

Confidential Report for Discussion— Not to be Circulated

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



Photo credit: Dave Butcher, The Wildwood Trust, Kent

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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 5 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: hantaviruses (PUUV); gram-negative enteric bacteria; *Streptococcus castoreus*; *Stichorchis subtriquetrus*; *Trichinella* spp., *Toxoplasma gondii*; *Emmonsia crescens*; SARS-CoV-2; road traffic collisions; persecution; captivity during translocation; *Yersinia enterocolitica* and *Y. pseudotuberculosis*; *Leptospira* spp.; *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 the Steering Committee 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* spp. 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, and 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 DRAHS at ZSL for use in free-living wildlife from the World Organization for Animal Health Import Risk Analysis, and conforming to IUCN guidelines, allows for ready re-analysis and revised risk estimation. In conclusion if the benefits of translocation are seen by the Steering Committee to exceed the costs, we recommend continued scrutiny and evaluation of the risks from disease as a disease risk management and post-release health surveillance protocol is drawn up.

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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## 1.0 Introduction

The Eurasian Beaver (*Castor fiber*) is believed to have become extinct in Great Britain during the 16<sup>th</sup> 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 authorized and unauthorized 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, as far as we are aware, have been sourced from Scotland (*ibid.*).

### 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.

### 1.3 The Changing Aims of this Disease Risk Analysis

The aims of this disease risk analysis (DRA), reported here, have changed during the work programme, completed over four months between February and June 2020. We have adapted to these changing objectives and some of the disease risk analyses carried out in the early part of the study refer to the original goals. From 1<sup>st</sup> February 2020, when the study commenced, the objective was ‘the analysis of the risks from disease from the translocation of beavers from Scotland, or continental Europe, to England’. Translocation of captive beavers in zoological collections was not considered because of the understanding that beavers in zoological collections were likely to have had exposure to exotic rodents and therefore to non-native parasites. From 3<sup>rd</sup> March 2020 the aim had changed to ‘assess the risks of disease related to the conservation translocation of beavers from either Norway or any area of the UK (including fenced enclosures where the beavers are not in contact with exotic rodents) to England’. This change was made on the basis of the results of a published disease risk analysis on *Echinococcus multilocularis* in relation to the import of beavers to the UK (Roberts, 2012). On 13<sup>th</sup> May 2020 the objectives of the study changed ‘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.’ 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 of these enclosure beavers have been exposed to exotic, non-native rodents in zoological collections, directly or indirectly, 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.

## 2.0 Assessing the risks from disease in wildlife translocations for conservation purposes

Wildlife translocations for conservation purposes (reintroduction, reinforcement, ecological replacement and assisted colonization) 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 of its associated parasites (Davidson and Nettles, 1992). The potential impact of infectious disease on the outcome of wildlife conservation interventions has only recently been recognized 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 World Organization for Animal Health’s (OIE)(Murray *et al.*'s (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.

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

### 3.0 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.

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 and the Steering Committee responsible for plans to reintroduce beavers to England through this report. The intention is that the Steering Committee 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.

### 4.0 Materials and methods

In this report we use the Sainsbury and Vaughan-Higgins' (2012) method (ZSL method) 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 World Organization for Animal Health (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). We included evidence for susceptibility of beavers, other rodents and other mammals to each potential hazard,

or similar agents of disease, in carrying out our evaluation. 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.

**STRESS 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 World Organization for Animal Health (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.

## 5.0 Results

### 5.1 Translocation Pathway

Following guidance from Natural England, 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 2000km if considering southern Norway as a source and 500km 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 1600km 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 PUUV; *Echinococcus multilocularis* and *Trichinella* spp..
- Fully assessed CARRIER HAZARDS included *Leptospira* spp.; *Yersinia enterocolitica* and *Y. pseudotuberculosis*; *Mycobacteria* spp.; *Emmonsia crescens*; gram-negative enteric bacteria; *Streptococcus castoreus*; *Stichorchis subtriquetrus*; *Toxoplasma gondii* and *Eimeria* spp.
- 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 SEOV and 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* spp. (risk to domestic and free-living wild animals).

Fifty-seven potential hazards received detailed scientific review as described in Appendix 1 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 improve.

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 <i>Rodentia</i> susceptibility to infection and/or disease	Reference	Hazard Category
Viral	Hantaviruses – SEOV, TATV	N/K	YES	Duggan <i>et al.</i> , 2017; Pounder <i>et al.</i> , 2013; Thomason <i>et al.</i> , 2017	DESTINATION
	Hantaviruses – PUMV			Vapalahati <i>et al.</i> , 2003	SOURCE
	SARS-CoV-2	N/K	YES	Chan <i>et al.</i> , 2020; Bao <i>et al.</i> , 2020	POPULATION
Bacterial	<i>Leptospira</i> spp.	YES (I, D)	YES	Nolet <i>et al.</i> , 1997	CARRIER
	<i>Francisella tularensis</i>	YES (I, D)	YES	Morner <i>et al.</i> , 1988; Mörner & Sandstedt, 1983; Schulze <i>et al.</i> , 2016	SOURCE
	<i>Yersinia pseudotuberculosis</i> and <i>Y. enterocolitica</i>	YES (I, D)	YES	Nolet <i>et al.</i> , 1997	CARRIER
	Gram-negative enteric bacteria	YES (I, D)	YES	Pratama <i>et al.</i> , 2019; Pilo <i>et al.</i> , 2015; Dollinger <i>et al.</i> , 1999	CARRIER
	<i>Streptococcus castoreus</i>	YES (I, D)	NO	Lawson <i>et al.</i> , 2005; Schulze <i>et al.</i> , 2015	CARRIER
	<i>Mycobacterium</i> spp.	YES (I, D)	YES	Gavier-Widen <i>et al.</i> , 2012; Nolet <i>et al.</i> , 2007	UNCLASSIFIED CARRIER

Endoparasites	<i>Stichorchis subtriquetrus</i>	YES (I, D)	NO	Demiaszkiewicz <i>et al.</i> , 2014	CARRIER
	<i>Echinococcus multilocularis</i>	YES (I, D)	YES	Barlow <i>et al.</i> , 2011; Britton and Barlow, 2019	SOURCE
	<i>Trichinella</i> spp.	YES (I)	YES	Seglina <i>et al.</i> , 2015; Rozycki <i>et al.</i> , 2020	SOURCE
Protozoa	<i>Toxoplasma gondii</i>	YES (I, D)	YES	Herrmann <i>et al.</i> , 2013	CARRIER POPULATION
	<i>Giardia duodenalis</i>	YES (I)	YES	Paziewska <i>et al.</i> , 2007; Tsui <i>et al.</i> , 2018; Sroka <i>et al.</i> , 2015	UNCLASSIFIED
	<i>Cryptosporidium parvum</i>	YES (I)	YES	Paziewska <i>et al.</i> , 2007; Mackie, 2014	UNCLASSIFIED
	<i>Eimeria</i> spp.	YES (I)	YES	Demiaszkiewicz <i>et al.</i> , 2014; Campbell-Palmer <i>et al.</i> , submitted	CARRIER
Fungi	<i>Emmonsia crescens</i>	YES (I, D)	YES	Morner <i>et al.</i> , 1999; Dolka <i>et al.</i> , 2017	CARRIER
Non-Infectious	Road traffic collisions	YES	NO	Brazier <i>et al.</i> , 2020; Campbell-Palmer <i>et al.</i> , 2015b; Stefen, 2018	POPULATION
	Captivity during translocation	YES	NO	Harrington <i>et al.</i> , 2010; Goodman <i>et al.</i> , 2012	POPULATION
	Illegal persecution	YES	NO	Campbell-Palmer <i>et al.</i> , 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. 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. One hazard was estimated to be of negligible risk (*Mycobacterium* spp. (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* spp.; *Mycobacterium* spp. (risk to beavers)), eleven medium risk (Hantaviruses (PUMV); gram-negative enteric bacteria; *Streptococcus castoreus*; *Stichorchis subtriquetrus*; *Trichinella* spp., *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* spp.; *Echinococcus multilocularis*).

\*Risk evaluated at 05 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.



#### 5.4.1 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, 2019). 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 (Vaehri *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 Puumala-virus (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 (Vaehri *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.

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

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

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 (Vapalahati *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. 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: Vapalahati *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 (Tatanale-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, it 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 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 (ECDC, 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 Tatanale-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.

#### 5.4.2 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 *et al.*, 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 anthroozoonosis, 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; Z. Shi and Hu, 2008; Wu *et al.*, 2005). 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 (Cheng *et al.*, 2007; Guan *et al.*, 2003; Wendong Li *et al.*, 2005; 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 (Balkema-Buschmann *et al.*, 2020; Shi *et al.*, 2020). Several preliminary reports have highlighted the ability of SARS-CoV-2 to infect ten non-human mammalian hosts: domestic cats (*Felis catus*), domestic dogs (*Canis familiaris*), transgenic house mice (*Mus musculus*), domestic ferrets (*Mustela putorius furo*), American mink (*Neovison vison*), fruit bats (*Rousettus aegyptiacus*), Syrian hamsters, Malayan tigers (*Panthera tigris jacksoni*), Amur tigers (*Panthera tigris altaica*), African lions (*Panthera leo*) and rhesus macaques (*Macaca mulatta*) (Balkema-Buschmann *et al.*, 2020; Bao *et al.*, 2020; Chan *et al.*, 2020; Deng *et al.*, 2020; Goumenou *et al.*, 2020; ProMed International Society for Infectious Diseases, 2020a; J. Shi *et al.*, 2020; World Organisation for Animal Health (OIE), 2020; Zhang *et al.*, 2020). In eight of these mammalian species (domestic ferrets, Malayan tigers, African lions, domestic cats, fruit bats, Syrian hamsters, American mink and transgenic house mice) it has been associated with disease (Balkema-Buschmann *et al.*, 2020; Bao *et al.*, 2020; Chan *et al.*, 2020; Deng *et al.*, 2020; Goumenou *et al.*, 2020; ProMed International Society for Infectious Diseases, 2020a; J. Shi *et al.*, 2020; World Organisation for Animal Health (OIE), 2020; Zhang *et al.*, 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 (Balkema-Buschmann *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

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. An outbreak of respiratory disease at two American mink farms in the Netherlands was thought to be associated with SARS-CoV-2 after clinically unwell animals at both farms tested positive for the virus (exact numbers not known) (ProMed International Society for Infectious Diseases, 2020a). This suggests that other members of the Mustelidae family may be susceptible to the disease.

Findings by Shi *et al.* (2020) are supported by preliminary results of an experimental study by Balkema-Buschmann *et al.* (2020), who reported that pigs and chickens were not susceptible to intranasal infection with SARS-CoV-2, however the virus could replicate efficiently in ferrets and high viral RNA yields were detected in nasal washes from ferrets two to eight days post infection. Furthermore, 100% (n=3) of non-inoculated ferrets which were kept in contact with experimentally infected ferrets also became infected and viral RNA was present, detected in nasal washing fluids starting at 12 days post contact. SARS-CoV-2 reactive antibodies were detected from day 8 in the inoculated ferrets and in one contact ferret on day 21 (Balkema-Buschmann *et al.*, 2020).

Balkema-Buschmann *et al.* (2020) also experimentally inoculated nine fruit bats intranasally with SARS-CoV-2, 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 (Balkema-Buschmann *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, 2020b; USDA Animal and Plant Health Inspection Service, 2020). These feline cases are of further concern when considered alongside the recent report of a 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), 2020a). The animals had shown mild respiratory disease signs after contact with an infected keeper along with one other Malayan tiger, two Amur tigers, and two other African lions which have not yet been confirmed to be positive for SARS-CoV-2 (Calle, 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



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 13<sup>th</sup> 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 (Kuba *et al.*, 2005; Li *et al.*, 2003). 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 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.

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.

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 6<sup>th</sup> April 2020. The evidence to date shows that at least seven mammal species, including two 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, R<sub>0</sub>, of SARS-CoV-2 in humans has been estimated to be 2 (Li *et al.*, 2020; Liu *et al.*, 2020). 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 al.*, 1985; Kampf *et al.*, 2020). Exposure through contact with infected surfaces could occur in beavers, as can occur for humans (Kampf *et al.*, 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.*, 2020). 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 anthroozoonosis, 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.

#### 5.4.3 Disease Risk Analysis for the Carrier Hazard *Leptospira* spp.

Leptospire 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 leptospire in their urine with warmth and moisture favouring leptospire persistence in the environment (Birtles, 2012). Leptospire 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). Leptospire 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* spp., 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 leptospire (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 asymptotically 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* spp. found dead, recorded lung haemorrhage as the most common lesion, consistent with fatal cases in humans (Marreros *et al.*, 2017).

*Leptospira* spp. 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* spp. 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* spp. in the environment via mucous membranes or skin abrasions as leptospire can survive in water for several months and shedding by infected reservoir hosts is prolonged.

There is scant evidence for *Leptospira* spp. 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* spp. 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 leptospire have been found in potential source sites and *Leptospira* spp. 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 leptospire for prolonged periods and leptospire are able to survive for prolonged periods in the environment, there is a high probability of beavers and other mammals being exposed to *Leptospira* spp. 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 leptospire in their urine into water and adjacent habitat. There is therefore a high likelihood that mammals at the destination site(s) will disseminate *Leptospira* spp. 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* spp. 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* spp. 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* spp. is HIGH.

## **Risk Management**

### ***Risk evaluation***

Based on the risk assessment above, preventative measures should be employed to reduce the risks from *Leptospira* spp. 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, 2012) 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.

## **5.4.4 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.*, 1988; 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* spp.) (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 (World Organisation for Animal Health (OIE), 2020b).

## **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.*, 1988; 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.*, 1988).

In the aquatic cycle, aquatic mammals including voles (*Microtus* spp.), 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* spp. 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* spp.) from the Eastern part of Norway in 2019 (Personal communication, Turid Vikøren, 11th 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 (World Organisation for Animal Health (OIE), 2020b). 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.*, 1988; 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 Organization, 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 (Larsen *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 Organization, 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.*, 1988). 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.*, 1988; 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 Organization, 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 Organization, 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 Organization, 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.

#### 5.4.5 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.*, 2015) 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 (*ibid.*). 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 (Najdenski, 2012). 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* spp. 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* spp. may not always be detected.



#### 5.4.6 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* spp., evaluated elsewhere in this report, *Salmonella* spp., *Shigella* spp. 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 Castelhana, 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*) (*ibid.*) 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* spp. but not *Salmonella* spp. detected in poultry on the same farm and proposed that rodents were not important reservoirs for *Campylobacter* and *Salmonella* spp.. However, Meerburg and Kijlstra (2007) reviewed several studies of *Campylobacter* and *Salmonella* spp. infections of small rodents and concluded that, in agricultural environments, rodents may maintain or amplify reservoirs of *Campylobacter* and *Salmonella* spp. infection.

Sub-clinical carriage of *Salmonella* spp. appears to be common in free-living wild animals (Gaffuri, 2012). *Salmonella* spp., 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* spp. 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* spp. 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, 2012). 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* spp.) in Germany and Spain (Speck, 2012), 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* spp.; *S. enteritidis* 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* spp. (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* spp. with potential virulence factors in pooled faecal samples from 12 free-living beavers in Poland.

Neither *Salmonella* spp. or *Campylobacter* spp. 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* spp. 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 spp. infections may shed bacteria in their faeces and contribute to environmental reservoirs of gram-negative bacterial spp. 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 spp. 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 spp. 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.

#### 5.4.7 Disease Risk Analysis for the Carrier Hazard *Streptococcus castoreus*

*Streptococcus* spp. 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, 2012).

#### Justification of Hazard Status

A novel *Streptococcus* spp. 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* spp. 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.

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

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

Further evaluation between 2010 and 2017 by Mühldorfer *et al.* (2019) of 27 *Streptococcus* spp. 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* spp., 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* spp. can be isolated from bodily fluids including nasal discharges, pus, milk and exudative infected tissues (Speck, 2012). As *Streptococcus* spp. 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* spp. 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 enteritidis* 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.

#### 5.4.8 Disease Risk Analysis for the Carrier Hazard and Hazard for Domestic and Free-living Mammals in England *Mycobacterium* spp.

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.

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



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

***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* spp.) 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).

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

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

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* spp.**

*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* spp. 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* spp. 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* spp. 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* spp. 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* spp., 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* spp. 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* spp. 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* spp. 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 anaesthetised 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* spp. Progress of disease following infection with *Mycobacteria* spp. 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* spp. 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* spp. 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* spp. 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* spp. 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* spp. 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* spp., 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* spp. 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* spp..

##### **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 to 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* spp. (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.

#### 5.4.9 Disease Risk Analysis for the Carrier Hazard *Stichorchis subtriquetrus*

##### **Justification of Hazard Status**

*Stichorchis subtriquetrus*, the beaver fluke, is a trematode of both Eurasian and Canadian beavers, not known to infect other species (Demiaszkiewicz *et al.*, 2016). Its life cycle involves infection of the intermediate host, aquatic snails of *Bithinia*, *Planorbis* and *Lymnaea* spp. (*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 (Drozd *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.*, Drozd *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.

#### 5.4.10 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).

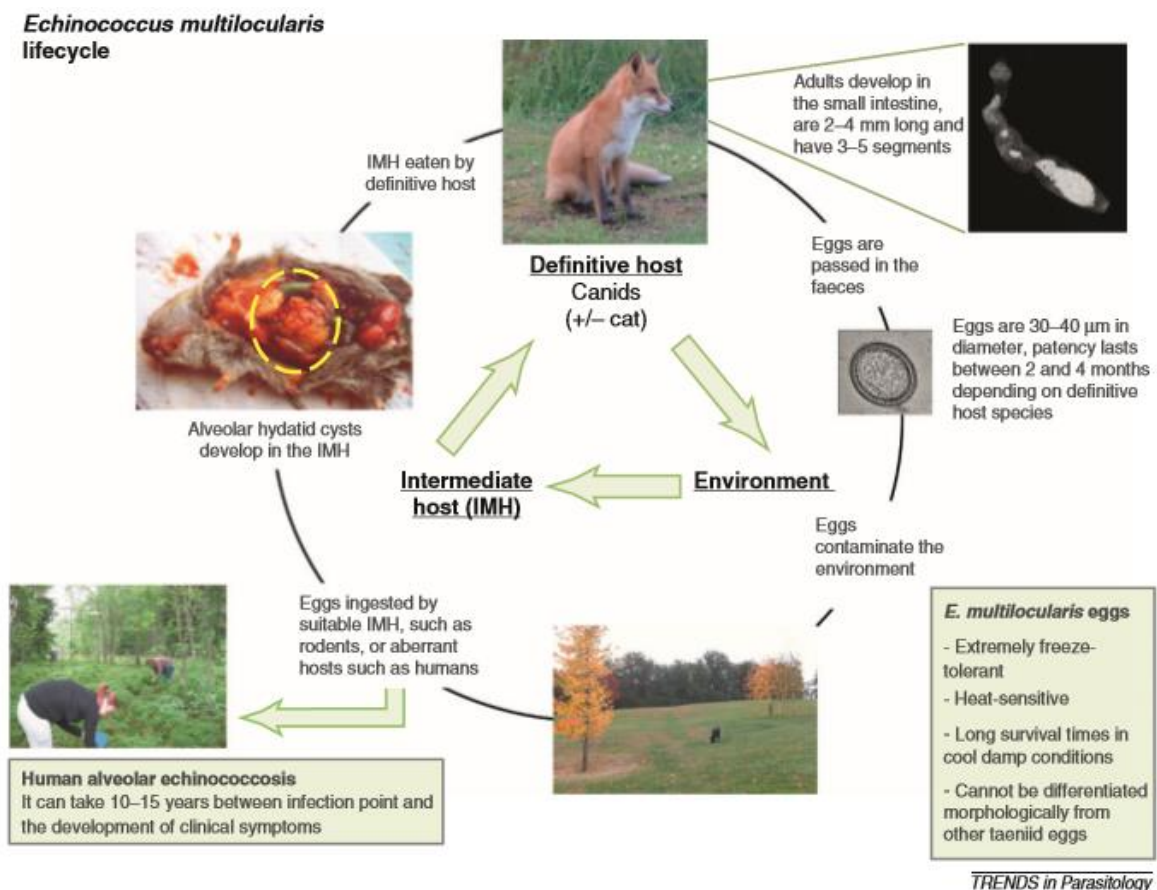


Figure 2: The transmission cycle of *Echinococcus multilocularis* (Source: Davidson *et al.*, 2012)

Intermediate hosts in Europe have been shown in metastudies by Oksanen *et al.* (2016) and Takeuchi-Storm *et al.* (2015) to be primarily *Cricetidae* spp. (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* spp. 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* spp. 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 4°C 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, 80km 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 1600km 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.*, 2015). Campbell-Palmer *et al.* (2015) 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.*, 2015). 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 *Echinococcus multilocularis*.

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



#### 5.4.11 Disease Risk Analysis for the Source Hazard *Trichinella* spp.

*Trichinella* spp. 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* spp. from domestic livestock (EFSA, 2019).

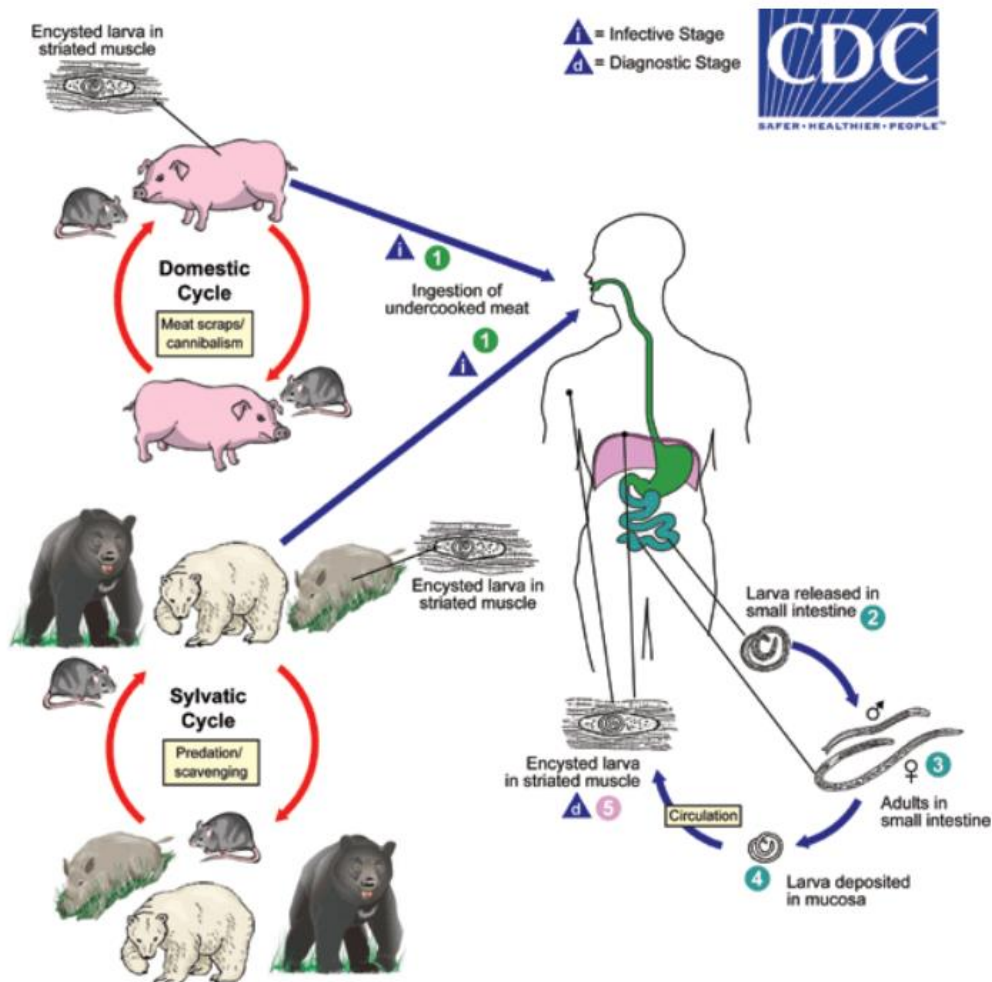


Figure 3: The life cycle of *Trichinella* spp. (Source: CDC, 2020)

*Trichinella* spp. 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* spp. 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* spp. infections in humans are of *Trichinella spiralis*, but infections with other *Trichinella* spp., including *T. britovi*, *T. nativa* and *T.pseudospiralis*, have also been reported (Bronstein and Lukashov, 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* spp. 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, 1200km south of the perceived boundary between *T. nativa* and *T. britovi*. 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* spp. 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, 2016). 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* spp. of interest in Europe, encysted *T. pseudospiralis* larvae lack a surrounding collagen capsule, making visual diagnosis using trichinostomy almost impossible historically (Pozio, 2016). Although the environmental survival of *T. pseudospiralis* is poor compared to other *Trichinella* spp., 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* spp. 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, 2016).

### **Justification of Hazard Status**

After several years of declining prevalence of *Trichinella* spp. 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* spp. 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* spp. 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 carcasses 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* spp. antibodies and found an average prevalence of infection with unspecified *Trichinella* spp. 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* spp. 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* spp., *Trichinella* spp. 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* spp. 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 Lukashov, 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* spp. 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* spp. 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* spp. 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* spp. 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* spp. 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* spp. 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* spp..

### **Exposure assessment**

There is a high likelihood that a sympatric carnivore or omnivore is infected by predated 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* spp. 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 -20°C 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* spp. cycles in Europe is facilitated by hunter activity and the survival of encapsulated *Trichinella* spp. larvae in carcasses is temperature and humidity dependent with optimum survival between 0 and -20°C. As the average winter temperature low in England is 0.9°C (Met Office, 2020) and sport hunting is less common than in Europe, there is a lower likelihood of *Trichinella* spp. establishing in sylvanian cycles in Great Britain compared with the same cycles on the continent. The probability of dissemination of *Trichinella* spp. 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* spp. 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* spp. 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* spp. 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* spp. (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* spp.. There is therefore a negligible likelihood of clinical disease in infected beavers and a negligible likelihood of translocation failure as a result of *Trichinella* spp. 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* spp.. 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* spp..

#### **Risk Management Options**

Detection of immature *Trichinella* spp. 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* spp. 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* spp..

Post-mortem examination of translocated beavers and sympatric species is strongly recommended to assess for entry of *Trichinella* spp. 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.

#### 5.4.12 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 recrudescence 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 ECDC) 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 (Havlíček *et al.*, 2001). In humans, there is evidence to suggest that infection with *T. gondii* leads to slower reaction times (Havlíček *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 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.

#### 5.4.13 Disease Risk Analysis for the Unclassified Hazard *Giardia duodenalis*

*Giardia* spp. 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* spp. 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, All, 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 All)). 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* spp. 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 (USAPA, 1999). Infection with *Giardia duodenalis* spp. 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 (ECCPD, 2017).

#### **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* spp. 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* spp.. Paziowska *et al.* (2007) isolated *Giardia* spp. 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* spp. 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.* 2015; 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* spp. (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* spp. 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* spp. or assemblages at the site.

Robertson and Gjerde (2001) detected *Giardia* spp. 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* spp..

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* spp. 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.

#### 5.4. 14 Disease Risk Analysis for the Unclassified Hazard *Cryptosporidium parvum*

*Cryptosporidium* spp. 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, 2017) 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* spp. 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 XIIId 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* spp. 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 IIId, 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* spp. 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* spp. 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* spp. 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 4<sup>th</sup> 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, 2019). *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* spp. 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* spp. infections in humans (n=5052) of any reporting country in Europe in 2017 (ECPDC, 2019) 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* spp. 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* spp. oocysts (Mackie *et al.*, 2014). 4/6 sites in Knapdale were found to contain *Cryptosporidium* spp. 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 IIc 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* spp. 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* spp.. 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* spp. 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* subtypes IIa 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.

#### 5.4.15 Disease Risk Analysis for the Carrier Hazard *Eimeria* spp.

##### 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* spp. 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* spp. 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* spp. 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* spp. 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* spp. 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* spp. 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* spp. 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* spp. are already present in the UK.

### **Risk estimation**

There is a low probability of beavers being exposed and infected with *Eimeria* spp. 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.

#### 5.4.16 Disease Risk analysis for the Carrier Hazard *Emmonsia crescens*

##### Justification for Hazard Status

*Emmonsia* spp. 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 saprophytic 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* spp.) 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).

Mammalian species	Prevalence (%)				Total
	Immature		Adults		
	M	F	M	F	
<i>Lutra lutra</i> (Otter)	4/8 (50)	4/8 (50)	4/19 (21.1)	6/20 (30)	18/55 (32.7)
<i>Mustela nivalis</i> (Weasel)	–	0/3	2/5 (40)	0/2	2/10 (20)
<i>Mustela erminea</i> (Stoat)	–	–	1/4 (25)	1/3 (33.3)	2/7 (28.6)
<i>Mustela vison</i> (Mink)	–	–	0/1	0/2	0/3
<i>Vulpes vulpes</i> (red Fox)	–	1/3 (33.3)	0/1	0/3	1/7 (14.3)
<i>Martes martes</i> (Pine marten)	–	1/1 (100)	0/1	–	1/2 (50)
<i>Talpa europaea</i> (Mole)	–	–	–	1/3 (33.3)	1/3 (33.3)
<i>Mus</i> sp. (Mice)	–	0/1	1/1 (100)	1/2 (50)	
<i>Rattus norvegicus</i> (Rat)	–	–	1/2 (50)	–	1/2 (50)
<i>Mustela furo</i> (Ferret)	–	–	0/1	–	0/1 (0)
<i>Sorex</i> 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 6: Prevalence of *E. crescens* in British wildlife 2003-5. (Source: Borman *et al.*, 2009)

Hosts	Number examined	Number infected
<i>Mus musculus</i> , house mouse	239	0
<i>Apodemus</i> spp., wood mice	102	0
<i>Sus</i> sp., domestic pig	60	0
<i>Sorex</i> sp., shrew	40	0
<i>Rattus norvegicus</i> , rat	27	0
<i>Clethrionomys</i> sp., red-backed mouse	22	0
<i>Mustela vison</i> , mink	22	0
<i>Vulpes</i> sp., fox	14	0
<i>Microtus</i> sp., vole	10	4
<i>Felis catus</i> , domestic cat	9	0
<i>Lepus</i> sp., rabbit	6	0
<i>Sciurus</i> sp., squirrel	4	0
<i>Mustela</i> sp., weasel	4	0
<i>Arvicola terrestris</i> , water vole	1	1
<i>Lemmus</i> sp., lemming	1	0
<i>Meles meles</i> , badger	1	0
<b>Total</b>	<b>562</b>	<b>5</b>

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

Borman *et al.* (2009) reported that *E. crescens* infection burdens in most animals were low ( $\leq 2$  adiaspores/cm<sup>3</sup> 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/cm<sup>3</sup> 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* spp. 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* spp. 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.

#### 5.4.17 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 1.5km 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, 9 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).

#### 5.4.18 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, 7th 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, 7th May 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).

#### 5.4.19 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.*, 2015). 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 due to increased activity levels from attempting to escape from the traps (Campbell-Palmer *et al.*, 2015).

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.* (2015), 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 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		Beaver susceptibility to infection and/or disease	Other <i>Rodentia</i> susceptibility to infection and/or disease	Other reasons for inclusion	Present in the UK?	Present in Scandinavia	Present in central Europe	Reference	Hazard Category
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	Weissenbo ck 2012	Not assigned
	Cowpox virus	No reports, LIKELY*	Bank voles, wood mice		YES	NORWAY		Hazel <i>et al.</i> , 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 <i>et al.</i> , 2013; Kaplan <i>et al.</i> , 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 <i>et al.</i> , 2020	Not assigned

Louping ill virus	No reports, LIKELY*	Bank vole, wood mouse		YES	NORWAY		Kaplan <i>et al.</i> , 1980	Not assigned
Lymphocytic choriomeningitis virus	No reports, LIKELY*	House mouse, <i>Apodemus</i> spp.		YES	N/K		Duh <i>et al.</i> , 2014	Not assigned
Omsk haemorrhagic fever virus	UNKNOWN	Muskrats		<b>NO</b>	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 <i>et al.</i> , 1980	Not assigned
Rabies virus	No reports, LIKELY*		Multi-host pathogen	<b>NO</b>	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 <i>et al.</i> , 1980	Not assigned
Tahyna virus (Californian encephalitis)	No reports, LIKELY*	Rodents	YES	YES	YES		Bennett <i>et al.</i> , 2011	Not assigned
Theiler's murine encephalomyelitis virus	UNKNOWN	Bank vole, house mouse		YES	N/K		Lipton <i>et al.</i> , 2001	Not assigned

	Tick borne encephalitis virus	No reports, LIKELY*	Wood mouse, yell-necked mouse, bank vole		YES	YES		Michelitsch <i>et al.</i> , 2019	POPULATION
Bacterial	<i>Aeromonas hydrophila</i>	YES		Fish; multi-host pathogen	YES	NORWAY		PM report M08K25; Citterio <i>et al.</i> , 2015	Not assigned
	<i>Anaplasma phagocytophilum</i>	No reports, LIKELY*	Bank vole, wood mouse, yellow-necked mouse	Multi-host pathogen	YES	NORWAY		Chastagner <i>et al.</i> , 2016; Birtles, 2012b	Not assigned
	<i>Arcanobacterium pyogenes</i>	YES		Multi-host pathogen	YES	NORWAY		Jost <i>et al.</i> , 1999; M08K31 (Collins, 2009)	Not assigned
	<i>Bartonella</i> spp.	No reports, LIKELY*		Multi-host pathogen	YES	NORWAY		Birtles, 2012a	CARRIER
	<i>Borrelia burgdorferi</i>	No reports, LIKELY*		Multi-host pathogen	YES	NORWAY		Ytrehus & Vikoren, 2012	Not assigned
	<i>Brucella</i> spp.	No reports, LIKELY*	Bank vole, <i>Apodemus</i> spp.		YES	NORWAY		Hammeri <i>et al.</i> , 2015	Not assigned
	<i>Campylobacter</i> spp.	No reports, LIKELY*	Brown rat, yellow-necked		YES	NORWAY		Backhans <i>et al.</i> , 2013	Not assigned

			mouse, house mouse						
<i>Chlamydia</i> spp.	UNKNOWN		Mice, hamsters		YES	NORWAY		Speck and Duff, 2012	Not assigned
<i>Clostridia</i> spp.	No reports, LIKELY*			Multi-host pathogen	YES	NORWAY		Neimanis and Speck, 2012; Simpson <i>et al.</i> , 2008; Krijger <i>et al.</i> 2019	CARRIER
<i>Coxiella burnetii</i>	No reports, LIKELY*		Bank vole, wood mouse, field mouse	Multi-host pathogen	YES	NO		Meredith <i>et al.</i> , 2015; Ruiz-Fons, 2012	Not assigned
<i>Erysipelothrix rhusiopathiae</i>	No reports, LIKELY*			Multi-host pathogen	YES	NORWAY		Wang <i>et al.</i> , 2010	CARRIER
<i>Lawsonia intracellularis</i>	No reports, LIKELY*		Yellow-necked mouse, house mouse	Multi-host pathogen	YES	NORWAY		Weissenbo ck, 2012	CARRIER
<i>Listeria monocytogenes</i>	No reports, LIKELY*			Multi-host pathogen	YES	YES		Ferroglio, 2012a	CARRIER
<i>Micrococcus</i> spp.	YES				YES	N/K		Cullen, 2003	CARRIER
<i>Mycoplasma</i> spp.	No reports, LIKELY*		Common vole, bank vole		YES	NORWAY		Pawelczyk <i>et al.</i> , 2004;	CARRIER

								Bajer <i>et al.</i> , 2001	
	<i>Pasteurella</i> spp.	No reports, LIKELY*	Coypu, brown rats,		YES	NORWAY		Ferroglio, 2012b	CARRIER
	<i>Pseudomonas</i> spp.	YES			YES	NORWAY		Cullen, 2003	Not assigned
	<i>Rickettsia</i> spp. incl. <i>Anaplasma phagocytophilum</i>	No reports, LIKELY*	Bank vole		YES	NORWAY		Birtles, 2012b	Not assigned
	<i>Staphylococcus</i> spp.	YES	Bank vole		YES	NORWAY		Speck, 2012c; Cullen, 2003	Not assigned
	<i>Yersinia frederikensii</i>	YES		Multi-host pathogen	YES	N/K		Campbell-Palmer, 2018; Healing and Greenwood, 1991	CARRIER
Fungal	<i>Candida albicans</i>	YES		Multi-host pathogen	YES	NORWAY		Saez, 1976	CARRIER
	<i>Dermatophyte</i> spp. (incl. <i>Trichophyton mentagrophyes</i> )	No reports, LIKELY*	Water vole, field vole, field mouse	Multi-host pathogen	YES	NORWAY		Pesterev and Stadukhin, 1987	CARRIER



	<i>Enterocytozoon</i> spp.	No reports, LIKELY*	Bank vole, house mouse, yellow-necked mouse	Multi-host pathogen	NO	N/K		Perec-Matysiak <i>et al.</i> , 2015	CARRIER
	<i>Histoplasma</i> spp.	UNKNOWN	Brown rat, house mouse	Multi-host pathogen	NO	N/K		Emmons, 1950	Not assigned
Protozoal	<i>Babesia</i> spp.	No reports, LIKELY*	Water vole, bank vole, yellow-necked mouse	Multi-host pathogen	YES	NORWAY		Gelling <i>et al.</i> , 2012; Beck <i>et al.</i> , 2011	Not assigned
	<i>Encephalitozoon cuniculi</i>	No reports, LIKELY*	Bank vole, field vole, wood mouse	Red fox	YES	NORWAY		Meredith <i>et al.</i> , 2015	CARRIER
	<i>Entamoeba</i> spp.	No reports, LIKELY*	Water vole		YES	NORWAY		Gelling <i>et al.</i> , 2012; Cox, 1987	CARRIER
	<i>Hepatozoon</i> spp.	No reports, LIKELY*	Bank vole, field vole, common vole, yellow-necked mouse		YES	NORWAY		Laakonen <i>et al.</i> , 2001	Not assigned
	<i>Neospora caninum</i>	No reports, LIKELY*	Water vole, common vole	Multi-host pathogen	YES	NORWAY		Fuehrer <i>et al.</i> , 2010	CARRIER
	<i>Sarcocystis</i> spp. incl. <i>Frenkelia</i> spp.	YES				YES	NORWAY		Cranwell, 2009; Fichet-

								Calvet <i>et al.</i> , 2014	
	<i>Trypanosoma</i> spp.	UNKNOWN		Eurasian Badger	YES	NORWAY		Ideozu <i>et al.</i> , 2015	Not assigned
	<i>Capillaria hepatica</i>	YES			YES	NORWAY		Fuehrer, 2014	CARRIER
	<i>Fasciola hepatica</i>	YES			YES	NORWAY		Shimalov and Shimalov, 2000	Not assigned
	<i>Hymenolepis</i> spp.	No reports, LIKELY*	Water vole		YES	NORWAY		Gelling <i>et al.</i> , 2012	Not assigned
	<i>Taenia</i> spp.	YES			YES	N/K		Campbell-Palmer <i>et al.</i> , 2015c	CARRIER
	<i>Travassosius rufus</i>	YES			YES	YES		Drozd <i>et al.</i> , 2000; Goodman <i>et al.</i> , 2014	CARRIER
Ectoparasite	<i>Demodex</i> spp.	YES			YES	NORWAY		Izdebska <i>et al.</i> , 2016	CARRIER
	<i>Ixodes</i> spp.	YES			YES	NORWAY		Haitlinger, 1991 Wodecka and	CARRIER

								Stotarczak, 2016	
	Mites incl. <i>Schizocarpus</i> spp.	YES			NO	SWEDEN	SW	Ahlen, 2001; Haitlinger, 1991	CARRIER
	<i>Platyssyllus castoris</i> (incl. <i>Leptinillus</i> spp.)	YES			YES	SWEDEN		Duff <i>et al.</i> , 2013	CARRIER
Non-Infectious	Environmental pollutants	YES			YES	N/K		Gizejewsk a <i>et al.</i> , 2015	POPULATION
	Mortality as a result of general anaesthesia	YES			N/A	N/A		Swain <i>et al.</i> , 1998; Campbell-Palmer <i>et al.</i> , 2015	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’.

## 6.0 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 5 non-infectious) potential hazards; and carried out a full disease risk analysis on twenty-one 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 twenty-one hazards selected for full disease risk analysis, eleven were medium risk (hantaviruses (PUUV); gram-negative enteric bacteria; *Streptococcus castoreus*; *Stichorchis subtriquetrus*; *Trichinella* spp., *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* spp.; *Echinococcus multilocularis*) for disease as a consequence of translocation. Of those fourteen high and medium risk hazards, seven are triggered by stressors and later in this discussion we set out how to minimize 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 and 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 un-licensed 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 prioritizing free-living beavers proven to have been born in Great Britain for translocations to England.

*Trichinella* spp. 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* spp. 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 thirty 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 the Steering Committee 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 through 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 1), 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 World Organization for Animal Health's (OIE) 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 19<sup>th</sup> 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 the Steering Committee for Beaver Translocation 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 the 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 ONE

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, 2012). 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, that 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* spp. are Gram negative bacteria exploiting a wide range of mammalian species, including humans, domestic animals and wildlife, as reservoir hosts. *Bartonella* spp. are generally species specific, causing chronic but asymptomatic infections in their hosts (Birtles, 2012). No reports of infection of beaver with *Bartonella* spp. have been found but 51% (n=93/183) of water voles were positive for *Bartonella* spp. 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* spp. are facultative intracellular pathogens responsible for disease and economic losses in domestic animals and multi-organ disease in humans (Hammeri *et al.*, 2015). *Brucella* spp. have been isolated from bank voles and *Apodemus* spp. 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* spp. 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.*, 2015). 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, 2012). 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, 2012). 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* spp. 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* spp. are not considered pathogenic in otherwise healthy hosts.
- *Mycoplasma* spp. 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* spp. may cause disease when host immunocompetence is reduced (Nicholas and Giacometti, 2012). There have been no reports in beavers.
- *Pasteurella* spp. 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* spp. 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).
- *Staphylococcus* spp. are gram-positive facultative bacteria commonly associated with suppurative infections and abscess formation but may also cause septicaemia and toxic shock syndrome (Speck, 2012c). Different *Staphylococcus* spp. 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.*). *Staphylococcus* spp. 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* spp. 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* spp. 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* spp. 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* spp. have not, to date, been identified in beavers but beavers may have potential to act as a reservoir.
- *Entamoeba* spp. 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.*, 2015). 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* spp. 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* spp. 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* spp. 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* spp. 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; Drozd *et al.*, 2004). It is assumed to be of low pathogenicity in otherwise healthy animals.

## ECTOPARASITES

- *Demodex* spp. 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* spp. are not normally pathogenic in immunocompetent hosts.
- *Ixodes* spp. 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* spp. 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* spp. 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).

## References

- ADHB. 2020. <https://pork.ahdb.org.uk/pig-production/>
- ADLER, B. (Ed.), (2015). *Leptospira and Leptospirosis*. Springer.
- ADLER, B. AND A. DE LA PEÑA MOCTEZUMA (2010). "Leptospira and leptospirosis." *Vet Microbiol* 140(3-4): 287-296.
- AGREN, E., VIKOREN T, MADSLIEN K, VAGE J (2019). Tularemia in hares and humans in Norway and Sweden in 2019. *European Wildlife Disease Association Newsletter Winter 2019*, p12.
- AHLEN, P. A. 2001. The parasitic and commensal fauna of the European beaver (*Castor fiber*) in Sweden. Honours thesis 2001. Swedish University of Agricultural Sciences, Uppsala, Sweden.
- AKERSTEDT, J. 2006. Encephalozoon cuniculi infection in carnivores with emphasis on serological methods. Honours thesis 2006. Norwegian school of Veterinary Science. Oslo, Norway.
- AKERSTEDT, J., A. LILLEHAUG, I-L. LARSEN, N. E. EIDE, J. M. ARNEMO AND K. HANDELAND (2010). "Serosurvey for Canine Distemper Virus, Canine Adenovirus, *Leptospira Interrogans* and *Toxoplasma Gondii* in free-ranging canids in Scandinavia and Svalbard." *J. Wild Dis* 46(2) 2010 474-480
- AKIMANA, C., AND KWAIK, Y. A. (2011). Francisella-arthropod vector interaction and its role in patho-adaptation to infect mammals. *Frontiers in Microbiology*, 2(FEB), 1–15.
- ALLEN, A. R., R. A. SKUCE AND A. W. BYRNE. 2018. Bovine Tuberculosis in Britain and Ireland – A Perfect Storm? the Confluence of Potential Ecological and Epidemiological Impediments to Controlling a Chronic Infectious Disease. *Frontiers in Vet Sci*. 5(109):1-17.
- APHA.2020.[http://apha.defra.gov.uk/External\\_OV\\_Instructions/TB\\_Instructions/Passive Surveillance/Other\\_Mycobacteria.html](http://apha.defra.gov.uk/External_OV_Instructions/TB_Instructions/Passive_Surveillance/Other_Mycobacteria.html). Visited 19 April 2020.
- ARAMINI, J. J., STEPHEN, C., DUBEY, J. P., ENGELSTOFT, C., SCHWANTJE, H., & RIBBLE, C. S. (1999). Potential contamination of drinking water with *Toxoplasma gondii* oocysts. *Epidemiology and Infection*, 122(2), 305–315.
- AVCIOGLU, H., E. GUVEN, I. BALKAYA AND R. KIRMAN. 2017. *Echinococcus multilocularis* in a Eurasian Lynx (*Lynx lynx*) in Turkey. *Parasitology*.
- AVIAT, F., B. BLANCHARD, V. MICHEL, B. BLANCHET, C. BRANGER, J. HARS, F. MANSOTTE, L. BRASME, C. DE CHAMPS, P. BOLUT, P. MONDOT, J. FALIU, S. ROCHEREAU, A. KODJO AND G. ANDRE-FONTAINE (2009). "*Leptospira* exposure in the human environment in France: A survey in feral rodents and in fresh water." *Comp Immunol Microbiol Infect Dis*. 32(6): 463-476.
- BACKHANS, A., M. JACOBSON, I. HANSSON, M. LEBBAD, S. THISTED-LAMBERTZ, E. GAMMELGARD, M. SAAGER, O. AKANDE AND C. FELLSTROM. 2013. *Epidemiol. Infect.* 141, 1885-1891.

- BAGRADE, G., G. DEKSNE, Z. OZOLINA, S. HOWLETT, M. INTERISANO, A. CASULLI AND E. POZIO. 2016. *Echinococcus multilocularis* in foxes and raccoon dogs: An increasing concern for Baltic countries. *Parasites and Vectors*. 9(1).
- BAJER, A., A. PAWELCZYK, J. M. BEHNKE, F. GILBERT AND E. SINSKI. 2001. Factors affecting the component community structure of haemoparasites in bank voles from the Mazury Lake District region of Poland. *Parasitology*. 2001. 122, 43-54.
- BAJER, A., M. BEDNARSKA, A. PAWELCZYK, J. M. BEHNKE, F. S. GILBERT AND E. SINSKI. 2002. Prevalence and abundance of *Cryptosporidium parvum* and *Giardia* spp. in wild rodents from the Mazury Lake district region of Poland. *Parasitology*. 125:21-34.
- BAJOMI, B. (2011). Reintroduction of the Eurasian beaver (*Castor fiber*) in Hungary.
- BAKASEJEVS, E., A. DAUKSTE, M. ZOLOVS AND A. ZDANOVSKA. 2012. Investigation of *Trichinella* in wildlife in Latgale region (Latvia). *Acat Biol. Univ. Dauugavp*. 12(1)1-5.
- BALKEMA-BUSCHMANN, A., BEER, M., BREITHAUPT, A., GRAAF, A., GROSCHUP, M., GRUND, C. H., HARDER, T., HOFFMANN, D., METTENLEITER, T H C RISSMANN, M., SCHLOTTAU, K., & SCHOEN, J. (2020). CORONAVIRUS DISEASE 2019 UPDATE (88): GERMANY, ANIMALS, RESEARCH, PIG, CHICKEN, BAT, FERRET. <https://promedmail.org/promed-post/?id=7196506>
- BAO, L., DENG, W., HUANG, B., GAO, H., LIU, J., REN, L., WEI, Q., YU, P., XU, Y., QI, F., QU, Y., LI, F., LV, Q., WANG, W., XUE, J., GONG, S., LIU, M., WANG, G., WANG, S., QIN, C. (2020). The Pathogenicity of SARS-CoV-2 in hACE2 Transgenic Mice. *BioRxiv*, 2020.02.07.939389.
- BARLOW, A., B. GOTTSTEIN AND n. MUELLER. 2011. *Echinococcus multilocularis* in an imported captive European beaver (*Castor fiber*) in Great Britain. *Veterinary Record*. 169(13).
- BARTHELMESS, E. L. (2014). Spatial distribution of road-kills and factors influencing road mortality for mammals in Northern New York State. *Biodiversity and Conservation*, 23(10), 2491–2514.
- BATBOLD, J., BATSAIKHAN, N., SHAR, S., HUTTERER, R., KRISTUFEK, B., YIGIT, N., MITSAIN, G., & PALOMO, L. (2017). *Castor fiber*. IUCN Red List of Threatened Species.
- BECK, R., L. VOJTA, S. CURKOVIC, V. MRLJAK, J. MARGALETIC AND B. HABRUN. 2011. Molecular survey of *Babesia microti* in wild rodents in Croatia. *Vector-borne and Zoonotic Diseases*. 11(1), 81-83.
- BECKER, S. D., M. BENNETT, J. P. STEWART AND J. L. HURST. 2006. Serological survey of virus infection among wild house mice (*Mus domesticus*) in the UK. *Laboratory Animals* 41 pp. 229-238.
- BENNETT, E., J. CLEMENT, P. SANSOM, I. HALL, S. LEACH, ANDJ. M. MEDLOCK. 2010. Environmental and ecological potential for enzootic cycles of Puumala hantavirus in Great Britain. *Epidemiol Infect* 138: 91-98.
- BENNETT, R. S., A. K. GRESKO AND B. R. MURPHY. 2011. Tahyna virus genetics, infectivity, and immunogenicity in mice and monkeys. *Virology* 8, 135.
- BERDAL, B. P., R. MEHL, N. K. MEIDELL, A. M. LORENTZEN-STYR AND O. SCHEEL. (1996). Field investigations of tularemia in Norway. *FEMS Immunology and Medical Microbiology*, 15, 1-10.

- Mehl, R., Meidell, N. K., Lorentzen-Styr, A. M., & Scheel, O. (1996). Field Investigations of Tularemia in Norway. *FEMS Immunology and Medical Microbiology*, 13(3), 191–195.
- BERDOY, M., WEBSTER, J. P., & MCDONALD, D. W. (2000). Fatal attraction in rats infected with *Toxoplasma gondii*. *Proceedings of the Royal Society B: Biological Sciences*, 267(1452), 1591–1594.
- BIRTLES, R. (2012). Leptospira Infections. In GAVIER-WIDEN D., J. P. DUFF AND A. MEREDITH (Eds.) *Infectious Diseases of Wild Mammals and Birds in Europe*, Blackwell Publishing Ltd.
- BIRTLES, R. 2012. Bartonella Infections. In GAVIER-WIDEN D., J. P. DUFF AND A. MEREDITH (Eds.) *Infectious Diseases of Wild Mammals and Birds in Europe*, Blackwell Publishing Ltd., UK.
- BIRTLES, R. 2012b. Rickettsial Infections. In GAVIER-WIDEN D., J. P. DUFF AND A. MEREDITH (Eds.) *Infectious Diseases of Wild Mammals and Birds in Europe*, Blackwell Publishing Ltd., UK.
- BLASDELL, K., C. MCCracken, A. MORRIS, A. A. NASH., M. BEGON, M. BENNETT AND J. P. STEWART. The wood mouse is a natural host for Murid herpesvirus. 4. *J. Gen. Virol.* 84: 111-113.
- BOBADILLA-SUAREZ, M., J. G. EWEN, J. J. GROOMBRIDGE, K. BECKMANN, J. SHOTTON, N. MASTERS, T. HOPKINS AND A. W. SAINSBURY. 2017. Using qualitative disease risk analysis for herpetofauna conservation translocations transgressing ecological and geographical barriers. *EcoHealth*, 14: S47-S70. DOI: 10.1007/s10393-015-1086-4
- BODEN, L. AND H. AUTY, 2015. Public health risk of Giardia and Cryptosporidium posed by reintroduction of beavers into Scotland. SNH.
- BORMAN, A. M., V. R. SIMPSON, M. D. PALMER, C. J. LINTON AND E. M. JOHNSON. 2009. Adiaspiromycosis due to *Emmonsia crescens* is widespread in native British mammals. *Mycopathologia*. 168:153-163.
- BORMAN, A. M., Y. JIANG, K. DUKIK, L. SIGLER, I. S. SCHWARTZ AND G. S. DE HOOG. 2018. Adiaspiromycosis and diseases caused by related fungi in Ajellomycetaceae. In SEYEDMOUSAVI, S. *et al.* (Eds.) *Emerging and Epizootic Fungal Infections in Animals*. Springer International Publishing.
- BOUFANA, B., M. STIDWORTHY, S. BELL, J. CHANTREY, N. MASTERS, S. UNWIN AND P. CRAIG. 2012. *Echinococcus* and *Taenia* spp. from captive mammals in the United Kingdom. *Veterinary Parasitology*. 190(1-2):95-103.
- BOWIE, W. R., KING, A. S., WERKER, D. H., ISAAC-RENTON, J. L., BELL, A., ENG, S. B., & MARION, S. A. (1997). Outbreak of toxoplasmosis associated with municipal drinking water. *Lancet*, 350(9072), 173–177.
- BRABB, T., NEWSOME, D., BURICH, A., & HANES, M. (2012). Infectious Diseases. In M. A. Suckow, K. A. Stevens, & R. P. Wilson (Eds.), *The Laboratory Rabbit, Guinea Pig, Hamster, and Other Rodents* (pp. 637–683). Elsevier Inc.

- BRAZIER, R. E., M. ELLIOTT, E. ANDISON, R. E. AUSTER, S. BRIDGEWATER, P. BURGESS, J. CHANT, H. GRAHAM, E. KNOTT, A. K. PUTTOCK, P. SANSUM AND A. VOWLES. 2020. River Otter beaver trial: Science and evidence report.
- BRITTON, A. AND A. BARLOW. 2019. Beaver Enclosure Report. Visit to Upcott Grange. Natural England. 26/04/2019.
- BROMAN, T., THELAUS, J., ANDERSSON, A. C., BÄCKMAN, S., WIKSTRÖM, P., LARSSON, E., GRANBERG, M., KARLSSON, L., BÄCK, E., ELIASSON, H., MATTSSON, R., SJÖSTEDT, A., & FORSMAN, M. (2011). Molecular detection of persistent *Francisella tularensis* subspecies holarctica in natural waters. International Journal of Microbiology, 2011.
- BRONSTEIN, A. M. AND A. N. LUKASHEV. 2019. case of trichinellosis associated with beaver (*Castor fiber*) meat. Journal of Helminthology. 0, 1, 3.
- BROOK, E. J., C. A. HART, N. P. FRENCH AND R. M. CHRISTLEY. 2009. Molecular epidemiology of *Cryptosporidium* subtypes in cattle in England. Veterinary Journal. 179(2009):378-382.
- BROWNLOW, A (2011). Post-Mortem Report of a Beaver M609250 (M08K30). Scottish Agricultural College Veterinary Services, Scotland.
- BRYANT, A. A., SCHWANTJE, H. M., & DE WITH, N. I. (2002). Disease and unsuccessful reintroduction of Vancouver Island marmots (*Marmota vancouverensis*). In K. B. Armitage & V. U. Rumianstev (Eds.), Holarctic Marmots as a Factor of Biodiversity (pp. 101–107). ABF Publishing House (Moscow).
- BURTHER, S., M. BENNETT AND A. KIPAR. 2008. Tuberculosis (*Mycobacterium microti*) in wild field vole populations. Parasitology, 135: 309-317.
- CALLE, P. (2020). CORONAVIRUS DISEASE 2019 UPDATE (84): USA, TIGERS. <https://promedmail.org/promed-post/?id=20200406.7191352>
- CAMPBELL, R. D., ROSELL, F., NOLET, B. A., & DIJKSTRA, V. A. A. (2005). Territory and group sizes in Eurasian beavers (*Castor fiber*): Echoes of settlement and reproduction? Behavioral Ecology and Sociobiology, 58(6), 597–607. <https://doi.org/10.1007/s00265-005-0942-6>
- CAMPBELL-PALMER, R AND GIRLING, S. 2019. Final beaver trapping and health screening report. River Otter Beaver Trial.
- CAMPBELL-PALMER, R. AND F. ROSELL. 2012. Captive care and welfare considerations for beavers. Zoo Biology. 34:101-109.
- CAMPBELL-PALMER, R. Annual Beaver Trapping 2018. ROBT.
- CAMPBELL-PALMER, R., & ROSELL, F. (2010). Conservation of the Eurasian beaver *Castor fiber*: An olfactory perspective. Mammal Review, 40(4), 293–312.
- CAMPBELL-PALMER, R., & ROSELL, F. (2013). Captive Management Guidelines for Eurasian Beavers (*Castor fiber*). The Royal Zoological Society of Scotland, Edinburgh Zoo.

- CAMPBELL-PALMER, R., A. PUTTOCK, H. GRAHAM, K. WILSON, G. SCHWAB, M. J. GAYWOOD AND R. E. BRAZIER. 2018. Survey of the Tayside area beaver population 2017-2018. SNH Commissioned Report No. 1013.
- CAMPBELL-PALMER, R., G. SCHWAB, S. GIRLING, S. LISLE AND D. GOW. 2015. Managing wild Eurasian beavers: A review of European management practices with consideration for Scottish application. SNH Commissioned Report No 812.
- CAMPBELL-PALMER, R., J. DEL POZO, B. GOTTSTEIN, S. GIRLING, J. CRACKNELL, G. SCHWAB, F. ROSELL AND R. PIZZI. 2015c. *Echinococcus multilocularis* detection in live Eurasian beavers using a combination of laparoscopy and abdominal ultrasound under field conditions. PLoS One. 2015. 10(7)
- CAMPBELL-PALMER, R., R. PIZZI, H. DICKINSON AND S. GIRLING. 2015b. Trapping and health screening of free-living beavers within the Tayside catchment, east Scotland. SNH report 681.
- CAMPBELL-PALMER, R., ROSELL, F., NAYLOR, A., MOTA, S., COLE, G., FRAZER, M., PIZZI, R., ELLIOT, M., WILSON, K., GAYWOOD, M., & GIRLING, S. (Submitted). Eurasian beaver (*Castor fiber*) health surveillance in Britain: assessing a disjunctive reintroduced population. Vet Record.
- CAMPBELL-PALMER, R., S. GIRLING, R. PIZZI, I.S. HAMNES AND J. DEL-POZO. 2013. *Stichorchis subtriquetrus* in a free-living beaver in Scotland. British Veterinary Association. *Veterinary Record* vol. 173 (2013)
- CAVANAGH, R., M. BEGON, M. BENNETT, T. ERGON, I. M. GRAHAM, P. E. W. DE HAAS, C. A. HART, M. KOEDAM, K. KREMER, X. LAMBIN, P. ROHOLL AND D. VAN SOOLINGEN. 2002. *Mycobacterium microti* infection (vole tuberculosis) in wild rodent populations. J. Clin. Microbiol. 40(9): 3281-3285.
- CDC (CENTERS FOR DISEASE CONTROL AND PREVENTION). <https://www.cdc.gov/rodents/diseases/direct.html>. Website visited 20/03/2020.
- CHAIGNAT, V., P. BOUJON, C. FREY, B. HENTRICH, N. MULLER AND B. GOTTSTEIN. 2015. The brown hare (*Lepus europaeus*) as a novel intermediate host for *Echinococcus multilocularis*. Parasitol Res. 114(8):3167-9.
- CHALMERS, R. M., G. ROBINSON, K. ELWIN AND R. ELSON. 2019. Analysis of the *Cryptosporidium* spp. and gp60 subtypes linked to human outbreaks of cryptosporidiosis in England and Wales, 2009 to 2017. Parasites & Vectors. 12(95):1-13.
- CHAMBERS, M. A. 2009. Review of the diagnosis and study of tuberculosis in non-bovine wildlife species using immunological methods. Transboundary and Emerg Dis. 56(2009): 215-227.
- CHAN, A. J. F., A. J. ZHANG, S. YUAN, AND V. KWOK. 2020. Simulation of the clinical and pathological manifestations of Coronavirus Disease 2019 (COVID-19) in golden Syrian hamster model: implications for disease pathogenesis and transmissibility Authors: Jasper Fuk-Woo Chan. Clinical Infectious Diseases, ciaa325.

- CHANTREY, J. C., A. M. BORMAN, E. M. JOHNSON AND A. KIPAR. 2006. *Emmonsia crescens* infection in a British water vole (*Arvicola terrestris*). *Medical Mycology*. 44: 375-378.
- CHAPMAN, H. D., BARTA, J. R., BLAKE, D., GRUBER, A., JENKINS, M., SMITH, N. C., SUO, X., & TOMLEY, F. M. (2013). A Selective Review of Advances in Coccidiosis Research. In *Advances in Parasitology* (1st ed., Vol. 83, Issue November 2017). Elsevier Ltd.
- CHASTAGNER, A., M. MOINET, G. PEREZ, E. ROY, K. D. MCCOY, O. PLANTARD, A. AGOULON, S. BASTIAN, A. BUTET, Y. RANTIER, H. VERHEYDEN, N. CEBE, A. LEBLOND AND G. VOURCH. 2016. Prevalence of *Anaplasma phagocytophilum* in small rodents in France. *Ticks and Tick-borne diseases*. 7(2016) 988-991.
- CHENG, V. C. C., LAU, S. K. P., WOO, P. C. Y., & KWOK, Y. Y. (2007). Severe acute respiratory syndrome coronavirus as an agent of emerging and re-emerging infection. *Clinical Microbiology Reviews*, 20(4), 660–694.
- CHIARI, M., N. FERRARI, D. GIARDELLO, P. LANFRANCHI, M. ZANONI, A. LAVAZZA AND L. G. ALBORALI. 2014. Isolation and identification of *Salmonella* spp. from red foxes (*Vulpes vulpes*) and badgers (*Meles meles*) in northern Italy. *Acta Veterinaria Scandinavica*. 56:86.
- CHMURZYNSKA, E., M. ROZYCKI, E. BILSKA-ZAJAC, K. NOCKLER, A. MAYER-SCHOLL, E. POZIO, T. CENCEK AND J. KARAMON. 2013. *Trichinella nativa* in red foxes (*Vulpes vulpes*) of Germany and Poland: Possible different origins. *Veterinary Parasitology*. 198(2013):254-257.
- ĆIROVIC, D., I. PAVLOVIC, V. IVETIC, M. MILENKOVIC, I. RADOVIC AND B. SAVIC. 2009. Reintroduction of the European beaver (*Castor fiber* L.) into Serbia and return of its parasite: the case of *Stichorchis subtriquetrus*. *Institut za Bioloska Istrazivanja. Arch Biol Sci* 61:141–145.
- ĆIROVIC, D., I. PAVLOVIC, Z. KULISIC, V. IVETIC, A. PENEZIC AND N. COSIC. 2012. *Echinococcus multilocularis* in the European beaver (*Castor fiber*) from Serbia: First report. *Veterinary Record*. 171(4):100.
- CITTERIO, B. And F. BIAVASCO. 2015. *Aeromonas hydrophila* virulence. *Virulence*. 6(5):417-418.
- CLEMENT, J., P. MCKENNA, V. VERGOTE, AND M. VAN RANST. 2014. Comment on Jameson *et al.*: Prevalence of antibodies against hantaviruses in serum and saliva of adults living or working on farms in Yorkshire, United Kingdom. *Viruses* 6: 3415-3424.
- COLLINS, R. 2009. Post-mortem report of a Beaver 21-M0035-05-09 (M08K31). *Veterinary Laboratories Agency, Starcross*.
- COUSO-PEREZ, S., E. ARES-MAZAS AND H. GOMEZ-COUSO. 2019. Novel *Cryptosporidium* genotypes in wild and cultured trout. *VIIth International Giardia and Cryptosporidium Conference*. Rouen, France.
- COVID-19 NATIONAL EMERGENCY RESPONSE CENTER. (2020). Coronavirus disease-19: The First 7,755 Cases in the Republic of Korea. *Osong Public Health and Research Perspectives*.
- COX, F. E. G. 1987. Protozoan parasites of British small rodents. *Mammal Review*. 17 (2/3) 59-66.

- CRANWELL, M (2009a). Post-mortem Report of a Beaver 21-M0161-02-09 (M08K20). Veterinary Laboratories Agency, Starcross.
- CRANWELL, M (2009b). Post-mortem Report of a beaver 21-M0414-04-09 (M08K34). Veterinary Laboratories Agency, Starcross
- CRANWELL, M. (2009c). Post-mortem report of beaver M08K33. VLA Ref. 21-M0090-04-09. Veterinary Laboratories Agency, Starcross
- CULL, B., A. G. C. VAUX, L. J. OTTOWELL, E. L. GILLINGHAM AND J. M. MEDLOCK. 2017. Tick infestation of small mammals in an English woodland. *J. Vector Ecology*. 42(1):74-83.
- CULLEN, C. L. 2003. Normal ocular features, conjunctival microflora and intraocular pressure in the Canadian beaver (*Castor canadensis*). *Veterinary Ophthalmology*. 6(4): 279-284.
- CUNNINGHAM, A. A. 1996. Disease risks of wildlife translocations. *Conservation Biology*, 10: 349-353.
- DANESI, P., C. FALCARO, K. DUKIK, Y. JIANG, A. P. RIZZOLI, R. ALLAVENA, V. SIMPSON, S. RAVAGNAN, C. ZANARDELLO, G. CAPELLI AND G. S. DE HOOG. 2020. Molecular diagnosis of Emmonsia-like fungi occurring in wild animals. *Mycopathologia*. 185:51-65.
- DAVIDSON, R. AND L. ROBERTSON. 2012. European pet travel: Misleading information from veterinarians and government agencies. *Zoonoses and Public Health*. 59(8):575-583.
- DAVIDSON, R. K., B. GJERDE, T. VIKOREN, A. LILLEHAUG AND K. HANDELAND. 2006. Prevalence of *Trichinella* larvae and extra-intestinal nematodes in Norwegian red foxes (*Vulpes vulpes*). *Vet Parasitol*. 136(3-4):307-16.
- DAVIDSON, R. K., I. ORPETVEIT, L. MOLLER AND C. M. O. KAPEL. 2009. Serological detection of anti-*Trichinella* antibodies in wild foxes and experimentally infected farmed foxes in Norway. *Veterinary Parasitology*. 163(2009):93-100.
- DAVIDSON, R. K., K. HANDELAND AND C. M. O. KAPEL. 2008. High tolerance to repeated cycles of freezing and thawing in different *Trichinella nativa* isolates. *Parasitol. Res*. 103:1005-1010.
- DAVIDSON, R., A. LAVIKAINEN, S. KONAYAEV, J. SCHURER, A. MILLER, A. OKSANEN AND E. JENKINS. 2016. Echinococcus across the north: Current knowledge, future challenges. *Food and Waterborne Parasitology*. 4:39-53. Elsevier.
- DAVIDSON, R., O. OINES, C. ALBIN-AMIOT, P. HOPP, K. MADDEN, A. HAGSTROM AND M. ISAKSSON. 2013. Ghost hunting. Is *Echinococcus multilocularis* really absent from mainland Norway? *Trop Med Int Health*. 18:97.
- DAVIDSON, R., T. ROMIG, E. JENKINS, M. TRYLAND AND L. ROBERTSON. 2012. The impact of globalisation on the distribution of *Echinococcus multilocularis*. *Trends in Parasitology*. 28(6):239-247.
- DAVIDSON, W. R. AND V. F. NETTLES. 1992. Relocation of wildlife: identifying and evaluating disease risks. *Transactions of the North American Wildlife and Natural Resources Conference*, 466-473.



- DAVIES, R. B. AND C. P. HIBLER. 1979. Animal reservoirs and cross-species transmission of *Giardia*. In JAKUBOWSKI, W. AND J. C. HOFF (Eds.). Waterborne Transmission of Giardiasis. Proceedings of Symposium, U. S. Environmental Protection Agency, Cincinnati, Ohio.
- DE GROOT, R., BAKER, S., BARIC, R., ENJUANES, L., GORBALENYA, A., HOLMES, K., PERLMAN, S., POON, L., ROTTIER, P., TALBOT, P., WOO, P., & ZIEBUHR, J. (2012). Family Coronaviridae. In Virus taxonomy: ninth report of the International Committee on Taxonomy of Viruses (pp. 806–828).
- DEFRA. 2014. Importation of European beaver from Europe. Voluntary code of practice. [www.gov.uk/government/publications](http://www.gov.uk/government/publications).
- DEFRA. 2020. Bringing your pet dog, cat or ferret to the UK. <https://www.gov.uk/take-pet-abroad/tapeworm-treatment-dogs>. Site visited 16/03/2020.
- DEMIASZKIEWICZ, A. W., J. LACHOWICZ, I. KULIGOWSKA, A. M. PYZIEL, G. BELZECKI, R. MILTKO, B. KOWALIK, W. GOGOLA AND Z. GIZEJEWSKI. 2014. Endoparasites of the European beaver (*Castor fiber* L. 1758) in north-eastern Poland. National Veterinary Research Institute. *Bull Vet Inst Pulawy* 58:223–227.
- DEMKOWSKA-KUTRZEPA, M., M. STUDZINSKA, K. TOMCZUK AND P. ROZANSKI. 2016. *Stichorchis subtriquetrus* as a common parasite of beavers indigenous to the Lublin region.
- DENG, W., BAO, L., GAO, H., XIANG, Z., QU, Y., SONG, Z., GONG, S., LIU, J., LIU, J., YU, P., QI, F., XU, Y., LI, F., XIAO, X., LV, Q., XUE, J., WEI, J., LIU, M., WANG, G., QIN, C. (2020). Rhesus macaques can be effectively infected with SARS-CoV-2 via ocular conjunctival route. February 2019, 1–13.
- DEPLAZES, P., L. RINALDI, C. ALVAREZ-ROJAS, P. TORGERSON, M. HARANDI, T. ROMIG AND E. JENKINS. 2017. Global distribution of alveolar and cystic echinococcosis. *Advances in Parasitology*. 95:315-493.
- DETER, J., J. BRYJA, Y. CHAVAL, M. GALAN, H. HENTTONEN, J. LAAKONEN, L. VOUTILAINEN, O. VAPALAHTI, A. VAHERI, A. R. SALVADOR, S. MORAND, J-F. COSSON AND N. CHARBONNEL. 2008. Association between the DQA MHC class II gene and Puumala virus infection in *Myodes glareolus*, the bank vole. *Inf. Genetics and Evol.* 8 (2008) 450-458.
- DEUCHANDE, R. 2009. Post-mortem report on beaver M08K22. SAC Veterinary Services. Ref: M094843.
- DHABHAR, F. S., & MCEWEN, B. S. (1997). Acute stress enhances while chronic stress suppresses cell-mediated immunity in vivo: A potential role for leukocyte trafficking. *Brain, Behavior, and Immunity*, 11(4), 286–306.
- DICKENS, M. J., DELEHANTY, D. J., & ROMERO, L. M. (2009). Stress and translocation: Alterations in the stress physiology of translocated birds. *Proceedings of the Royal Society B: Biological Sciences*, 276(1664), 2051–2056.
- DICKENS, M., D. DELEHANTY AND L. ROMERO. 2010. Stress: an inevitable component of animal translocation. *Biological Conservation*.143 (6): 1329-1341.

- DOLKA, I., A. GIZEJEWSKA, Z. GIZEJWSKI, J. KOLODZIEJSKA-LESISZ AND W. KLUCINSKI. 2017. Pulmonary adiaspiromycosis in the Eurasian beaver (*Castor fiber*) inhabiting Poland. Polish Journal of Veterinary Sciences. 20(3):615-617.
- DORONINA, L., MATZKE, A., CHURAKOV, G., STOLL, M., HUGE, A., & SCHMITZ, J. (2017). The Beaver's Phylogenetic Lineage Illuminated by Retroposon Reads. Scientific Reports, 7, 1–8.
- DREXLER, J. F., CORMAN, V. M., & DROSTEN, C. (2014). Ecology, evolution and classification of bat coronaviruses in the aftermath of SARS. Antiviral Research, 101, 45–56.
- DROZDZ, J., A. W. DEMIASZKIEWICZ AND J. LACHOWICZ. 2004. Endoparasites of the beaver *Castor fiber* in northeast Poland.
- DUBEY, J. P. (1998). *Toxoplasma gondii* Oocyst Survival under Defined Temperatures. Journal of Parasitology, 84(4), 862–865.
- DUFF, A. G., R. CAMPBELL-PALMER AND R. NEEDHAM. 2013. The beaver beetle *Platypsyllus castoris* Ritsema (Leiodidae: Platypsyllinae) apparently established on reintroduced beavers in Scotland, new to Britain. The Coleopterist. 22(1): 9-19.
- DUGGAN, J. M., R. CLOSE, L. MCCANN, D. WRIGHT, M. KEYS, N. MCCARTHY, T. MANNES, A. WALSH, A. CHARLETT, AND T. J. G. BROOKS. 2017. A seroprevalence study to determine the frequency of hantavirus infection in people exposed to wild and pet fancy rats in England. Epidemiol Infect 145: 2458-2465.
- DUH, D., E. VARLIJEN-BUZAN, S. HASIC AND R. CHARREL. 2014. Increased seroprevalence of lymphocytic choriomeningitis virus infection in mice sampled in illegal waste sites. Parasites & Vectors. 7(Suppl 1): 031.
- ECDPC (EUROPEAN CENTRE FOR DISEASE PREVENTION AND CONTROL). 2019. Cryptosporidiosis. In ECDPC. Annual Epidemiological Report for 2017. Stockholm. ECDPC. 2019.
- EFSA (EUROPEAN FOOD SAFETY AUTHORITY) AND ZANCANARO, G. 2019. Scientific report on the annual assessment of *Echinococcus multilocularis* surveillance reports submitted in 2019 in the context of Commission Delegated Regulation (EU) 2018/772. EFSA Journal. 17(11):5906.
- EFSA. 2019. The European Union One Health 2018 Zoonoses Report. European Food Safety Authority and European Centre for Disease Prevention and Control. EFSA Journal. 17(12):5926.
- EFSA. 2020. <https://efsa.onlinelibrary.wiley.com/doi/10.2903/j.efsa.2019.5926>. Site visited 15 April 2020.
- ELLIS, J., OYSTON, P. C. F., GREEN, M., & TITBALL, R. (2002). Tularemia. Clinical Microbiology Reviews, 15(4), 631–646.
- ELLIS, P. A., AND A. E. WRIGHT. (1961). Coccidiosis in guinea-pigs. Journal of Clinical Pathology, 14, 394–396.
- EMMONS, C. W. 1950. Histoplasmosis: Animal reservoirs and other sources in nature of the pathogenic fungus, histoplasma. American Journal of Public Health. April. pp. 436-440.

- ENVIRONMENT AGENCY. (2019). Annual Fisheries Report 2017 to 2018 April 2019.
- ENVIRONMENTAL AGENCY. (2020). Fisheries annual report 2018 to 2019.
- ESTÈVE, R. (1988). An analysis of Beaver mortality in Haute-Savoie. *Bièvre*, 9, 171–176.
- ETHELBERG, S., K. E. P. OLSEN, P. GERNER-SMIDT AND K. MOLBAK. 2007. The significance of the number of submitted samples and patient-related factors for faecal bacterial diagnostics. *Clinical Microbiology and Infection*. 13(11):1095-1099.
- EUDEN, P. R. 1990. Salmonella isolates from wild animals in Cornwall. *British Veterinary Journal*. 146(3):228-232
- FSA (Food Standards Agency). 2020. <https://www.food.gov.uk/safety-hygiene/e-coli>. Site visited 27/05/2020.
- EUROPEAN CENTRE FOR DISEASE PREVENTION AND CONTROL. 2014. Prevention measures and communications strategies for hantavirus infection in Europe.
- EUROPEAN CENTRE FOR DISEASE PREVENTION AND CONTROL (ECDC), 2019. Hantavirus infection. Annual Epidemiological report for 2017.
- EUROPEAN FOOD SAFETY AUTHORITY AND EUROPEAN CENTRE FOR DISEASE PREVENTION AND CONTROL. (2018). The European Union summary report on trends and sources of zoonoses, zoonotic agents and food-borne outbreaks in 2017. In *EFSA Journal* (Vol. 16, Issue 12, pp. 176–180).
- EVANGELISTA, K. V. AND J. COBURN (2010). "*Leptospira* as an emerging pathogen: a review of its biology, pathogenesis and host immune responses." *Future Microbiol* 5(9): 1413-1425.
- FANG, L. Z., L. ZHAO, H. L. WEN, Z. T. ZHANG, J. W. LIU, S. T. HE, Z. F. XUE, D. Q. MA, X. S. ZHANG, Y. ZHANG, AND X. J. YU. 2015. Reservoir host expansion of hantavirus, China. *Emerg Infect Dis* 21: 170-171.
- FAYER, R., M. SANTIN, J. M. TROUT, S. DESTEFANO, K. KOENEN AND T. KAUR. 2006. Prevalence of *microsporidia*, *Cryptosporidium* spp. and *Giardia* spp. in beavers *Castor canadensis* in Massachusetts. *J Zoo and Wildlife Med*. 37(4):492-497.
- FENG, Y. AND L. XIAO. 2011. Zoonotic potential and molecular epidemiology of *Giardia* species and Giardiasis. *Clin Microbiol Reviews*. 24(1):110-140.
- FENG, Y., U. M. RYAN AND L. XIAO. 2018. Genetic diversity and population structure of *Cryptosporidium*. *Trends in Parasitology*. 34(11):997-1011.
- FERROGLIO, E. Listeria infections. In G. Gavier-Widen, P.D. Duff & A. Meredith (Eds), *Infectious Diseases of Wild Mammals and Birds in Europe* (First edition pp. 345-362). Blackwell Publishing Ltd., UK.
- FERROGLIO, E. Pasteurella infections. 2012b. In G. Gavier-Widen, P.D. Duff & A. Meredith (Eds), *Infectious Diseases of Wild Mammals and Birds in Europe* (First edition pp. 345-362). Blackwell Publishing Ltd., UK.

- FEVOLA, C. 2019. Distribution and clinical associations of Ljungan virus (Parechovirus B). PhD thesis. University of Helsinki.
- FEVOLA, C., C. ROSSI, F. ROSSO, M. GIRARDI, R. ROSA, M. MANICA AND H. C. HAUFFE. 2020. Geographical distribution of Ljungan Virus in small mammals in Europe. *Vector-borne and Zoonotic Diseases*. In Press.
- FICHET-CALVET, E., E. B. KIA, P. GIRAUDOUX, J. P. QUERE, P. DELATRÉE AND R. W. ASHFORD. 2004. *Frenkelia* parasites in a small mammal community. Dynamics of infection and effect on the host. *Parasite*. 11(3) 301-310.
- FLEGR, J., HAVLÍČEK, J., KODYM, P., MALÝ, M., & SMAHEL, Z. (2002). Increased risk of traffic accidents in subjects with latent toxoplasmosis: A retrospective case-control study. *BMC Infectious Diseases*, 2, 1–6.
- FORBES, K. M., L. VOUTILAINEN, A. JÄÄSKELÄINEN, T. SIRONEN, P. M. KINNUNEN, P. STUART, O. VAPALAHTI, H. HENTTONEN, ANDO. HUITU. 2014. Serological survey of rodent-borne viruses in Finnish field voles. *Vector Borne Zoonotic Dis* 14: 278-283.
- FORBES, K. M., T. SIRONEN, ANDA. PLYUSNIN. 2018. Hantavirus maintenance and transmission in reservoir host populations. *Curr Opin Virol* 28: 1-6.
- FORMISANO, P., B. ALDRIDGE, Y. ALONY, L. BEEKHUIS, E. DAVIES AND J. DEL POZO. 2013. Identification of *Sarcocystis capracanis* in cerebrospinal fluid from sheep with neurological disease. *Veterinary Parasitology*, 193(1-3): 252-255.
- FORZÁN, M. J., & FRASCA, S. (2004). Systemic Toxoplasmosis in a Five-Month-Old Beaver, (*Castor Canadensis*). *Journal of Zoo and Wildlife Medicine*, 35(1), 113–115. <https://doi.org/10.1638/03-031>
- FREDERICK, J., & STEWART, J. (1975). Chronic shedding tularemia nephritis in rodents, possible relation to occurrence of *Francisella tularensis* in lotic waters. *Journal of Wildlife Diseases*, 11(July), 421–430.
- FRENKEL, J. K., RUIZ, A., & CHINCHILLA, M. (1975). Soil survival of *Toxoplasma* oocysts in Kansas and Costa Rica. *The American Journal of Tropical Medicine and Hygiene*, 24(3), 439–443.
- FRIEND, M. (2006). Tularemia: Reston, Va., U.S. Geological Survey, Circular 1297, 68 p.
- FUEHRER, H. 2014. An overview of the host spectrum and distribution of *Calodium hepaticum*: part 2 – Mammalia. *Parasitol. Res.* 113:641-651.
- GAFFURI, A. 2012. *Salmonella* infections in wild mammals. In Gavier-Widen, G., Duff, P.D. & Meredith, A. (Eds), *Infectious Diseases of Wild Mammals and Birds in Europe* (First edition pp. 392-397). Blackwell Publishing Ltd., UK.
- GALVÁN-RAMÍREZ, M. D. L. L., SÁNCHEZ-OROZCO, L. V., RODRÍGUEZ, L. R., RODRÍGUEZ, S., ROIG-MELO, E., TROYO SANROMÁN, R., CHIQUETE, E., & ARMENDÁRIZ-BORUNDA, J. (2013). Seroepidemiology of *Toxoplasma gondii* infection in drivers involved in road traffic accidents in the metropolitan area of Guadalajara, Jalisco, Mexico. *Parasites and Vectors*, 6(1), 1–9.

- GAO, G. F., W. R. JIANG, M. H. HUSSAIN, K. VENUGOPAL, T. S. GRITSUN, H. W. REID AND E. A. GOULD. 1993. Sequencing and antigenic studies of a Norwegian virus isolated from encephalomyelitic sheep confirm the existence of Louping Ill Virus outside Great Britain and Ireland. *J Gen Virol.* 74(1):109-114.
- GASPER, P. W. AND R. P. WATSON in Williams, E. S. & Barker, I. K. (Eds.). 2001. Plague and Yersiniosis. *Infectious Diseases of Wild Mammals* (3<sup>rd</sup> edition) pp. 313-329. Iowa State University Press.
- GAVIER-WIDEN, D., M. CHAMBERS, C. GORTAZAR, R. DELAHAY, R. CROMIE AND A. LINDEN. 2012. Mycobacteria Infections. In G. Gavier-Widen, P.D. Duff & A. Meredith (Eds), *Infectious Diseases of Wild Mammals and Birds in Europe* (First edition pp. 345-362). Blackwell Publishing Ltd., UK.
- GAVIER-WIDEN, D., M. M. COOKE, J. GALLAGHER, M. A. CHAMBERS AND C. GORTAZAR. 2009. A review of infection of wildlife hosts with *Mycobacterium bovis* and the diagnostic difficulties of the 'no visible lesion' presentation. *New Zealand Veterinary Journal.* 57(3), 2009.
- GAYWOOD, M., D. BATTY AND C. GALBRAITH. 2008. Reintroducing the European beaver in Great Britain. *British Wildlife.* Aug 2008.
- GAYWOOD, M., STRINGER, A., BLAKE, D., HALL, J., HENNESSY, M., TREE, A., GENNEY, D., MACDONAL, L., TONHASCA, A., BEAN, C., MCKINNELL, J., COHEN, S., RAYNOR, R., WATKINSON, P., BALE, D., TAYLOR, K., SCOTT, J., & BLYTH, S. (2015). Beavers in Scotland - A report to the Scottish Government. In *Scottish Natural Heritage.*
- GELLING, M., D. MACDONALD, S. TELFER, T. JONES, K. BOWN, R. BIRTLES AND F. MATHEWS. 2012. Parasites and pathogens of wild populations of water voles in the UK. *Eur. J. Wildlife. Res.* 58(3) 615-619.
- GELLING, M., W. ZOCHOWSKI, D. W. MACDONALD, A. JOHNSON, M. PALMER AND F. MATHEWS (2015). "Leptospirosis acquisition following the reintroduction of wildlife." *Vet Rec* 177(17): 440.
- GIOVANNINI S., M-P. RYSER-DEGIORGIS, S. TAGLIABUE, M. PEWSNER AND F. C. ORIGGI (2012). "Leptospirosis in European Beavers (*Castor fiber*) from Switzerland."
- GIRLING, S. J., G. GOODMAN, P. BURR, R. PIZZI, A. NAYLOR, G. COLE, D. BROWN, M. FRASER, F. N. ROSELL, G. SCHWAB, M. ELLIOTT AND R. CAMPBELL-PALMER. 2019c. "Evidence of *Leptospira* species and their significance during reintroduction of Eurasian beaver (*Castor fiber*) to Great Britain." *Vet Rec* 185 (15): 482.
- GIRLING, S. J., L. M. MCELHINNEY, M. A. FRASER, D. GOW, R. PIZZI, A. NAYLOR, G. COLE, D. BROWN, F. ROSELL, G. SCHWAB, ANDR. CAMPBELL-PALMER. 2019. Absence of hantavirus in water voles and Eurasian beavers in Britain. *Vet Rec* 184: 253.
- GIRLING, S., A. NAYLOR, A., M. FRASER, R. CAMPBELL-PALMER. 2019b. Reintroducing beavers *Castor fiber* to Britain: a disease risk analysis *Mammal Review* doi: 10.1111/mam.12163

- GIZEJEWESKA, A., A. SPODNIIEWSKA, D. BARSKI AND J. FATTEBERT. 2015. Beavers indicate metal pollution away from industrial centers in northern Poland. *Environ. Sci. Pollut. Res.* 22:3969-3975.
- GLASER, R., & KIECOLT-GLASER, J. K. (2005). Stress- induced immune dysfunction: implications for health. *Nature Reviews. Immunology*, 5 (March), 243–251. <https://doi.org/10.3390/nu5041241>.
- GOHARDEHI, S., SHARIF, M., SARVI, S., MOOSAZADEH, M., ALIZADEH-NAVAEI, R., HOSSEINI, S. A., AMOUEI, A., PAGHEH, A., SADEGHI, M., & DARYANI, A. (2018). The potential risk of toxoplasmosis for traffic accidents: A systematic review and meta-analysis. *Experimental Parasitology*, 191(March), 19–24.
- GOLDEN, J. W., C. D. HAMMERBECK, E. M. MUCKER AND R. L. BROCATO. 2015. Animal models for the study of rodent-borne haemorrhagic fever viruses: Arenaviruses and Hantaviruses. *Biomed. Res. Int.* 2015, July 21.
- GOODMAN, G. 2014. The Scottish beaver trial: Veterinary Monitoring of the Knapdale beaver populations 2009-2014.
- GOODMAN, G., A. MEREDITH, S. GIRLING, F. ROSELL AND R. CAMPBELL-PALMER. 2017. Outcomes of a ‘One Health’ monitoring approach to a five-year beaver (*Castor fiber*) reintroduction trial to Scotland. *EcoHealth*. 14: 139-143.
- GOODMAN, G., S. GIRLING, R. PIZZI, A. MEREDITH, F. ROSELL AND R. CAMPBELL-PALMER. 2012. Establishment of a health surveillance programme for reintroduction of the Eurasian beaver (*Castor fiber*) into Scotland. *J. Wild. Dis.* 48(4):971-978.
- GORBALENYA, A. E., BAKER, S. C., BARIC, R. S., DE GROOT, R. J., DROSTEN, C., GULYAEVA, A. A., HAAGMANS, B. L., LAUBER, C., LEONTOVICH, A. M., NEUMAN, B. W., PENZAR, D., PERLMAN, S., POON, L. L. M., SAMBORSKIY, D. V., SIDOROV, I. A., SOLA, I., & ZIEBUHR, J. (2020). The species Severe acute respiratory syndrome-related coronavirus: classifying 2019-nCoV and naming it SARS-CoV-2. *Nature Microbiology*, 5(March).
- GORTAZAR, C., M. J. TORRES, P. ACEVEDO, J. AZNAR, J. DE LA FUENTE AND J. VICENTE. 2011. Fine-tuning the space, time, and host distribution of mycobacteria in wildlife. *BMC Microbiology*. 11:27.
- GOTTSTEIN, B., A. LACHENMAYER, G. BELDI, J. WANG, B. MERKLE, X. VU AND N. MULLER. 2019. Diagnostic and follow-up performance of serological tests for different forms/courses of alveolar echinococcosis. *Food and Waterborne Parasitology*. 16.
- GOTTSTEIN, B., C. FREY, R. CAMPBELL-PALMER, R. PIZZI, A. BARLOW, B. HENTRICH AND M. RYSER-DEGIORGIS. 2014. Immunoblotting for the serodiagnosis of alveolar echinococcosis in alive and dead Eurasian beavers (*Castor fiber*). *Veterinary Parasitology*. 205(1-2):113-118.
- GOTTSTEIN, B., E. POZIO AND K. NOCKLER. 2009. Epidemiology, diagnosis, treatment and control of Trichinellosis. *Clinical Microbiology Reviews*. 22(1):127-145.

- GOUMENOU, M., SPANDIDOS, D., & TSATSAKIS, A. (2020). Possibility of transmission through dogs being a contributing factor to the extreme Covid-19 outbreak in North Italy. *Molecular Medicine Reports*, 2–4.
- GOV.UK, 2019. <https://www.gov.uk/guidance/hantaviruses>. Visited 30/01/2019
- GRANGE, J. M. 1990. The avian tubercle bacillus and its relatives. *J. App. Bact.* 68:411-431.
- GRILO, C., ASCENSÃO, F., SANTOS-REIS, M., & BISSONETTE, J. A. (2011). Do well-connected landscapes promote road-related mortality? *European Journal of Wildlife Research*, 57(4), 707–716.
- GRUBEŠIĆ, M., MARGELETIĆ, J., ČIROVIĆ, D., VUCELJA, M., BJEDOV, L., BURAZEROVIĆ, J., & TOMLIJANOVIĆ, K. (2015). Analysis of Beaver (*Castor fiber* L.) Mortality in Croatia and Serbia. *Sumarski List*, 139(3–4), 137–144.
- GRUNTAR, I., B. PAPIĆ, M. PATE, U. ZAJC, M. OCEPEK AND D. KUSAR. 2020. *Helicobacter labacensis* sp. nov., *Helicobacter mehlei* sp. nov., and *Helicobacter vulpis* sp. nov., isolated from gastric mucosa of red foxes (*Vulpes vulpes*). *Int. J. Systematic and Evolutionary Microbiology*. 70:2395-2404.
- GRZYBEK, A., A. CYBULSKA, K. TOLKACZ, M. ALSARRAF, J. BEHNKE-BOROWCZYK, K. SZCZEPANIAK, A. STRACHECKA, J. PALEOLOG, B. MOSKWA, J. M. BEHNKE AND A. BAJER. 2019. Seroprevalence of *Trichinella* spp. infection in bank voles (*Myodes glareolus*) – a long term study. *Parasites and Wildlife*. 9(2019):144-148.
- GUAN, Y., ZHENG, B. J., HE, Y. Q., LIU, X. L., ZHUANG, Z. X., CHEUNG, C. L., LUO, S. W., LI, P. H., ZHANG, L. J., GUAN, Y. J., BUTT, K. M., WONG, K. L., CHAN, K. W., LIM, W., SHORTRIDGE, K. F., YUEN, K. Y., PEIRIS, J. S. M., & POON, L. L. M. (2003). Isolation and characterization of viruses related to the SARS coronavirus from animals in Southern China. *Science*, 302(5643), 276–278.
- GULINELLO, M., ACQUARONE, M., KIM, J., SPRAY, D., BARBOSA, H., SELLERS, R., TANOWITZ, H., & WEISS, L. (2010). Acquired infection with *Toxoplasma gondii* in adult mice results in sensorimotor deficits but normal cognitive behavior despite widespread brain pathology. *Microbes and Infection*, 12(7), 528–537.
- GURNELL, J., A. M. GURNELL, D. DEMERITT, P. W. LURZ, M. D. F. SHIRLEY, S. P. RUSHTON, C. G. FAULKES, S. NOBERT AND E. J. HARE. 2009. The feasibility and acceptability of reintroducing the European beaver to England. Natural England Commissioned Report NECR002.
- GYURANECZ, M. (2012). Tularaemia. In D. Gavier-Widén, J. P. Duff, & A. Meredith (Eds.), *Infectious Diseases of Wild Mammals and Birds in Europe* (pp. 303–309). Wiley-Blackwell Oxford.
- GYURANECZ, M., SZEREDI, L., MAKRAI, L., FODOR, L., MÉSZÁROS, Á. R., SZÉPE, B., FÜLEKI, M., & ERDÉLYI, K. (2010). Tularemia of European brown hare (*Lepus europaeus*): A pathological, histopathological, and immunohistochemical study. *Veterinary Pathology*, 47(5), 958–963.
- HAITLINGER, R. 1991. Arthropods appearing on European beaver (*Castor fiber*) in Poland. *Katedra Zoologii*. 1991. pp. 107-109.

- HALLEY, D. J., & ROSELL, F. (2002). The beaver's reconquest of Eurasia: status, population development and management of a conservation success. *Mammal Review*, 32(3), 153–178.
- HALLEY, D., F. ROSELL AND A. P. SAVELJEV. 2012. Population and distribution of Eurasian beaver (*Castor fiber*). *Baltic Forestry* 18(1): 168-175.
- HAMMERI, J. A., R. G. ULRICH, C. IMHOLT, H. C. SCHOLZ, J. JACOB, N. KRATZMANN, K. NOCKLER AND S. AL DAHOUK. 2015. Molecular survey on Brucellosis in Rodents and Shrews – Natural reservoirs of novel Brucella species in Germany? *Transboundary and Emerging Diseases*. 64: 663-671.
- HANDELAND, K., L. L. NESSE, A. LILLEHAUG, T. VIKOREN, B. DJONNE AND B. BERGSJO. 2008. Natural and experimental *Salmonella Typhimurium* infections in foxes Norway. *Veterinary Microbiology*. 132(2008):129-134.
- HARBOUR, S. AND P. SUTTON. 2008. Immunogenicity and pathogenicity of *Helicobacter* infections of veterinary animals. *Veterinary Immunology and Immunopathology*. 122: 191-203.
- HARRINGTON, L. A., FEBER, R., & MACDONALD, D. W. (2010). The Scottish Beaver Trial: Ecological monitoring of the European beaver *Castor fiber* and other riparian mammals - 1st Annual Report 2010. 7, 53.
- HARRINGTON, L. A., M. GELLING, V. SIMPSON, A. HARRINGTON AND D. W. MACDONALD. 2012. Notes on the health status of free-living, non-native American mink, *Neovison vison*, in southern England. *Eur. J. Wild. Res.* 58:875-880.
- HARTMAN, G. (1995). Patterns of spread of a reintroduced beaver *Castor fiber* population in Sweden. *Wildlife Biology*, 1(1), 97–103.
- HAVLÍČEK, J., GAŠOVÁ, Z., SMITH, A. P., ZVÁRA, K., & FLEGR, J. (2001). Decrease of psychomotor performance in subjects with latent “asymptomatic” toxoplasmosis. *Parasitology*, 122(5), 515–520.
- HAZEL, S. M., M. BENNETT, J. CHANTREY, K. BOWN, R. CAVANAGH, T. R. JONES, D. BAXBY AND M. BEGON. 2000. A longitudinal study of an endemic disease in its wildlife reservoir: Cowpox and wild rodents. *Epidemiol. Infect.* 124: 551-562.
- HEALING, T. D. AND M. H. GREENWOOD. 1991. Frequency of isolation of *Campylobacter* spp., *Yersinia* spp. and *Salmonella* spp. from small mammals from two sites in southern Britain. *International Journal of Environmental Health Research*. 1(1):54-62.
- HEPBURN, M. J., & SIMPSON, A. J. H. (2008). Tularemia: current diagnosis and treatment options. *Expert Review of Anti-Infective Therapy*, 6(2), 231–240.
- HERRMANN, D. C., G. WIBBELT, M. GOTZ, F. J. CONRATHS AND G. SCHARES. 2013. Genetic characterization of *Toxoplasma gondii* from European beavers (*Castor fiber*) and European wildcats (*Felis silvestris silvestris*). *Veterinary Parasitology*. 191 (2013) 108-111.
- HESTVIK, G., WARNS-PETIT, E., SMITH, L. A., FOX, N. J., UHLHORN, H., ARTOIS, M., HANNANT, D., HUTCHINGS, M. R., MATTSSON, R., YON, L., & GAVIER-WIDEN, D. (2015). The status of



- tularemia in Europe in a one-health context: a review. *Epidemiology and Infection*, 143(10), 2137–2160.
- HEYMAN, P., J. KLINGSTRÖM, F. DE JAEGERE, G. LECLERCQ, F. ROZENFELD, S. ESCUTENAIRE, C. VANDENVELDE, M. ZIZI, A. PLYUSNIN, ANDA. LUNDKVIST. 2002. Tula hantavirus in Belgium. *Epidemiol Infect* 128: 251-256.
- HILL, D. E., CHIRUKANDOTH, S., & DUBEY, J. P. (2005). Biology and epidemiology of *Toxoplasma gondii* in man and animals. *Animal Health Research Reviews*, 6(1), 41–61.
- HINZE, G. (1950). *Der Biber: Körperbau und Lebensweise, Verbreitung und Geschichte*. Akademie-Verlag.
- HOFFMAN, J., S. WEISS, M. KUHNS, A. ZINKE, H. HEINSBERGER, D. H. KRUGER. 2018. Importation of human SEOUL virus infection to Germany from Indonesia. *Emerg. Inf. Dis.* 2018: 24 (6).
- HOFFMANN, M., KLEINE-WEBER, H., SCHROEDER, S., KRÜGER, N., HERRLER, T., ERICHSEN, S., SCHIERGENS, T. S., HERRLER, G., WU, N.-H., NITSCHKE, A., MÜLLER, M. A., DROSTEN, C., & PÖHLMANN, S. (2020). SARS-CoV-2 Cell Entry Depends on ACE2 and TMPRSS2 and Is Blocked by a Clinically Proven Protease Inhibitor. *Cell*, 181: 1–10.
- HOLDING, M., S. D. DOWALL, J. M. MEDLOCK, D. P. CARTER, L. MCGINLEY, M. CURRAN-FRENCH, S. T. PULLAN, J. CHAMBERLAIN, K. M. HANSFORD, M. BAYLIS, R. VIPOND AND R. HEWSON. 2019. Detection of new endemic focus of tick-borne encephalitis virus (TBEV), Hampshire/Dorset border, England, September 2019. *Euro Surveill.* 2019:24(47).
- HOLDING, M., S. D. DOWALL, J. M. MEDLOCK, D. P. CARTER, S. T. PULLAN, J. LEWIS, R. VIPOND, M. S. ROCCHI, M. BAYLIS AND R. HEWSON. 2020. Tick-borne encephalitis virus, United Kingdom. *Emerg. Inf. Dis.* 26(1):90-96.
- HOLLINGS, T., JONES, M., MOONEY, N., & MCCALLUM, H. (2013). Wildlife disease ecology in changing landscapes: Mesopredator release and toxoplasmosis. *International Journal for Parasitology: Parasites and Wildlife*, 2(1), 110–118.
- HOLMES, E. C., AND Z. ZHANG. 2015. The evolution and emergence of hantaviruses. *Curr Opin Virol* 10: 27-33.
- HOLSHUE, M. L., DEBOLT, C., LINDQUIST, S., LOFY, K. H., WIESMAN, J., BRUCE, H., SPITTERS, C., ERICSON, K., WILKERSON, S., TURAL, A., DIAZ, G., COHN, A., FOX, L. A., PATEL, A., GERBER, S. I., KIM, L., TONG, S., LU, X., LINDSTROM, S., PILLAI, S. K. (2020). First case of 2019 novel coronavirus in the United States. *New England Journal of Medicine*, 382(10), 929–936.
- HORCICKOVA, M., S. CONDLOVA, N. HOLUBOVA, B. SAK, D. KVETONOVA, L. HLASKOVA, R. KONECNY, F. SEDLACEK, M. CLARK, C. GIDDINGS, J. MCEVOY AND M. KVAC. 2019. Diversity of *Cryptosporidium* in common voles and description of *Cryptosporidium alticolis* sp. N. and *Cryptosporidium microti* sp. N. (*Apicomplexa: Cryptosporidiidae*). *Parasitology*. 146(2):220-233.
- HORTON, B., H. BRIDLE, C. L. ALEXANDER AND F. KATZER. 2018. *Giardia duodenalis* in the UK: Current knowledge of risk factors and public health implications. *Parasitology*. 146:413-424.

- HOWIE, F. 2009. Post-mortem report on beaver M08K29. SAC Veterinary Services. Ref: M094745.
- HUBALEK, Z. 1999. Emmonsiosis of wild rodents and insectivores in Czechland. *J. Wildlife Dis.* 35(2): 243-249.
- HUBÁLEK, Z., & HALOUZKA, J. (1997). Mosquitoes (Diptera: Culicidae), in Contrast to Ticks (Acari: Ixodidae), Do Not Carry *Francisella tularensis* in a Natural Focus of Tularemia in the Czech Republic. *Journal of Medical Entomology*, 34(6), 660–663.
- HUGHES, K. AND A. M. BORMAN. 2018. Adiaspiromycosis in a wild European rabbit and a review of the literature. *Journal of Veterinary Diagnostic Investigation.* 30(4):614-618.
- IDEOZU, E. J., A. M. WHITEOAK, A. J. TOMLINSON, A. ROBERTSON, R. J. DELAHAY AND G. HIDE. 2015. High prevalence of trypanosomes in European badgers detected using ITS-PCR. *Parasites & Vectors.* 2015 (8):480.
- IDEXX. (2020). IDEXX SARS-CoV-2 (Covid-19) RealPCR Test. <https://www.idexx.com/en/veterinary/reference-laboratories/idexx-sars-cov-2-covid-19-realpcr-test/>
- IJAZ, M. K., BRUNNER, A. H., SATTAR, S. A., NAIR, R. C., & JOHNSON-LUSSENBURG, C. M. (1985). Survival characteristics of airborne human coronavirus 229E. *Journal of General Virology*, 66(12), 2743–2748.
- IUCN. 2013. Guidelines for reintroductions and other conservation translocations. Version 1.0. Gland, Switzerland: IUCN Species Survival Commission.
- IZDEBSKA, J. N., S. FRYDERYK AND L. ROLBIECKI. 2016. *Demodex castoris* sp. Nov. (Acari: Demodecidae) parasitising *Caster fiber* (Rodentia) and other parasitic arthropods associated with *Castor* spp. *Diseases of Aquatic Organisms.* 118: 1-11.
- JAKOB-HOFF, R.M., S. C. MACDIARMID, C. LEES, P. S. MILLER, D. TRAVIS AND R. KOCK. 2014. Manual of Procedures for Wildlife Disease Risk Analysis. World Organisation for Animal Health, Paris, 160 pp. Published in association with the international Union for Conservation of Nature and the Species Survival Commission. ISBN: 978-92-9044-957-7.
- JAMESON, L. J., A. NEWTON, L. COOLE, E. N. NEWMAN, M. W. CARROLL, N. J. BEECHING, R. HEWSON, ANDR. M. CHRISTLEY. 2014. Prevalence of antibodies against hantaviruses in serum and saliva of adults living or working on farms in Yorkshire, United Kingdom. *Viruses* 6: 524-534.
- JANISZEWSKI, P., V. HANZAL AND W. MISIUKIEWCZ. 2014. The European beaver (*Castor fiber*) as a keystone species – a literature review. *Baltic Forestry* 20(2):277-286.
- JANOVSKY, M., L. BACCIARINIA, H. SAGER, A. GRONE AND B. GOTTSTEIN. 2002. *Echinococcus multilocularis* in a European beaver from Switzerland. *Journal of Wildlife Diseases.* 38(3):618-620.
- JARQUIN-DIAZ, V. H., A. BALARD, A. MACOVA, J. JOST, T. R. VON SZEPESBELA, K. BERKTOLD, S. TANK, J. KVICEROVA AND E. HEITLINGER. 2019. Generalist *Eimeria* species in rodents: Multilocus analyses indicate resolution of established markers. *Ecology and Evolution.* 2020. 10:1378-1389.

- JELLISON, W. L. AND J. W. VINSON. 1961. The distribution of *Emmonsia Crescens* in Europe. *Mycologia*. 53:5, 524-535.
- JENUM, P. A., KAPPERUD, G., STRAY-PEDERSEN, B., MELBY, K. K., ESKILD, A., & ENG, J. (1998). Prevalence of *Toxoplasma gondii* specific immunoglobulin G antibodies among pregnant women in Norway. *Epidemiology and Infection*, 120(1), 87–92.
- JONES, A. C. L., HALLEY, D. J., GOW, D., BRANSCOME, J., & AYKROYD, T. (2012). Welsh Beaver Assessment Initiative Report: An investigation into the feasibility of reintroducing European beaver (*Castor fiber*) to Wales.
- JONES, J. AND CAMPBELL-PALMER, R. 2013. The Battle for the British beaver. *British Wildlife*. Aug. 2013.
- JONES, J. AND R. CAMPBELL-PALMER. 2014. The Scottish Beaver Trial: The story of Britain's first licensed release into the wild. SWT AND RZSS.
- JONES, K. E., PATEL, N. G., LEVY, M. A., STOREYGARD, A., BALK, D., GITTLEMAN, J. L., & DASZAK, P. (2008). Global trends in emerging infectious diseases. *Nature*, 451(7181), 990–993.
- JORDAN, C. N., KAUR, T., KOENEN, K., DESTEFANO, S., ZAJAC, A. M., & LINDSAY, D. S. (2005). Prevalence of Agglutinating Antibodies to *Toxoplasma gondii* and *Sarcocystis neurona* in Beavers (*Castor canadensis*) From Massachusetts. *Journal of Parasitology*, 91(5), 1228–1229.
- JORGENSEN, H. J., K. HAUGE, H. LANGE, E. MACDONALD, T. M. LYNGSTAD, B. T. HEIER. 2018. The Norwegian Zoonoses Report. Norwegian Veterinary Institute.
- JOST, B. H., J. G. SONGER AND S. J. BILLINGTON. 1999. An arcanobacterium (*Actinomyces*) pyogenes mutant deficient in production of the pore-forming cytolysin Pyolysin has reduced virulence. *Infection and Immunity*. 67(4): 1723-1728.
- KALLIO, E. R., H. HENTTONEN, E. KOSKELA, A. LUNDKVIST, T. MAPPES AND O. VALALAHTI. 2013. Maternal antibodies contribute to sex-biased difference in hantavirus transmission dynamics. *Biol Lett* 2013, 9.
- KALLIO, E. R., J. KLINGSTRÖM, E. GUSTAFSSON, T. MANNI, A. VAHERI, H. HENTTONEN, O. VAPALAHTI, ANDA. LUNDKVIST. 2006. Prolonged survival of Puumala hantavirus outside the host: evidence for indirect transmission via the environment. *J Gen Virol* 87: 2127-2134.
- KAMPEN, A. H., G. T. TESSEMA, A. AGDESTAIN, T. MOLDAL, M. VALHEIM AND C. ER. 2019. The surveillance programme for paratuberculosis in Norway 2018. Mattilsynet, Norwegian Veterinary Institute.
- KAMPF, G., TODT, D., PFAENDER, S., & STEINMANN, E. (2020). Persistence of coronaviruses on inanimate surfaces and their inactivation with biocidal agents. *Journal of Hospital Infection*, 104(3), 246–251.
- KANG, E., A. CROUSE, L. CHEVALLIER, S. M. PONTIER, A. ALZHRANI, N. SILUE, F.X. CAMPBELL-VALOIS, X. MONTAGUTELLI, S. GRUENHEID AND D. MALO. 2018. Enterobacteria and host resistance to infection. *Mammalian Genome*. 29, 558-576.

- KAPEL, C. M. O. 2001. Sylvatic and domestic *Trichinella* spp. in wild boars: Infectivity, muscle larvae distribution and antibody response. *J. of Parasitol.* 87(2):309-314.
- KAPLAN, C., T. D. HEALING, N. EVANS, L. HEALING AND A. PRIOR. 1980. Evidence of infection by viruses in small British field rodents. *J. Hyg.* 84, 285.
- KAPPERUD, G. 1975. *Yersinia enterocolitica* in small rodents from Norway, Sweden and Finland. *Acta Pathologica Microbiologica Scandinavica*.
- KARAMON, J., M. SAMOREK-PIEROG, M. KOCHANOWSKI, J. DABROWSKA, J. SROKA, E. GOLAB AND T. CENCEK. 2016. First detection of *Echinococcus multilocularis* in dogs in a highly endemic area of Poland. *Folia Parasitologica.* 63.
- KEIM, P., JOHANSSON, A., & WAGNER, D. M. (2007). Molecular epidemiology, evolution, and ecology of *Francisella*. *Annals of the New York Academy of Sciences*, 1105, 30–66.
- KEMP, P. S., WORTHINGTON, T. A., LANGFORD, T. E. L., TREE, A. R. J., & GAYWOOD, M. J. (2012). Qualitative and quantitative effects of reintroduced beavers on stream fish. *Fish and Fisheries*, 13(2), 158–181. <https://doi.org/10.1111/j.1467-2979.2011.00421.x>
- KESMINAS, V., STEPONENAS, A., PLIURAITĖ, V., & VIRBICKAS, T. (2013). Ecological impact of Eurasian beaver (*Castor fiber*) activity on fish communities in Lithuanian trout streams. *Rocznik Ochrona Srodowiska*, 15(1), 59–80.
- KEYMER, I. F. (1983). Diseases of squirrels in Britain. *Mammal Review*, 13(2-4), 155–158.
- KIM, J-H, LEE, J. Y., & CHOI, S. H. (2005). Odontoplasty for the treatment of malocclusion of the incisor teeth in a beaver (*Castor canadensis*). *Veterinary Record*, 156, 114–115.
- KIM, JOONG-HYUN, LEE, J. Y., HAN, T., HAN, K., KANG, S. S., BAE, C. S., & CHOI, S. H. (2005). Veterinary Science A case of maloccluded incisor teeth in a beaver (*Castor canadensis*). *Journal of Veterinary Science*, 6(2), 173–175.
- KITCHENER, A. (2001). *Beavers*. Whittet Books Ltd.
- KLINGSTRÖM, J., P. HEYMAN, S. ESCUTENAIRE, K. B. SJÖLANDER, F. DE JAEGERE, H. HENTTONEN, ANDA. LUNDKVIST. 2002. Rodent host specificity of European hantaviruses: evidence of Puumala virus interspecific spillover. *J Med Virol* 68: 581-588.
- KLOSKOWSKI, J. (2011). Human-wildlife conflicts at pond fisheries in eastern Poland: Perceptions and management of wildlife damage. *European Journal of Wildlife Research*, 57(2), 295–304.
- KLUN, I., N. COSIC, D. CIROVIC, D. VASILEV, V. TEODOROVIC AND O. DJURKOVIC-DJAKOVIC. 2019. *Trichinella* spp. in wild mesocarnivores in an endemic setting. *Acta Veterinaria Hungarica*. 67(1):34-39.
- KNAPP, J., P. GIRAUDOUX, B. COMBES, G. UMHANG, F. BOUE, Z. SAID-ALI AND F. RAOUL. 2018. Rural and urban distribution of wild and domestic carnivore stools in the context of *Echinococcus multilocularis* environmental exposure. *International Journal for Parasitology*. 48(12):937-946.

- KOCAZEYBEK, B., ONER, Y. A., TURKSOY, R., BABUR, C., CAKAN, H., SAHIP, N., UNAL, A., OZASLAN, A., KILIC, S., SARIBAS, S., ASLAN, M., TAYLAN, A., KOC, S., DIRICAN, A., UNER, H. B., OZ, V., ERTEKIN, C., KUCUKBASMALI, O., & TORUN, M. M. (2009). Higher prevalence of toxoplasmosis in victims of traffic accidents suggest increased risk of traffic accident in Toxoplasma-infected inhabitants of Istanbul and its suburbs. *Forensic Science International*, 187(1–3), 103–108.
- KOSMIDER, R., A. PATERSON, A. VOAS AND H. ROBERTS. 2013. *Echinococcus multilocularis* introduction and establishment in wildlife via imported beavers. *Veterinary Record*. 172(3):606.
- KRAMER, A., I. SCHWEBKE AND G. KAMPF. 2006. How long do nosocomial pathogens persist on inanimate surfaces? A systematic review. *BMC Infect Dis* 6, 130.
- KRIJGER, I. M., B. G. MEERBURG, C. HARMANUS AND S. A. BURT. 2019. *Clostridium difficile* in wild rodents and insectivores in the Netherlands. *Letters in Applied Microbiol.* 69(1).
- KUBA, K., IMAI, Y., RAO, S., GAO, H., GUO, F., GUAN, B., HUAN, Y., YANG, P., ZHANG, Y., DENG, W., BAO, L., ZHANG, B., LIU, G., WANG, Z., CHAPPELL, M., LIU, Y., ZHENG, D., LEIBBRANDT, A., WADA, T., ... PENNINGER, J. M. (2005). A crucial role of angiotensin converting enzyme 2 (ACE2) in SARS coronavirus-induced lung injury. *Nature Medicine*, 11(8), 875–879.
- LAACKONEN, J., A. SUKURA, A. OKSANEN, H. HENTTONEN AND T. SOVERI. 2001. Haemogregarines of the genus Hepatozoon in rodents from northern Europe. *Folia Parasitologica* 48:263-267.
- LAMPIO, T. (1967). Sex ratios and the factors contributing to them in the squirrel, *Sciurus vulgaris*, in Finland. *Finish Game Research*, 29, 5–69.
- LANGE, H., O. H. JOHANSEN, L. VOLD, L. J. ROBERTSON. I. L. ANTHONISEN AND K. NYGARD. Second outbreak of infection with a rare *Cryptosporidium parvum* genotype in schoolchildren associated with contact with lambs and goat kids at a holiday farm in Norway. *Epidemiol Infect.* 142:2105-2113.
- LARSEN, K. W., AFSET, J. E., HEIER, B. T., KROGH, T., HANDELAND, K., VIKØREN, T., & BERGH, K. (2011). Outbreak of tularaemia in central Norway, January to March 2011. *Eurosurveillance*, 16(13), 10–12.
- LASSEN, B., LEPIK, T., & BANGOURA, B. (2013). Persistence of *Eimeria bovis* in soil. *Parasitology Research*, 112(7).
- LAURENT, F. 2019. How innate immune responses shape *Cryptosporidium* infection. VIIIth International *Giardia* and *Cryptosporidium* Conference. Rouen, France.
- LAWSON, P. A., G. FOSTER, E. FALSEN, S. J. MARKOPOULOS AND M. D. COLLINS. 2005. *Streptococcus castoreus* sp. nov. isolated from a beaver (*Castor fiber*). *Int. J. Systematic and Evol. Microbiol.* 55, 843-846.
- LEARMOUNT, J., V. BOUGHTFLOWER, P. C. ALLANSON, K. M. HARTLEY, A. BARRECHEGUREN GUTIERREZ, N. A. STEPHENS, G. MARUCCI AND G. C. SMITH. 2015. *Veterinary Parasitology*. 208:259-262.

- LEIGHTON, F.A. 2002. Health risk assessment of the translocation of wild animals. *Revue scientifique et technique – Office international des epizooties*, 21: 187-216.
- LEVETT, P. N. (2001). "Leptospirosis." *Clin Microbiol Rev* 14(2): 296-326.
- LI, Q., GUAN, X., WU, P., WANG, X., ZHOU, L., TONG, Y., REN, R., LEUNG, K. S. M., LAU, E. H. Y., WONG, J. Y., XING, X., XIANG, N., WU, Y., LI, C., CHEN, Q., LI, D., LIU, T., ZHAO, J., LIU, M., FENG, Z. (2020). Early Transmission Dynamics in Wuhan, China, of Novel Coronavirus-Infected Pneumonia. *The New England Journal of Medicine*, 382(13), 1199–1207.
- LI, WENDONG, SHI, Z., YU, M., REN, W., SMITH, C., EPSTEIN, J. H., WANG, H., CRAMERI, G., HU, Z., ZHANG, H., ZHANG, J., MCEACHERN, J., FIELD, H., DASZAK, P., EATON, B. T., ZHANG, S., & WANG, L. F. (2005). Bats are natural reservoirs of SARS-like coronaviruses. *Science*, 310(5748), 676–679.
- LI, WENHUI, MOORE, M. J., VASILIEVA, N., SUI, J., WONG, S. K., BERNE, M. A., SOMASUNDARAN, M., SULLIVAN, J. L., LUZURIAGA, K., GREENOUGH, T. C., CHOE, H., & FARZAN, M. (2003). Angiotensin-converting enzyme 2 is a functional receptor for the SARS coronavirus. *Nature*, 426(NOVEMBER), 450–454.
- LINDBERG, A. (Ed.). 2018. Surveillance of infectious diseases in animals and humans in Sweden. National Veterinary Institute (SVA), Uppsala, Sweden.
- LINDSAY, D. S., COLLINS, M. V., MITCHELL, S. M., COLE, R. A., FLICK, G. J., WETCH, C. N., LINDQUIST, A., & DUBEY, J. P. (2003). Sporulation and Survival of *Toxoplasma gondii* Oocysts in Seawater. *Journal of Eukaryotic Microbiology*, 50(SUPPL.), 687–688.
- LING J., J. VERNER-CARLSSON, P. ERIKSSON, A. PLYUSNINA, M. LOHMUS, J. D. JARHULT, F. VAN DER GROOT, A. PLYUSIN, A. LUNDKVIST AND T. SIRONEN. 2019. Genetic analysis of SEOUL hantavirus genome recovered from rats (*Rattus norvegicus*) in the Netherlands unveils diverse routes of spread into Europe. *J. Med. Vir.* 2019: 91(5).
- LIPTON, H. H., B. S. KIM, H. YAHIKOZAWA AND C. F. NADLER. 2001. Serological evidence that *Mus musculus* is the natural host of Theiler's Murine Encephalomyelitis Virus. *Virus Res.* 76(1):79-86.
- LIU, Q., WANG, Z. D., HUANG, S. Y., & ZHU, X. Q. (2015). Diagnosis of toxoplasmosis and typing of *Toxoplasma gondii*. *Parasites and Vectors*, 8(1), 1–14.
- LIU, Y., EGGO, R. M., & KUCHARSKI, A. J. (2020). Secondary attack rate and superspreading events for SARS-CoV-2. *The Lancet*, 395(10227), e47.
- LIU, Y., YAN, L. M., WAN, L., XIANG, T. X., LE, A., LIU, J. M., PEIRIS, M., POON, L. L. M., & ZHANG, W. (2020). Viral dynamics in mild and severe cases of COVID-19. *The Lancet Infectious Diseases*, 2019(20), 2019–2020.
- LU, C., LU, X., & JIA, Z. (2020). 2019-nCoV transmission through the ocular surface must not be ignored. *Chaolin*. *The Lancet*, 395(February), e39.
- LU, R., ZHAO, X., LI, J., NIU, P., YANG, B., WU, H., WANG, W., SONG, H., HUANG, B., ZHU, N., BI, Y., MA, X., ZHAN, F., WANG, L., HU, T., ZHOU, H., HU, Z., ZHOU, W., ZHAO, L., TAN, W. (2020). Genomic

characterisation and epidemiology of 2019 novel coronavirus: implications for virus origins and receptor binding. *The Lancet*, 395(10224), 565–574.

- MACA, O., I. PAVLASEK AND A. VOREL. 2015. *Stichorchis subtriquetrus* (Digenea: Paramphistomatidae) from Eurasian beaver (*Castor fiber*) in the Czech Republic. Springer Verlag. *Parasitol Res* 114:2933–2939.
- MACDONALD, D. W., TATTERSALL, F. H., BROWN, E. D., & BALHARRY, D. (1995). Reintroducing the European Beaver to Britain: nostalgic meddling or restoring biodiversity? *Mammal Review*, 25(4), 161–200. <https://doi.org/10.1111/j.1365-2907.1995.tb00443.x>
- MACIEIRA, M. R. 2019. Screening of diseases in Swedish muskrats. Swedish National Veterinary Institute.
- MACKIE, P. 2014. Scottish Beaver Trial: Independent Public Health Monitoring 2009-2014. Report and Recommendations. Argyll and Bute Council.
- MAIR, N. S. 1973. Yersiniosis in wildlife and its public health implications. *Journal of Wildlife Diseases*, 9: 64-71.
- MARREROS N., S. ZURCHER-GIOVANNINI, F. C. ORIGGI, Z. DJELOUADJI, J. WIMMERSHOFF, M. PEWSNER, E. AKDESIR, M. BATISTA LINHARES, A. KODJO AND M-P. RYSER-DEGIORGIS (2018). “Fatal Leptospirosis in free-ranging Eurasian beavers (*Castor fiber*) Switzerland.” *Transbound Emerg Dis* 2018. 00:1-10
- MARTIN, L., A. LECLERQ, C. SAVIN AND E. CARNIEL. 2009. Characterisation of atypical isolates of *Yersinia intermedia* and definition of two new biotypes. *J. Clin. Microbiology* Aug. 2009 pp. 2377-2380.
- MASTERS, P. S. (2006). The Molecular Biology of Coronaviruses. *Advances in Virus Research*, 65(06), 193–292.
- MATEO, M., M. HERNANDEZ DE MINGO, A. DE LUCIO, L. MORALES, A. BALSEIRO, A. ESPI, M. BARRAL, J. F. L. BARBERO, M. A. HABELA, J. L. FERNANDEZ-GARCIA, R. C. BERNAL, P. C. KOSTER, G. A. CARDONA AND D. CARMENA. 2017. *Veterinary Parasitology*. 235(2017) 86-93.
- MATHEWS, F., L. M. KUBASIEWICZ, J. GURNELL, C. A. HARROWER, R. A. MCDONALD AND R. F. SHORE. 2018. A Review of the Population and Conservation Status of British Mammals. A report by the Mammal Society under contract to Natural England, Natural Resources Wales and Scottish Natural Heritage. Natural England, Peterborough. ISBN 978-1-78354-494-3. Natural England Access to Evidence Catalogue code JP025
- MATHEWS, F., MORO, D., STRACHAN, R., GELLING, M. AND BULLER, N., 2006. Health surveillance in wildlife reintroductions. *Biological Conservation*. 131:2.
- MATHIS, A. AND P. DEPLAZES. 2002. In Craig and Pawlowski (Eds.). *Cestode Zoonoses: Echinococcosis and Cisticercosis. An emergent and global problem*. IOS Press.
- MAURIN, M. AND M. GYURANECZ. (2016). Tularaemia: clinical aspects in Europe. *The Lancet Infectious Diseases*, 16(1), 113–124. [https://doi.org/10.1016/S1473-3099\(15\)00355-2](https://doi.org/10.1016/S1473-3099(15)00355-2)

- MAYER, M., FRANK, S. C., ZEDROSSER, A., & ROSELL, F. (2020). Causes and consequences of inverse density-dependent territorial behaviour and aggression in a monogamous mammal. *Journal of Animal Ecology*, 89(2), 577–588.
- MAYER-SCHOLL, A., J. A. HAMMERL, S. SCHMIDT, R. G. ULRICH, M. PFEFFER, D. WOLL, H. C. SCHOLZ, A. THOMAS AND K. NÖCKLER (2014). "*Leptospira* spp. in rodents and shrews in Germany." *Int J Environ Res Public Health* 11(8): 7562-7574.
- MCCOY, G. W. (1911). A plague-like disease of rodents. *Public Health Bull*, 43, 53–71.
- MCCOY, G. W., & CHAPIN, C. W. (1912). Bacterium tularensis, the cause of a plague-like disease of rodents. *Public Health Bull*, 53, 17–23.
- MCDONALD, V., & SHIRLEY, M. W. (2009). Past and future: vaccination against *Eimeria*. *Parasitology*, 136, 1477–1489. <https://doi.org/10.1017/S0031182009006349>
- MCEWING, R., H. SENN AND R. CAMPBELL-PALMER. 2015. Genetic assessment of free-living beavers in and around the river Tay catchment, East Scotland. SNC Commissioned Report No. 682.
- MCGILL, I., Y. FELTRER, C. JEFFS, G. SAYERS, R. M. MARSHALL, M. A. PEIRCE, M. P. STIDWORTHY, A. M. POCKNELL AND A. W. SAINSBURY. 2010. Isosporoid coccidiosis in translocated ciril buntings (*Emberiza cirilus*). *Veterinary Record* 167: 656-660.
- MCKINSTRY, M. C., & ANDERSON, S. H. (2002). Survival, fates and success of transplanted beavers, *Castor canadensis*, in Wyoming. *The Canadian Field-Naturalist*, 116, 60–68.
- MCKOWN, R. D., VEATCH, J. K., ROBEL, R. J., AND, S., & UPTON, S. J. (1995). Research Note - Endoparasites of Beaver (*Castor canadensis*) from Kansas. *Journal of the Helminthological Society of Washington*, 62(1), 89–93.
- MEERBURG, B. G. AND A. KIJLSTRA. 2007. *Role of rodents in transmission of Salmonella and Campylobacter*. *J. Sci. Food Agric.* 87:2774-2781.
- MELTER, O. AND R. CASTELHANO (Eds.). 2019. Enterobacteria and Enteric Pathogens. In *The Microbook: Clinical Microbiology for Medical Students*. Charles University Karolinum Press.
- MEREDITH, A. 2012. Rotavirus infections. In G. Gavier-Widen, P.D. Duff & A. Meredith (Eds.), *Infectious Diseases of Wild Mammals and Birds in Europe* (First edition pp. 345-362). Blackwell Publishing Ltd., UK. Chapter 19.
- MEREDITH, A. L., S. C. CLEVELAND, J. BROWN, A. MAHAJAN AND D. J. SHAW. 2015. Seroprevalence of Encephalitozoon cuniculi in wild rodents, foxes and domestic cats in three sites in the United Kingdom. *Transboundary and Emerging Diseases*, vol. 62 (2) 148-156.
- MEREDITH, A., J. DEL POZO, S. SMITH, E. MILNE, K. STEVENSON AND J. MCLUCKIE. 2014. Leprosy in red squirrels in Scotland. *The Veterinary Record*, 175(11): 285-286. DOI: 10.1136/vr.g5680
- MEREDITH, A., S. CLEVELAND, M. J. DENWOOD, J. BROWN AND D. SHAW. 2015. *Coxiella burnetii* (Q fever) seroprevalence in prey and predators in the United Kingdom: evaluation of infection in



- wild rodents, foxes and domestic cats using a modified ELISA. *Transboundary and Emerging Diseases*, vol. 62, no. 6, pp. 639-649.
- MESFIN, G. M., BELLAMY, J. E. C., & STOCKDALE, P. H. G. (1977). The Pathological Changes Caused by *Eimeria falciformis* var. *pragensis* in Mice. *Canadian Journal of Comparative Medicine*.
- MET OFFICE. 2020. <https://www.metoffice.gov.uk/research/climate/maps-and-data/uk-climate-extremes#?tab=climateExtremes>
- MICHELET, L., K. DE CRUZ, G. ZANELLA, R. AAZIZ, T. BULACH, C. KAROUL, S. HENAULT, G. JONCOUR AND M. L. BOSCHOROLI. 2015. Infection with *Mycobacterium microti* in animals in France. *J. Clin. Microbiol.* 53(3):981-985.
- MICHELITSCH, A., K. WERNIKE, C. KLAUS, G. DOBLER AND M. BEER. 2019. Exploring the reservoir hosts of Tick-Borne Encephalitis Virus. *Viruses*. 11:969.
- MILLAN, J., G. ADURIZ, B. MORENO, R. A. JUSTE AND M. BARRAL. 2004. *Salmonella* isolates from wild birds and mammals in the Basque Country (Spain). *Rev. Sci. Tech. Off. Int. Epiz.* 23(3):905-911.
- MILLER, A., G. OLSSON, M. WALBURG, S. SOLLENBERG, M. SKARIN AND J. HOGLUND. 2017. Transmission ecology of taeniid larval cestodes in rodents in Sweden, a low endemic area for *Echinococcus multilocularis*. *Parasitology*. 144(8):1041-1051.
- MILLER, A., G. OLSSON, M. WALBURG, S. SOLLENBERG, M. SKARIN, C. LEY AND J. HOGLUND. 2016. First identification of *Echinococcus multilocularis* in rodent intermediate hosts in Sweden. *Int. J. for Parasitol: Parasites and Wildlife*. 5(1):56-63.
- MILLER, P.S. 2007. Tools and techniques for disease risk assessment in threatened wildlife conservation programmes. *International Zoo Yearbook*, 41: 38-51.
- MINETTI, C., K. LAMDEN, C. DURBAND, J. CHEESBROUGH, K. PLATT, A. CHARLETT, S. J. O'BRIEN, A. FOX AND J. M. WASTLING. 2015. Case-control study of risk factors for sporadic giardiasis and parasite assemblages in North-West England. *J Clin Microbiol.* 53(10):3133-40.
- MONAHAN, A., J. J. CALLANAN AND J. E. NALLY (2009). "Host-pathogen interactions in the kidney during chronic leptospirosis." *Vet Pathol* 46: 792-799.
- MORLEY, L. C. (1934). Report on internal parasites of beaver. *Pennsylvania Game News*, 5(16), 5.
- MÖRNER, T. (1992). The ecology of tularaemia. *Revue Scientifique et Technique (International Office of Epizootics)*, 11(4), 1123–1130.
- MÖRNER, T., & SANDSTEDT, K. (1983). A serological survey of antibodies against *Francisella tularensis* in some Swedish mammals. *Nordisk Veterinärmedicin*, 35(2), 82–85.
- MÖRNER, T., A. AVENAS AND R. MATTSON. 1999. Adiaspiromycosis in a European beaver from Sweden. *J. Wildlife Dis.* 35(2): 367-70.
- MORNER, T., C. BROJER, M-P. RYSER-DEGIORGIS, D. GAVIER-WIDEN, H-O. NILSSON AND T. WADSTROM. 2008. Detection of gastric *Helicobacter* species in free-ranging lynx (*Lynx lynx*) and red foxes (*Vulpes vulpes*) in Sweden. *Journal of Wildlife Diseases*. 44(3):697-700.

- MÖRNER, T., SANDSTROM, G., & MATTSSON, R. (1988). Comparison of serum and lung extracts for surveys of wild animals for antibodies to *Francisella tularensis* biovar *palaeartica*. *Journal of Wildlife Diseases*, 24(1), 10–14.
- MÖRNER, T., SANDSTROM, G., MATTSSON, R., & NILSSON, P. (1988). Infections with *Francisella tularensis* biovar *palaeartica* in hares (*Lepus timidus*, *Lepus europaeus*) from Sweden. *Journal of Wildlife Diseases*, 24(3), 422–433.
- MUHLDORFER, K., J. RAU, A. FAWZY, C. HEYDEL, S. P. GLAESER, M. VAN DER LINDEN, P. KUTZER, T. KNAUF-WITZENS, M. HANCZARUK, A. S. ECKERT AND T. EISENBERG. 2019. *Streptococcus castoreus*, an uncommon *A. Streptococcus* in beavers. *Antonie van Leeuwenhoek*. 112:1663-1673.
- MÜLLER, F., (2014). Individuelle Variation von Körpermerkmalen bei Biber *Castor fiber*, Castoridae. Tagungsband Ergebnisse der Nationalen Bibertagung in Dessau – Rosslau, Sachsen – Anhalt, Dessau, s. 163–167.
- MURRAY, N., S. MACDIARMID, M. WOOLDRIDGE, B. GUMMOW, R. MORLEY, S. WEBER, A. GIOVANNINI AND D. Wilson. (2004). *Handbook on Import Risk Analysis for Animals and Animal Products – Introduction and Qualitative Risk Analysis*. Paris: OIE (World Organisation for Animal Health).
- NOLET, B. A. AND F. ROSELL. 1998. Comeback of the beaver *Castor fiber*: An overview of old and new conservation problems. *Biological Conservation*. 83(2): 165-173.
- NAJDENSKI, H. in GAVIER-WIDEN, D., J. P. DUFF AND A. MEREDITH (Eds.), 2012, *Infectious Diseases of Wild Mammals and Birds in Europe*, Wiley Blackwell. Ch. 21 pp. 293-300.
- NATIONAL CENTRE FOR BIOTECHNOLOGICAL INFORMATION. (2020). NCBI Orthologs - ACE2 - angiotensin I converting enzyme 2.
- NEIMANIS, A. AND S. SPECK. Clostridium Species. Chapter 36. In G. Gavier-Widen, P.D. Duff & A. Meredith (Eds), *Infectious Diseases of Wild Mammals and Birds in Europe* (First edition pp. 345-362). Blackwell Publishing Ltd., UK.
- NEWS.GOV.HK. (2020). Pet Cat Tests Positive for Covid-19. [https://www.news.gov.hk/eng/2020/03/20200331/20200331\\_220128\\_110.html](https://www.news.gov.hk/eng/2020/03/20200331/20200331_220128_110.html)
- NFSA. 2020. Norwegian Food Safety Authority. Mattilsynet. <http://mattilsynet.no>. Visited 15/03/2020.
- NICHOLAS, A. J. AND M. GIACOMETTI. 2012. Mycoplasma infections. In G. Gavier-Widen, P.D. Duff & A. Meredith (Eds), *Infectious Diseases of Wild Mammals and Birds in Europe* (First edition pp. 345-362). Blackwell Publishing Ltd., UK.
- NICHOLS, G., R. CHALMERS, I. LAKE, W. SOPWITH, M. REGAN, P. HUNTER, P. GRENDEL, F. HARRISON AND C. LANE. 2006. Cryptosporidiosis: A report on the surveillance and epidemiology of *Cryptosporidium* infection in England and Wales. *Cryptosporidium Epidemiology*. Drinking Water Inspectorate. DWI 70/2/201.

- NISKANEN, T., J. WALDENSTRÖM, M. FREDRIKSSON-AHOMAA, B. OLSEN, ANDH. KORKEALA. 2003b. virF-positive *Yersinia pseudotuberculosis* and *Yersinia enterocolitica* found in migratory birds in Sweden. *Appl Environ Microbiol* 69: 4670-4675.
- NOLET B. A., S. BROEKHUIZEN, G. M. DORRESTEIN AND K. M. RIENKS (1997) Infectious diseases as main causes of mortality to beavers after translocation to the Netherlands. *J. Zool., Lond.* (1997) 241 35-42.
- NORDSTOGA, A., HANDELAND, K., JOHANSEN, T. B., IVERSEN, L., GAVIER-WIDÉN, D., MATTSSON, R., WIK-LARSSON, K., AFSET, J. E., NÆVERDAL, R., & LUND, A. (2014). Tularaemia in Norwegian dogs. *Veterinary Microbiology*, 173(3–4), 318–322.
- NORTON, C. C., & CHARD, M. J. (1983). The oocyst sporulation time of *Eimeria* species from the fowl. *Parasitology*, 86, 193–198.
- O'BRIEN, M. F., MELDRUM, J., & FOSTER, I. (2018). Medical and surgical management of intraspecific wounds in a European beaver kit (*Castor fiber*). *Veterinary Record Case Reports*, 6(e000561), 1–5. <https://doi.org/10.1136/vetreccr-2017-000561>
- OGLESBEE, B. L. AND J. R. JENKINS. 2012. Gastrointestinal Diseases. In QUESENBERRY, K. E. AND J. W. CARPENTER (Eds.). *Ferrets, Rabbits and Rodents. Elsevier Public Health Emergency Collection.* Chapter 15. 193-204.
- OIE (2018). *Terrestrial Manual*. Chapter 3.1.12 – Leptospirosis pp. 503-509.
- OKSANEN, A., M. SILES-LUCAS, J. KARAMON, A. POSSENTI, F. CONRATHS, T. ROMIG AND A. CASULLI. 2016. The geographical distribution and prevalence of *Echinococcus multilocularis* in animals in the European Union and adjacent countries: A systematic review and meta-analysis. *Parasites and Vectors*. 9(1).
- OLDHAM, J. N. AND W. P. BERESFORD-JONES. 1957. *British Veterinary Journal*, 113: 34-35.
- OLIVER, M. K., S. TELFER AND S. B. PIERTNEY. 2009. Major histocompatibility complex (MHC) heterozygote superiority to natural multi-parasite infections in the water vole (*Arvicola terrestris*). *Proceedings of the Royal Society of Biology*. 2009. 276:1119-1128.
- OTCENASEK, M., B. ROSICKY, K. KRIVANEC, J. DVORAK AND K. RASIN. 1974. The muskrat as reservoir in natural foci of adiaspiromycosis. *Folia Parasitologica (Praha)*. 21:55-57.
- OTCENASEK, M., K. KRIVANEC AND J. SLAIS. 1975. *Emmonsia parva* as causal agent of adiaspiromycosis in in fox. *Sabouradia*. 13:52-57.
- OWEN, M. R., & TREES, A. J. (1998). Vertical transmission of *Toxoplasma gondii* from chronically infected house (*Mus musculus*) and field (*Apodemus sylvaticus*) mice determined by polymerase chain reaction. *Parasitology*, 116(4), 299–304.
- PARAMESWARAN, N., O'HANDLEY, R. M., GRIGG, M. E., WAYNE, A., & THOMPSON, R. C. A. (2009). Vertical transmission of *Toxoplasma gondii* in Australian marsupials. *Parasitology*, 136(9), 939–944.

- PAVEY, C. R., ELDRIDGE, S. R., & HEYWOOD, M. (2008). Population dynamics and prey selection of native and introduced predators during a rodent outbreak in arid Australia. *Journal of Mammalogy*, 89(3), 674–683.
- PAWELCZYK, A., A. BAJER, J. M. BEHNKE, F. S. GILBERT AND E. SINSKI. 2004. Factors affecting the component community structure of haemoparasites in common voles (*Microtus arvalis*) in the Mazury Lake District region of Poland. *Parasitol. Res.* 2004. 92:270-284.
- PAZIEWSKA, A., M. BEDNARSKA, H. NIEWEGLOWSKI, G. KARBOWIAK AND A. BAJER. 2007. Distribution of *Cryptosporidium* and *Giardia* spp. in selected species of protected and game mammals from north-eastern Poland. *Ann Agric Environ Med.* 14:265-270.
- PEARSON, A. D. (1975). Epidemiology of Rodent Tularemia in Norway and Sweden. *Ecological Bulletins*, 19, 99–111.
- PELLÉRDY, L. R. (1974). *Coccidia and Coccidiosis* (2nd Edition). Verlag Paul Parey.
- PENNYCOTT, T. W., R. N. CINDERREY, A. PARK, H. A. MATHER AND G. FOSTER. 2002. *Salmonella enterica* subspecies *Enterica* serotype Typhimurium and *Escherichia coli* O86 in wild birds at two garden sites in south-west Scotland. *Vet Record.* 151(19):563-7.
- PERCIVAL, S. L. AND D. W. WILLIAMS. 2014. Mycobacterium. In *Microbiology of Waterborne Diseases*. Ch. 9. Elsevier Ltd. 2014 pp. 177-207.
- PEREC-MATYSIAK, A., K. BUNKOWSKA-GAWLIK, M. KVEC, S. BOHUMIL, J. HILDEBRAND AND K. LESNIAŃSKA. 2015. Diversity of *Enterocytozoon bineusi* genotypes among small rodents in southwestern Poland. *Veterinary Parasitology.* 214(2015) 242-246.
- PESTEREV, P. N. AND O. V. STADUKHIN. 1987. Basic carriers of *Trichophyton mentagrophytes* var. *Gypseum* in the Sverdlovsk region. *J. Hyg. Epidemiol. Microbiol. Immunol.* 1987. 31(3): 259-67.
- PETERSEN, J. M., MEAD, P. S., & SCHRIEFER, M. E. (2009). *Francisella tularensis*: An arthropod-borne pathogen. *Veterinary Research*, 40(2).
- PIECHOCKI, R. (1977). Ökologische todesursachenforschung am Elbebiber (*Castor fiber albicus*). *Beitr. Jagd-Wildforsch*, 10, 332–341.
- PLATT-SAMORAJ, A., K. SYCZYLO, A. BANCERZ-KISIEL, A. SCZERBA-TUREK, A. GIZEJEWSKA AND W. SZWEDA. 2015. *Yersinia Enterocolitica* strain isolated from beavers (*Castor fiber*). *Polish J. Vet. Sci.* 18(2) 449-451.
- POKORNY, I., KNUTH, D., TEUBNER, J., TRUBNER, J. (2014). Die wissenschaftliche Belegsammlung zum Biber (*Castor fiber*) des Naturkundemuseums Potsdam. Tagungsband Ergebnisse der Nationalen Bibertagung in Dessau – Rosslau, Sachsen – Anhalt, Dessau, s. 142–146.
- POLACK, B. AND K. T. ADJOU. 2019. Giardiasis in domestic mammals: clinical importance and public health consequence. VIIth International Giardia and Cryptosporidium Conference. Rouen, France.

- POSAUTZ, A., R. PARZ-GOLLNER, G. HOLZLER, L. SCHWAIGER AND A. KUBBER-HEISS. 2015. *Echinococcus multilocularis* in the beaver (*Castor fiber*) in eastern Austria. Proc. Int. Conf. Dis. Zoo Wild Anim. 2015. 199.
- POUNDER, K. C., M. BEGON, T. SIRONEN, H. HENTTONEN, P. C. WATTS, L. VOUTILAINEN, O. VAPALAHTI, B. KLEMPA, A. R. FOOKS, AND L. M. MCELHINNEY. 2013. Novel Hantavirus in Wildlife, United Kingdom. Emerg Infect Dis 19: 673-675.
- POZIO, E. 2000. Factors affecting the flow among domestic, synanthropic and sylvatic cycles of *Trichinella*. Veterinary Parasitology. 93(2000):241-262.
- POZIO, E. 2016. *Trichinella pseudospiralis* an elusive nematode. Veterinary Parasitology. 231(2016):97-101.
- POZIO, E. 2016b. Adaptation of *Trichinella* spp. for survival in cold climates. Food and Waterborne Parasites. 4(2016): 4-12.
- POZIO, E. 2019. *Trichinella* and trichinellosis in Europe. Veterinarski Glasnik. 73(2):65-84.
- POZIO, E. 2020. Scientific achievements of the last 60 years. From a single to a multispecies concept of the genus *Trichinella*. Veterinary Parasitology. Article in Press.
- POZIO, E., L. RINALDI, G. MARUCCI, V. MUSELLA, F. GALATI, G. CRINGOLI, P. BOIREAU AND G. LA ROSA. 2009. Hosts and habitats of *Trichinella spiralis* and *Trichinella britovi* in Europe. Int. J. Parasitol. 39:71-79.
- PRATAMA, R., D. SCHNEIDER, T. BOER AND R. DANIEL. 2019. First insights into bacterial gastrointestinal tract communities of the Eurasian Beaver (*Castor fiber*). Frontiers in Microbiology. 10:1646.
- PROMED INTERNATIONAL SOCIETY FOR INFECTIOUS DISEASES. (2020A). CORONAVIRUS DISEASE 2019 UPDATE (135): NETHERLANDS (NORTH BRABANT) ANIMAL, FARMED MINK. <https://promedmail.org/promed-post/?id=20200427.7272289>
- PROMED INTERNATIONAL SOCIETY FOR INFECTIOUS DISEASES. (2020B). CORONAVIRUS DISEASE 2019 UPDATE (58): BELGIUM, CAT, CLINICAL CASE, REQUEST FOR INFORMATION. <https://promedmail.org/promed-post/?id=20200327.7151215>
- QUINN, P. J., B. K. MARKEY, F. C. LEONARD, E. S. FITZPATRICK, S. FANNING AND P. J. HARTIGAN. 2011. Veterinary Microbiology and Microbial Disease. Chapter 15. Pp. 188-196. 2<sup>nd</sup> Edition. Blackwell Publishing Limited.
- RANQUE, S., B. FAUGERE, E. POZIO, G. LA ROSA, A. TAMBURRINI, J-F. PELLISSIER AND P. BROUQUI. 2000. *Trichinella pseudospiralis* outbreak in France. Emerging Infectious Diseases., 6(5): 543-547.
- REINTJES, R., DEDUSHAJ, I., GJINI, A., JORGENSEN, T. R., COTTER, B., LIEFTUCHT, A., D'ANCONA, F., DENNIS, D. T., KOSOY, M. A., MULLIQI-OSMANI, G., GRUNOW, R., KALAVESHI, A., & GASHI, L. (2002). Tularemia outbreak investigation in Kosovo: Case control and environmental studies. Emerging Infectious Diseases, 8(1), 69–73.

- RIDEOUT, B.A., A. W. SAINSBURY AND P. J. HUDSON. 2017. Which parasites should we be most concerned about in wildlife translocations? *EcoHealth*, 14, S42-S46.
- ROAD TRAFFIC STATISTICS - DEPARTMENT FOR TRANSPORT. (2018). <https://roadtraffic.dft.gov.uk/#13/56.5749/-0.3440/basemap-countpoints>.
- ROBERTS, H. 2012. What is the risk of introducing *Echinococcus multilocularis* to the UK wildlife population by importing beavers which subsequently escape or are released? Qualitative Risk Assessment. DEFRA.
- ROBERTS, J. A., P. CUMBERLAND, P. N. SOCKETT, J. WHEELER, L. C. RODRIGUES, D. SETHI AND P. J. RODERICK. 2003. The study of infectious disease in England: socio-economic impact. *Epidemiol. Infect.* 130:1-11.
- ROBERTSON, L. AND B. GJERDE. 2001. *Cryptosporidium* oocysts and *Giardia* cysts in raw waters in Norway. *Scand J Public Health*. 29:200-207.
- ROBERTSON, L. J., A. T. CAMPBELL AND H. V. SMITH. 1992. Survival of *Cryptosporidium parvum* oocysts under various environmental pressures. *Applied and Environmental Microbiology*. 58(11):3494-35.
- ROBERTSON, L., J. LASSEN, M. TRYLAND AND R. DAVIDSON. 2012. Norwegian Scientific Committee for Food Safety (VKM). Final assessment of risk of introduction of *Echinococcus multilocularis* to mainland Norway.
- ROJAS-ESPINOSA, O. AND M. LOVIC. 2001. *Mycobacterium lepraemurium* infections in domestic and wild animals. *Revue Scientifique et Technique (International Office of Epizootics)*, 20(1): 219-251.
- ROMIG, T., P. DEPLAZES, D. JENKINS, P. GIRAUDOUX, A. MASSOLO, P. CRAIG AND M. DE LA RUE. 2017. Ecology and life cycle patterns of *Echinococcus* species. *Advances in Parasitology*. 95:213-314.
- ROSELL, F., & KVINLAUG, J. K. (1998). Methods for live-trapping beaver (*Castor* spp.). *Fauna Norvegica*, 19, 1–28.
- ROSELL, F., O. ROSEF AND H. PARKER. 2001. Investigations of waterborne pathogens in Eurasian beaver (*Castor fiber*) from Telemark county, Norway. *Acta Vet. Scand.* 42(4): 479-482.
- ROSSOW, H., SISSONEN, S., KOSKELA, K. A., KINNUNEN, P. M., HEMMILÄ, H., NIEMIMAA, J., HUITU, O., KUUSI, M., VAPALAHTI, O., HENTTONEN, H., & NIKKARI, S. (2014). Detection of *Francisella tularensis* in Voles in Finland. *Vector-Borne and Zoonotic Diseases*, 14(3), 193–198.
- ROTHAN, H. A., & BYRAREDDY, S. N. (2020). The epidemiology and pathogenesis of coronavirus disease (COVID-19) outbreak. *Journal of Autoimmunity*, February, 102433.
- ROZYCKI, M., E. BILSKA-ZAJAC, M. KOCHANOWSKI, K. GRADZIEL-KRUKOWSKA, J. ZDYBEL, J. KARAMON, J. WISNIEWSKI AND T. CENCEK. 2020. First case of *Trichinella spiralis* infection in beavers (*Castor fiber*) in Poland Europe. *Parasites and Wildlife*. 11(2020):46-49.
- ROZYCKI, M., E. BILSKA-ZAJAK, M. KOCHANOWSKI, K. GRADZIEL-KRUKOWSKA, J. ZDYBEL, J. *Trichinella spiralis* infection in beavers (*Castor fiber*) in Poland. *Parasites and Wildlife*. 11(2020) 46-49.

- RUIZ-FONS, F. 2012. *Coxiella burnetii* infection. In G. Gavier-Widen, P.D. Duff & A. Meredith (Eds), *Infectious Diseases of Wild Mammals and Birds in Europe* (First edition pp. 409-412). Blackwell Publishing Ltd., UK.
- RYAN, U. AND S. M. CACCIO. 2013. Zoonotic potential of *Giardia*. *Int J for Parasitol.* 43(2013):943-956.
- RYAN, U., A. ZAHEDI AND A. PAPARINI. 2016. *Cryptosporidium* in humans and animals – A one health approach to prophylaxis. *Parasite Immunology.* 39:535-547.
- RYAN, U., R. FAYER AND L. XIAO. 2014. *Cryptosporidium* species in humans and animals: Current understanding and research needs. *Parasitology* 141:1667-1685.
- SAGER, H., D. KONJEVIC, M. GRUBESIC, Z. JANICKI, K. SEVERIN AND R. Beck R. 2005. *Stichorchiis subtriquetrus* in European beaver from Croatia: First report. *Eur J Wildl Res* 51:63–64.
- SAINSBURY, A.W. AND R. J. Vaughan-Higgins. 2012. Analysing disease risks associated with translocations. *Conservation Biology*, 26: 442-452.
- SALVADOR, A. R., E. GUIVIER, A. XUEREB, Y. CHAVAL, P. CADET, M-L POULLE, T. SIRONEN, L. VOUTILAINEN, H. HENTTONEN, J-F. COSSON AND N. CHARBONNEL. 2011. Concomitant influence of helminth infection and landscape on the distribution of Puumala hantavirus in its reservoir, *Myodes glareolus*. *Microbiology* 2011, 11:30.
- SAVELJEV, A. P., BATBAYAR, N., BOLDBAATAR, S., & DASHBIAMBA, B. (2016). Self-eating in beavers — trophic opportunism or reaction on stress? Extreme case from Mongolia. *Russian Journal of Theriology*, 15(1), 68–74.
- SCHEELE, B. C., F. PASMANS, L. F. SKERRATT, L. BERGER, A. MARTEL, W. BEUKEMA, A. A. ACEVEDO AND S. CANESSA. S 2019. Amphibian fungal panzootic causes catastrophic and ongoing loss of biodiversity. *Science* 363: 1459-1463
- SCHMIDT, R. E. (1995). Protozoal Diseases of Rabbits and Rodents. *Seminars in Avian and Exotic Pet Medicine*, 4(3), 126–130.
- SCHMIDT-CHANASIT, J., S. ESSBAUER, R. PETRAITYTE, K. YOSHIMATSU, K. TACKMANN, F. J. CONRATHS, K. SASNAUSKAS, J. ARIKAWA, A. THOMAS, M. PFEFFER, J. J. SCHARNINGHAUSEN, W. SPLETTSTOESSER, M. WENK, G. HECKEL, ANDR. G. ULRICH. 2010. Extensive host sharing of central European Tula virus. *J Virol* 84: 459-474.
- SCHOEB, T. R. 2000. Respiratory diseases of rodents. *Veterinary Clinics of North America: Exotic Animal Practice.* 3(2). 481-496
- SCHREIBER, P. W., L. ACETO, R. KORACH, N. MARREROS, M-P. RYSER-DEGIORGIS AND H. F. GUNTARD (2015). “Cluster of Leptospirosis acquired through river surfing in Switzerland.” *Open Forum Infect Dis*, 2 (3), ofv102
- SCHRENZEL, M. D., C. L. WITTE, J. BAHL, T. A. TUCKER, N. FABIAN, H. GREGER, C. HOLLIS, G. HSIA, E. SILTAMAKI AND B. A. RIDEOUT. Genetic characterisation and epidemiology of *Helicobacters* in non-domestic animals. *Helicobacter.* 15(2):126-142.

- SCHULZE, C., HEUNER, K., MYRTENNÄS, K., KARLSSON, E., JACOB, D., KUTZER, P., GROßE, K., FORSMAN, M., & GRUNOW, R. (2016). High and novel genetic diversity of *Francisella tularensis* in Germany and indication of environmental persistence. *Epidemiology and Infection*, 144(14), 3025–3036.
- SCHULZE, C., P. KUTZER, N. WINTERHOFF, A. ENGELHARDT, S. BILK AND J. TEUBNER. 2015. Isolation and antimicrobial susceptibility of *Streptococcus castoreus* isolated from carcasses of European beavers (*Castor fiber*) in Germany. *Berliner und Munchener Tierarztliche Wochenschrift*. 128, 394-396.
- SCOTTISH NATIONAL HERITAGE. (1998). Re-introduction of the European Beaver to Scotland. A Public Consultation.
- SEGLINA, Z. AND E. BAKASEJEVS. 2015. New finding of *Trichinella britovi* in a European beaver (*Castor fiber*) in Latvia. *Parasitol Res*. 114:3171-3173.
- SEGLINA, Z., E. BAKASEJEVS, G. DEKSNE, V. SPUNGIS AND M. KURJUSINA. 2015. New finding of *Trichinella britovi* in a European beaver (*Castor fiber*) in Latvia. *Parasitol. Res*. 2015 Aug 114(8):3171-3
- SHANSON, D. C. 1989. Opportunistic infections. In *Microbiology in Clinical Practice*. 2<sup>nd</sup> edition. Butterworth Heinemann. Pp. 151-167.
- SHEARER, K. E., M. J. HARTE, D. OJKIC, J. DELAY AND D. CAMPBELL (2014). "Detection of *Leptospira* spp. in wildlife reservoirs in Ontario through comparison of immunohistochemical and polymerase chain reaction genotyping methods." *Can Vet J*. 2014 Mar 55(3): 240-24.
- SHEN, B., YUAN, Y., CHENG, J., PAN, M., XIA, N., ZHANG, W., WANG, Y., ZHOU, Y., & ZHAO, J. (2016). Activation of chronic toxoplasmosis by transportation stress in a mouse model. *Oncotarget*, 7(52), 87351–87360.
- SHI, J., WEN, Z., ZHONG, G., YANG, H., WANG, C., LIU, R., HE, X., SHUAI, L., SUN, Z., ZHAO, Y., LIANG, L., CUI, P., WANG, J., ZHANG, X., GUAN, Y., CHEN, H., & BU, Z. (2020). Susceptibility of ferrets, cats, dogs, and different domestic animals to SARS-coronavirus-2. *BioRxiv*.
- SHI, Z., & HU, Z. (2008). A review of studies on animal reservoirs of the SARS coronavirus. *Virus Research*, 133(1), 74–87.
- SHIMALOV, V. V. AND V. T. SHIMALOV. 1999. Findings of *Fasciola hepatica* Linnaeus 1758 in Belorussian Polesye. *Parasitology Research*. 86(4) 342-342.
- SIKOROWSKI K., T. NIEMIEC, E. CZERNIAWSKA-PIATKOWSKA, M. MAKARSKI, B. J. BARTYZEL, S. PAŚKO AND P. KOCZOŃ. 2016. Biology and parasitology of European beaver (*Castor fiber*) – selected issues. *Folia Pomeranae Univ Technol Stetin Agric Aliment Piscaria Zootech* 328:203–210.
- SIMPSON, V. 2008. Wildlife as reservoirs of zoonotic diseases in the UK. *In Practice*. 30: 486-494.
- SIMPSON, V. R., A. J. TOMLINSON, K. STEVENSON, J. A. MCLUCKIE, J. BENAVIDES AND M. P. DAGLEISH. 2016. A post-mortem study of respiratory disease in small mustelids in south-west England. *BMC Veterinary Research*. 12(72):1-13.



- SIMPSON, V. R., J. HARGREAVES, H. M. BUTLER, N. J. DAVISON AND D. J. EVEREST. 2013. Causes of mortality and pathological lesions observed post-mortem in red squirrels (*Sciurus vulgaris*) in Great Britain. BMC Veterinary Research. 9(229):1-13.
- SIMPSON, V. R., J. HARGREAVES, R. J. BIRTLES, H. MARSDEN AND D. L. WILLIAMS. 2008. Tyzzer's disease in a Eurasian otter (*Lutra lutra*) in Scotland. The Veterinary Record, 163: 539-543.
- SIMPSON, V. R., N. J. DAVISON AND M. P. DAGLEISH. 2019. Causes of mortality and lesions observed post-mortem in European moles (*Talpa europaea*) in Cornwall, south-west England. J. Comp. Path. 167: 18-25.
- SKARIAH, S., MCINTYRE, M. K., & MORDUE, D. G. (2010). *Toxoplasma gondii*: determinants of tachyzoite to bradyzoite conversion. Parasitology Research, 107(2), 253–260.
- SMERDON, W. J., T. NICHOLS, R. M. CHALMERS, H. HEINE AND M. REACHER. 2003. Foot and Mouth disease in livestock and reduced cryptosporidiosis in humans, England and Wales. Emerg Inf Dis. 9(1):22-28.
- SMITH, D., & FRENKEL, J. K. (1995). Prevalence of antibodies to *Toxoplasma gondii* in wild mammals of Missouri and East Central Kansas: Biologic and ecologic considerations of transmission. Journal of Wildlife Diseases, 31(1), 15–21.
- SPECK, S. 2012. *Campylobacter* infections. In Gavier-Widen, G., Duff, P.D. & Meredith, A. (Eds), *Infectious Diseases of Wild Mammals and Birds in Europe* (First edition pp. 398-401). Blackwell Publishing Ltd., UK.
- SPECK, S. 2012. *Streptococcus* infections. In Gavier-Widen, G., Duff, P.D. & Meredith, A. (Eds), *Infectious Diseases of Wild Mammals and Birds in Europe* (First edition pp. 435-438). Blackwell Publishing Ltd., UK.
- SPECK, S. 2012c. *Staphylococcus* infections. In Gavier-Widen, G., Duff, P.D. & Meredith, A. (Eds), *Infectious Diseases of Wild Mammals and Birds in Europe* (First edition pp. 435-438). Blackwell Publishing Ltd., UK
- SPECK, S. AND J. P. DUFF. 2012. *Chlamydiaceae* Infections, chapter 26. In G. Gavier-Widen, P.D. Duff & A. Meredith (Eds), *Infectious Diseases of Wild Mammals and Birds in Europe* (First edition pp. 345-362). Blackwell Publishing Ltd., UK.
- SROKA, J., Z. GIZEJEWSKI, A. WOJCIK-FATLA, K. STOJECKI, E. BILSKA-ZAJAC, J. DUTKIEWICZ, T. CENCEK, J. KARAMON, V. ZAJAC, P. KUSYK, J. DABROWSKA AND M. KOCHANOWSKI. 2015. Potential role of beavers (*Castor fiber*) in contamination of water in the Masurian Lake District (north-eastern Poland) with protozoan parasites *Cryptosporidium* spp. and *Giardia duodenalis*. Bull Vet Inst Pulawy. 59,215-228.
- STAPLES, J. E., KUBOTA, K. A., CHALCRAFT, L. G., MEAD, P. S., & PETERSEN, J. M. (2006). Epidemiologic and molecular analysis of human tularemia, United States, 1964-2004. Emerging Infectious Diseases, 12(7), 1113–1118.
- STEENKAMP, G., VENTER, L., CROSSLEY, D., & BUSS, P. (2009). Mandibular Incisor Apicoectomy in a Canadian Beaver. Journal of Veterinary Dentistry, 26(3), 164–167.

- STEFEN, C. 2018. Causes of death of beavers (*Castor fiber*) from Eastern Germany and observations on parasites, skeletal diseases and tooth anomalies – a long term analysis. *Mammal Research*. 64, 279-288(2019).
- STEPANOVA, E. V., KONDRASHIN, A. V., SERGIEV, V. P., MOROZOVA, L. F., TURBABINA, N. A., MAKSIMOVA, M. S., BRAZHNIKOV, A. I., SHEVCHENKO, S. B., & MOROZOV, E. N. (2017). Significance of chronic toxoplasmosis in epidemiology of road traffic accidents in Russian Federation. *PLoS ONE*, 12(9), 1–9.
- STING, R., RUNGE, M., EISENBERG, T., BRAUNE, S., MULLER, W., & OTTO, P. (2013). Comparison of bacterial culture and polymerase chain reaction (PCR) for the detection of *F. tularensis* subsp. *holarctica* in wild animals. *Berl Munch Tierarztl Wochenschr*, 126, 285–290.
- STUART, B. P., W. A. CROWELL, W. V. ADAMS AND D. T. MORROW (1978). "Spontaneous renal disease in beaver in Louisiana." *J. Wild Dis* 14 250-253.
- STURDEE, A. P., A. T. BODLEY-TICKELL, A. ARCHER AND R. M. CHALMERS. 2003. Long term study of *Cryptosporidium* prevalence on a lowland farm in the United Kingdom. *Veterinary Parasitology*. 116(2003):97-113.
- SUZUKI, Y., ORELLANA, M. A., SCHREIBER, R. D., & REMINGTON, J. S. (1988). Interferon- $\gamma$ : The major mediator of resistance against *Toxoplasma gondii*. *Science*, 240(4851), 516–518.
- SWAIN, U. G., F. F. GILBERT AND J. D. ROBINETTE. 1988. Heart rates in the captive, free-ranging beaver. *Comp. Biochem. Physiol.* 91A 3, 431-435.
- TAKEUCHI-STORM, N., L. WOOLSEY, P. JENSEN, B. FRIEDENSBORG, C. PIPPER AND C. KAPEL. 2015. Predictors of *Echinococcus multilocularis* in definitive and intermediate hosts. A meta-analysis approach. *Journal of Parasitology*. 101(3):297-303.
- TAN, Y., N. LI, M. SONG, D. M. ROELLIG, Y. FENG AND L. XIAO. 2016. Development of a multilocus sequence typing tool for high-resolution subtyping and genetic structure characterisation of *Cryptosporidium ubiquitum*. *Infection, Genetics and Evolution*. 45(2016):256-261.
- TÄRNVIK, A., & CHU, M. (2007). New approaches to diagnosis and therapy of tularemia. *Annals of the New York Academy of Sciences*, 1105(1), 378–404.
- TÄRNVIK, A., SANDSTRÖM, G., & SJÖSTEDT, A. (1996). Epidemiological analysis of tularemia in Sweden 1931-1993. *FEMS Immunology and Medical Microbiology*, 13(3), 201–204.
- TAYLOR, B. D., & GOLDINGAY, R. L. (2010). Roads and wildlife: Impacts, mitigation and implications for wildlife management in Australia. *Wildlife Research*, 37(4), 320–331. <https://doi.org/10.1071/WR09171>
- TAYLOR, M.A., R. L. COOP AND R. L. WALL. 2007. *Veterinary Parasitology* (3<sup>rd</sup> Edition). Blackwell Publishing Ltd., UK.
- TEIXEIRA, C. P., DE AZEVEDO, C. S., MENDL, M., CIPRESTE, C. F., & YOUNG, R. J. (2006). Revisiting translocation and reintroduction programmes: the importance of considering stress. *Animal Behaviour*, 73(1), 1–13.

- TENTER, A. M., HECKEROTH, A. R., & WEISS, L. M. (2000). *Toxoplasma gondii*: From animals to humans. *International Journal for Parasitology*, 30(12–13), 1217–1258.
- THELAUS, J., ANDERSSON, A., BROMAN, T., BÄCKMAN, S., GRANBERG, M., KARLSSON, L., KUOPPA, K., LARSSON, E., LUNDMARK, E., LUNDSTRÖM, J. O., MATHISEN, P., NÄSLUND, J., SCHÄFER, M., WAHAB, T., & FORSMAN, M. (2014). *Francisella tularensis* Subspecies *holarctica* Occurs in Swedish Mosquitoes, Persists Through the Developmental Stages of Laboratory-Infected Mosquitoes and Is Transmissible During Blood Feeding. *Microbial Ecology*, 67(1), 96–107.
- THOMASON, A. G., M. BEGON, J. E. BRADLEY, S. PATERSON, ANDJ. A. JACKSON. 2017. Endemic Hantavirus in Field Voles, Northern England. *Emerg Infect Dis* 23: 1033-1035.
- THOMPSON, R. C. A. AND A. ASH. 2019. Molecular epidemiology of *Giardia* and *Cryptosporidium* infections. What's New? *Infection, Genetics and Evolution*. 75(11):1-5.
- THOMPSON, R. C. A., M. E. OLSON, G. ZHU, S. ENOMOTO, M. S. ABRAHAMSEN AND N. S. HIJAWI. 2005. *Cryptosporidium* and Cryptosporidiosis. In *Advances in Parasitology*. Vol 59: 78-40. Elsevier.
- TITTENSOR, A. M. (1975). Red Squirrel. In *Forestry Commission Forest Record* 101.
- TITTENSOR, A. M. (1977). Red Squirrel. Grey Squirrel. In G. B. Corbet & H. N. Southern (Eds.), *The Handbook of British Mammals* (2<sup>nd</sup> Edition, pp. 153–172). Blackwell Scientific Publications.
- TORGERSON, P. AND P. CRAIG. 2009. Risk assessment of importation of dogs infected with *Echinococcus multilocularis* into the UK. *Veterinary Record*. 165:366-368.
- TOTH, B., A. FROST AND H. ROBERTS. DEFRA. 2010. The change in likelihood of *Echinococcus multilocularis* (Alveolar echinococcosis) introduction into the United Kingdom as a consequence of adopting existing harmonised Community rules for the non-commercial movements of pet animals. DEFRA.
- TREFANCOVA, A., A. MACOVA, J. KVICEROVA. 2019. Isosporan oocysts in the faeces of bank voles (*Myodes glareolus*). Real parasites or pseudoparasites? *Protist*. 17:104-12.
- TSUI, C. K-M., R. MILLER, M. UYAGUARI-DIAZ, P. TANG, C. CHAUVE, W. HSAIO, J. ISAAC-RENTON AND N. PRYSTAJECKY. 2018. Beaver Fever: Whole genome characterisation of waterborne outbreak and sporadic isolates to study the zoonotic transmission of giardiasis. *Clinical Science and Epidemiology*. 3(2):1-17.
- UMHANG, G., J. LAHOREAU, V. HORMAZ, J. BOUCHER, A. GUENON, D. MONTANGE AND F. BOUE. 2016. Surveillance and management of *Echinococcus multilocularis* in a wildlife park. *Parasitology International*. 65(3):245-250.
- USDA ANIMAL AND PLANT HEALTH INSPECTION SERVICE. (2020). Confirmation of COVID-19 in Two Pet Cats in New York. <https://content.govdelivery.com/accounts/USDAAPHIS/bulletins/287d882>
- USEPA (UNITED STATES ENVIRONMENTAL PROTECTION AGENCY). 1999. *Giardia Drinking Water Health Advisory*. Office of Water, Washington.

- VAHERI, A., O. VAPALAHTI, ANDA. PLYUSNIN. 2008. How to diagnose hantavirus infections and detect them in rodents and insectivores. *Rev Med Virol* 18: 277-288.
- VAN KEULEN, H., P. T. MACECHKO, S. WADE, S. SCHAAF, P. M. WALLIS AND S. L. ERLANDSEN. 2002. Presence of human *Giardia* in domestic, farm and wild animals, and environmental samples suggests a zoonotic potential for giardiasis. *Veterinary Parasitol.* 108(2002):97-100.
- VAN LANGEVELDE, F., VAN DOOREMALEN, C., & JAARSMA, C. F. (2009). Traffic mortality and the role of minor roads. *Journal of Environmental Management*, 90(1), 660–667.
- VEIT, P., B. BILGER, V. SCHAD, J. SCHAFER, W. FRANKF AND A. LUCIUS. 1995. Influence of environmental factors on the infectivity of *Echinococcus multilocularis* eggs. *Parasitology.* 110(1):79-86.
- VENGUST, G., A. BIDOVEC, A. VERGLES-RATAJ AND D. ZELE. 2009. *Stichorchis subtriquetrus* in two beavers (*Castor fiber*) from Slovenia. *Helminthologia* 46:59–61.
- VIGGERS, K. L., D. LINDENMAYER AND D. M. SPRATT. 1993. The importance of disease reintroduction programmes. *Wildlife Res.* 1993. 20 (5)
- VIKØREN, T., THARALDSEN, J., FREDRIKSEN, B., & HANDELAND, K. (2004). Prevalence of *Toxoplasma gondii* antibodies in wild red deer, roe deer, moose, and reindeer from Norway. *Veterinary Parasitology*, 120(3), 159–169.
- VIRBICKAS, T., STAKENAS, S., & STEPONENAS, A. (2015). Impact of beaver dams on abundance and distribution of anadromous salmonids in two lowland streams in Lithuania. *PLoS ONE*, 10(4), 1–12. <https://doi.org/10.1371/journal.pone.0123107>
- VISAVET. 2020. European Reference Laboratory for Bovine Tuberculosis. <https://www.visavet.es/bovinetuberculosis/mycodb.eu.php>
- VOUTILAINEN, L., T. SIRONEN, E. TONTERI, A. T. BÄCK, M. RAZZAUTI, M. KARLSSON, M. WAHLSTRÖM, J. NIEMIMAA, H. HENTTONEN, ANDÅ. LUNDKVIST. 2015. Life-long shedding of Puumala hantavirus in wild bank voles (*Myodes glareolus*). *J Gen Virol* 96: 1238-1247.
- VYAS, A., KIM, S. K., & SAPOLSKY, R. M. (2007). The effects of toxoplasma infection on rodent behavior are dependent on dose of the stimulus. *Neuroscience*, 148(2), 342–348.
- VÝROSTEKOVÁ, V. (1993). Transstadial transmission of *Francisella tularensis* in the tick, *Ixodes ricinus*, infected during the larval stage. *Ceskoslovenska Epidemiologie, Mikrobiologie, Imunologie*, 42(2), 71–75.
- WAHLSTROM, H., E. TYSEN, E. O. ENGVALL, B. BRANDSTROM, E. ERIKSSON, T. MORNER AND I. VAGSHOLM. 2003. Survey of *Campylobacter* species, VTEC O157 and *Salmonella* species in Swedish wildlife. *Veterinary Record.* 153:74-80.
- WAHLSTROM, H., H. ENEMARK, R. DAVIDSON AND A. OKSANEN. 2015. Present status, actions taken and future considerations due to the findings of *E. multilocularis* in two Scandinavian countries. *Veterinary Parasitology.* 213(4):172-181.

- WANG, L. F., & CRAMERI, G. (2014). Emerging zoonotic viral diseases. *OIE Revue Scientifique et Technique*, 33(2), 569–581.
- WANG, Q., B. J. CHANG AND T. V. RILEY. 2010. *Erysipelothrix rhusiopathiae*. *Veterinary Microbiology*. 140: 405-417.
- WASSENAAR, T. M., & ZOU, Y. (2020). 2019\_nCoV/SARS-CoV-2: rapid classification of betacoronaviruses and identification of Traditional Chinese Medicine as potential origin of zoonotic coronaviruses. *Letters in Applied Microbiology*, 1–7.
- WEBSTER, J. P., ANDD. W. MACDONALD. 1995b. Parasites of wild brown rats (*Rattus norvegicus*) on UK farms. *Parasitology* 111 (Pt 3): 247-255.
- WEBSTER, J. P., BRUNTON, C. F. A., & MACDONALD, D. W. (1994). Effect of *Toxoplasma Gondii* Upon Neophobic Behaviour in Wild Brown Rats, *Rattus Norvegicus*. *Parasitology*, 109(1), 37–43.
- WEBSTER, P. AND C. M. O. KAPEL. 2005. Studies on vertical transmission of *Trichinella* spp. in experimentally infected ferrets (*Mustela putorius furo*), foxes (*Vulpes vulpes*), pigs, guinea pigs and mice. *Veterinary Parasitology*. 130(2205) 255-262.
- WEISS, L. M., LAPLACE, D., TARKVORIAN, D., TANOWITZ, H. B., & WITTNER, M. (1996). The Association of the Stress Response and *Toxoplasma gondii* Bradyzoite Development. *The Journal of Eukaryotic Microbiology*, 43(5), 120S-120S.
- WEISSENBOCK, H. 2012. Borna Disease Virus. In Gavier-Widen, G., Duff, P.D. & Meredith, A. (Eds), *Infectious Diseases of Wild Mammals and Birds in Europe* (First edition pp. 251-252). Blackwell Publishing Ltd., UK.
- WEISSENBOCK, H. 2012. *Lawsonia Intracellularis* infections. In G. Gavier-Widen, P.D. Duff & A. Meredith (Eds), *Infectious Diseases of Wild Mammals and Birds in Europe* (First edition pp. 345-362). Blackwell Publishing Ltd., UK.
- WEN, H., L. VUITTON, T. TUXUN, J. LI, D. VUITTON, W. ZHANG AND M. MCMANUS. 2019. Echinococcosis: Advances in the 21<sup>st</sup> century. *Clinical Microbiology Reviews*. 32(2). American Society for Microbiology.
- WHARY, M. T. AND J. G. FOX. 2004. Natural and Experimental Helicobacter Infection. *Comparative Medicine*. 54(2):128-158.
- WHARY, M. T., BAUMGARTH, N., FOX, J. G., & BARTHOLD, S. W. (2015). Biology and Diseases of Mice. In J. G. Fox, L. C. Anderson, G. M. Otto, K. R. Pritchett-Corning, & M. T. Whary (Eds.), *Laboratory Animal Medicine* (Third Edit, pp. 43–148).
- WHO (WORLD HEALTH ORGANISATION). 2018. WHO Expert Consultation on rabies. WHO Press, Switzerland.
- WHO (WORLD HEALTH ORGANISATION). 2020. <https://www.sciencedirect.com/science/article/abs/pii/S1084952115000336>. Page visited 27/05/2020.

- WHO (WORLD HEALTH ORGANISATION). 2020. <https://www.who.int/news-room/fact-sheets/detail/echinococcosis>. Site visited 17/05/2020.
- WITKOWSKI, P. T., C. C. PERLEY, R. L. BROCATO, J. W. HOOPER, C. JÜRGENSEN, J. D. SCHULZKE, D. H. KRÜGER, ANDR. BÜCKER. 2017. Gastrointestinal Tract as Entry Route for Hantavirus Infection. *Front Microbiol* 8: 1721.
- WOBESER, G. 2007. *Disease in wild animals: Investigation and Management*. 2<sup>nd</sup> Ed. Springer - Verlag Berlin, Heidelberg.
- WODECKA, B., & SKOTARCZAK, B. (2016). Identification of host blood-meal sources and *Borrelia* in field-collected *Ixodes ricinus* ticks in north-western Poland. *Annals of Agricultural and Environmental Medicine*, 23(1), 59–63.
- WOLL, D., C. KARNATH AND M. PFEIFFER (2012). "Characterisation of *Leptospira* spp. from beavers found dead in south-west Germany." *Vet Microbiology* 158 (2012) 232-234.
- WOOLSEY, I., P. JENSES, P. DEPLAZES AND C. KAPEL. 2016. Peroral *Echinococcus multilocularis* egg inoculation in *Myodes glareolus*, *Mesocricetus auratus* and *Mus musculus*. *Int. J. for Parasitol: Parasites and Wildlife*. 5(2):158-163.
- WORLD HEALTH ORGANIZATION. (2020). Coronavirus disease 2019 (COVID-19) - Situation Report 77. World Health Organization. <https://doi.org/10.1001/jama.2020.2633>
- WORLD HEALTH ORGANIZATION. 2007. WHO guidelines on tularaemia: epidemic and pandemic alert and response. [https://doi.org/ISBN 978 92 4 154737 6](https://doi.org/ISBN%20978%2092%204%20154737%206).
- WORLD ORGANISATION FOR ANIMAL HEALTH (OIE). (2020). SARS-CoV-2/COVID-19, United States of America. [https://www.oie.int/wahis\\_2/public/wahid.php/Reviewreport/Review?page\\_refer=MapFullEventReport&reportid=34054](https://www.oie.int/wahis_2/public/wahid.php/Reviewreport/Review?page_refer=MapFullEventReport&reportid=34054)
- WORLD ORGANISATION FOR ANIMAL HEALTH (OIE). (2020). World Animal Health Information Database (WAHID). WAHIS Interface. [https://www.oie.int/wahis\\_2/public/wahid.php/Diseaseinformation/Diseasetimelines](https://www.oie.int/wahis_2/public/wahid.php/Diseaseinformation/Diseasetimelines)
- WU, D., TU, C., XIN, C., XUAN, H., MENG, Q., LIU, Y., YU, Y., GUAN, Y., JIANG, Y., YIN, X., CRAMERI, G., WANG, M., LI, C., LIU, S., LIAO, M., FENG, L., XIANG, H., SUN, J., CHEN, J., KONG, X. (2005). Civets Are Equally Susceptible to Experimental Infection by Two Different Severe Acute Respiratory Syndrome Coronavirus Isolates. *Journal of Virology*, 79(4), 2620–2625.
- XIAO, F., TANG, M., ZHENG, X., LIU, Y., LI, X., & SHAN, H. (2020). Evidence for Gastrointestinal Infection of SARS-CoV-2. *Gastroenterology*, February 2019, 1–13.
- YAKIMOFF, W. L. (1934). Die Biberkokzidiose. *Berliner Tierärztlicher Wochenschrift*, 50(294).
- YEO, C., KAUSHAL, S., & YEO, D. (2020). Enteric involvement of coronaviruses: is faecal–oral transmission of SARS-CoV-2 possible? *The Lancet Gastroenterology and Hepatology*, 5(4), 335–337.

- YERELI, K., BALCIOĞLU, I. C., & ÖZBILGIN, A. (2006). Is *Toxoplasma gondii* a potential risk for traffic accidents in Turkey? *Forensic Science International*, 163(1–2), 34–37.
- YTREHUS, B., & T. VIKOREN. 2012. *Borrelia* infections. In G. Gavier-Widen, P.D. Duff & A. Meredith (Eds), *Infectious Diseases of Wild Mammals and Birds in Europe* (First edition pp. 345-362). Blackwell Publishing Ltd., UK.
- ZHANG, Q., ZHANG, H., HUANG, K., YANG, Y., HUI, X., GAO, J., HE, X., LI, C., GONG, W., ZHANG, Y., PENG, C., GAO, X., CHEN, H., ZOU, Z., SHI, Z., & JIN, M. (2020). SARS-CoV-2 neutralizing serum antibodies in cats: a serological investigation. *BioRxiv*, 2020.04.01.021196.
- ZHANG, Y. Z. 2014. Discovery of hantaviruses in bats and insectivores and the evolution of the genus Hantavirus. *Virus Res* 187: 15-21.
- ZHENG, J. (2020). SARS-CoV-2: An Emerging Coronavirus that Causes a Global Threat. *International Journal of Biological Sciences*, 16(10), 1678–1685.
- ZIMMERMANN, P., A. FINN AND N. CURTIS. 2018. Does BCG vaccination protect against non-tuberculous mycobacterial infection? A systematic review and meta-analysis. *J Inf Dis* 2018:218 (679-687).

