprevalence in UK rodents was reported as 17.3% (Meredith et al., 2015a). No reports of infection or disease have been found in beavers.
- Erysipelothrix rhusiopathiae is an ubiquitous and environmentally persistent facultative gram-positive bacillus found as a commensal or pathogen in at least 50 species of wild mammals, including rodents, and over 30 species of wild birds (Wang et al., 2010). It is recognised as a cause of occupational disease in humans with strains of varying pathogenicity (ibid.). E. rhusiopathiae has not been found in beavers and it is assumed that it would be of low pathogenicity in otherwise healthy animals.
- Lawsonia intracellularis is an obligate intracellular bacterium found worldwide that is capable of infecting a wide range of species but only occasionally causing disease in wildlife hosts (Weissenbock, 2012b). Rodent species, including the house mouse and yellow-necked mouse, and the red fox are likely carriers (ibid.). Infection has not been reported in beavers.
- Listeria monocytogenes is a Gram-positive bacterium found worldwide and responsible for a disease, listeriosis, that can affect both animals and humans (Ferroglio, 2012a). It is found in soil, decomposing matter but also in the gastrointestinal tract of healthy animals of many species, including rodents. To date infection has not been reported in beavers.
- Micrococcus species are environmental gram-positive bacteria that have been isolated from the eyes of 5/16 Canadian beavers with no signs of ocular disease (Cullen, 2003). Micrococcus species are not considered pathogenic in otherwise healthy hosts.
- Mycoplasma species are a numerous class of wall-less bacteria, mainly non-pathogenic, although some species are responsible for respiratory disease, that have been isolated from the bank and common voles (Bajer et al., 2001; Pawelczyk et al., 2004). Normally non-pathogenic Mycoplasma species may cause disease when host immunocompetence is reduced (Nicholas and Giacometti, 2012). There have been no reports in beavers.
- Pasteurella species are worldwide multi-host pathogens, often found as commensal organisms in a wide range of hosts, but reported as the cause of pneumonia and septicaemia in the red fox, brown rat and coypu (Ferroglio, 2012b). Stressors such as weather changes and poor body condition are associated with an increased likelihood of mortality in wildlife species (ibid.). There have been no reports in beavers.

- Pseudomonas species are gram-negative rod bacteria of which the most common, P. aeruginosa, is found in the environment and as a commensal organism, occasionally causing abscesses in rodents. It has been reported in the eye of an otherwise healthy Canadian beaver (Cullen, 2003).
- Stapphylococcus species are gram-positive facultative bacteria commonly associated with suppurative infections and abscess formation but may also cause septicaemia and toxic shock syndrome (Speck, 2012b). Different Stapphylococcus species are associated with different animal species but most diseases of wildlife are attributed to S. aureus (ibid.). S. stephanovicii has been found in the bank vole and the field mouse in association with enteric and skin disease (ibid.). Stapphylococcus species were found in the eyes of 3/10 otherwise healthy beavers (Cullen, 2003).
- Yersinia frederikensii is a non-pathogenic gram-negative coccobacillus that has been isolated from bank voles in Dorset, England (Healing and Greenwood, 1991) and a beaver in Devon (Campbell-Palmer, 2018). It is unlikely to be pathogenic in otherwise healthy animals.

FUNGI

- Candida albicans is an opportunistic yeast which has been reported in association with a cutaneous infection in a Canadian beaver (Saez, 1976). It is unlikely to be pathogenic in an otherwise healthy animal.
- Dermatophyte species are ubiquitous organisms responsible for skin diseases in humans and animals, of which the most common is ringworm (Pesterev and Stadukhin, 1987). Cases in beavers have not been reported but, given the ubiquity and multi-host potential of the agent, beavers may be susceptible.
- Enterocytozoon species are intracellular microsporidial parasites of enterocytes associated with chronic and potential mortality in humans and animals (Perec-Matysiak et al., 2015). Rodents may act as reservoir species (ibid.).

PROTOZOA

- Anaplasma phagocytophilum is an emerging tick-borne pathogen causing disease in a wide range of mammals, including humans (Chastagner et al., 2016). It has not been found in beavers but several species of sympatric vole are believed to act as reservoirs (ibid.).
- Babesia species are the causative agent of zoonotic babesiosis with widespread prevalence in Europe. Rodents are regarded as an important reservoir with transmission via the tick vector Ixodes ricinus but there are no reports of associated disease (Beck et al., 2011). Babesia species have not, to date, been identified in beavers but beavers may have potential to act as a reservoir.
- Entamoeba species are commensal intestinal parasites ubiquitous in species including rodents, rarely associated with dysentery (Cox, 1987). Pathogenicity is assumed to be low in otherwise healthy adult animals.
- Encephalitozoon cuniculi is an obligate intracellular spore-forming protozoan which is the causative agent of encephalitozoonosis, an important emerging disease of

humans and animals which, in addition to its main hosts of rabbits and hares, has been found in several species of small rodent and red foxes (Meredith et al., 2015b). Infection in rodents is usually asymptomatic but infected animals can exhibit neurological signs and renal failure (ibid.). A strain previously isolated from small rodents has been reported in farmed Arctic foxes and mink in Norway (Akerstedt, 2006).

- Hepatozoon species are obligate intra-erythrocytic parasites found in a wide range of mammals that have not been associated with disease in rodent hosts (Laakkonen et al., 2001). As sympatric species, beavers may be susceptible to infection.
- Neospora caninum is a coccidian species closely related to Toxoplasma gondii that is a recognised pathogen of dogs and cattle (Fuehrer et al., 2010). Rodents may play a role as intermediate hosts in the sylvatic cycle (ibid.). It is not known whether beavers are susceptible to infection.
- Sarcocystis species are obligate intracellular protozoa with a complex indirect life cycle which have been reported in a beaver, M08K20, as an incidental finding (Cranwell, 2009). Infection is usually asymptomatic in the final host, while disease may be seen in intermediate hosts (Formisano et al., 2013). The beaver's possible role as either intermediate or final host is not known.

ENDOPARASITES

- Capillaria hepatica is a zoonotic nematode with worldwide distribution described in more than 90 rodent host species (Fuehrer, 2014). Adult worms invade the liver of the host (usually rodents) and lay ova in the surrounding parenchyma. Ova are not passed in the faeces of the host, being released in the environment only when the host dies and decomposes (ibid.). Capillaria hepatica has been reported in beavers (ibid.) but is considered of low pathogenicity.
- Fasciola hepatica is a trematode found worldwide that colonises the bile ducts of its definitive host, most commonly domestic ruminants, with aquatic lymnaeid snails as its intermediate host. It is the cause of considerable economic losses from livestock morbidity and occasional mortality. It has been reported in beavers in Belarus (Shimalov and Shimalov, 1999).
- Hymenolepis species are cestode parasites found in humans and rodents and has been detected in water voles in Great Britain (Gelling et al., 2012). There are no reports of infection in beavers but, as sympatric species, they may be susceptible.
- Taenia species are small intestinal cestodes with a worldwide distribution. The life cycle is indirect, with small mammals/herbivores acting as intermediate hosts and carnivores being the final hosts. Pathogenicity is likely to be very low in the final host, unless there is a high parasite burden (Taylor et al., 2007). A cyst of Taenia martis was detected by PCR in a Bavarian beaver by Campbell-Palmer et al., (2015c).
- Travossosius rufus is a species-specific nematode that has been reported in numerous studies of beavers (Goodman et al., 2014; Drozdz et al., 2004). It is assumed to be of low pathogenicity in otherwise healthy animals.

ECTOPARASITES

- Demodex species are arachnid mites, with a worldwide distribution and likely to be host-specific. D. castoris has been reported from beavers in Poland (Izdebska et al., 2016). Demodex species are not normally pathogenic in immunocompetent hosts.
- Ixodes species are ticks endemic to the UK, with many avian and mammalian species involved in the life cycle. Both Ixodes ricinus and I. hexagonus have been reported on free-living beavers (Wodecka and Stotarczak, 2016; Haitlinger, 1991). Ixodes species are vectors for a number of parasites that beavers may be susceptible to. Disease associated with tick parasitism (excluding tick-borne pathogens) is likely to be intensity-dependent and related to irritation and anaemia.
- Mites and lice are usually host-specific ectoparasites. Schizocarpus species have been identified in Eurasian beavers (Ahlen, 2001; Haitlinger, 1991). Heavy infestations in other species can cause pruritus and anaemia. It is not known if infection is associated with disease in beavers but it is assumed that pathogenicity will be low in healthy adult animals.
- Platypsyllus castoris, the Beaver Beetle, is a species-specific obligate ectoparasite of beavers which has been widely found in free-living beavers, including in Great Britain (Duff et al., 2013). It is not believed to be associated with disease in otherwise healthy animals.

NON-INFECTIOUS

- Heavy metal traces including cadmium, lead, copper, mercury and zinc, have been found in tissues from beavers in agricultural areas in Poland, remote from industrial centres (Gizejeweska et al., 2015). Beavers may be susceptible to toxicity from bioaccumulation of pathogenic elements.
- Reports of beavers dying during general anaesthesia (Helen Roberts, pers. comm.) suggest that the species may be susceptible to side effects associated with anaesthetic drugs or stressors associated with anaesthesia. Canadian beavers exhibit bradychardia when diving and also when threatened on land (Swain et al., 1998).

List of tables

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Appendices

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