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Risk Assessment of

Import of dogs and cats to Iceland

- with special attention to Guide-dogs

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CHAPTER 1. INTRODUCTION

In the current trend of globalisation, animal health measures have increasing importance to facilitate safe international trade of animals and animal products, while avoiding unnecessary impediments to trade. In light of this, the Agreement on the Application of Sanitary and Phytosanitary Measures (SPS Agreement) encourages the members of the World Trade Organization (WTO) to base their sanitary measures on international standards, guidelines and recommendations, where they exist.

The OIE is the WTO reference organisation for standards relating to animal health and zoonoses. The OIE publishes 2 codes (Terrestrial and Aquatic) and 2 manuals (Terrestrial and Aquatic) as the principle references for WTO members.

The Terrestrial Animal Health Code (TAHC) aims to assure the sanitary safety of international trade in terrestrial animals and their products. Chapter 2.1. Import risk analysis describes the following relevant concepts, which are discussed in more details in the OIE Handbook of Import Risk Analysis for Animals and Animal Products, vol. 1 (2004):

- Risk: means the likelihood of the occurrence and the likely magnitude of the biological and economic consequences of an adverse event or effect to animal or human health.
- Risk analysis: means the process composed of hazard identification, risk assessment, risk management and risk communication.
- Hazard identification: The hazard identification involves identifying the pathogenic agents which could potentially produce adverse consequences associated with the importation of a commodity.

  The hazards identified would be those appropriate to the species being imported, or from which the commodity is derived, and which may be present in the exporting country. It is then necessary to identify whether each hazard is already present in the importing country, and whether it is a notifiable disease or is subject to control or eradication in that country and to ensure that import measures are not more trade restrictive than those applied within the country.

- Risk assessment: means the evaluation of the likelihood and the biological and economic consequences of entry, exposure, establishment and spread of a hazard.
- Risk communication: means the interactive transmission and exchange of information and opinions throughout the risk analysis process concerning risk, risk-related factors and risk perceptions among risk assessors, risk managers, risk communicators, the general public and other interested parties.
- Risk management: means the process of identifying, selecting and implementing measures that can be applied to reduce the level of risk.

Background

Due to their relative geographical isolation, the Icelandic animal populations have so far been free from many transmissible diseases. Imports of live animals during the past centuries have, however, in several cases brought diseases with them, such as sheep scabies, Scrapie, and the so-called Karakul diseases: Maedi/Visna, Jaagsiekte and paratuberculosis. The more recent experiences in Iceland with the three diseases Maedi/Visna in sheep, Infectious Pyrexia and the disease caused by a “new” strain of the bacterium *Streptococcus equi* subsp. *zooepidemicus* (ST2309) in horses, clearly demonstrate the susceptibility and vulnerability of the native animal populations in Iceland. Strict regulations on the import of live animals and animal products have therefore been established to minimize the risk of introducing non-indigenous infectious animal disease agents to Iceland. Import bans or strict quarantine conditions are therefore still applied to animals and animal products. During the quarantine period, tests to detect the occurrence of

various potentially pathogenetic organisms are carried out, and several examples of such detections have been published by Icelandic authorities and researchers, as can be found in the list of references in this report.

International agreements on trade issues and the world-wide expansion of movement of people and products, as well as the economic and social benefits of tourism and international cooperation, however, have led to regular feasibility checks of the protective legislative measures in other countries with somewhat similar geographical conditions as Iceland, such as Australia and New Zealand.

Imports of dogs and cats to Iceland

In the area of import of dogs and cats to Iceland, a Risk Analysis was requested by the Ministry of Agriculture in 2001 from a working group established on behalf of the CVO. The group reported in September 2002 with a report written in Icelandic, which has been partly translated into English: "Risk Analysis regarding the import of dogs and cats to Iceland". The report contains considerations about 54 listed diseases/infections (see Appendix 1).

The introduction of this document includes the sentence: "Chapter five deals with diseases that need to be taken into account when importing dogs and cats". It may therefore be assumed, that this is *de facto* what is referred to in Regulation 935/2004 , paragraph 10: "At the Isolation Facility, samples shall be taken to determine the existence or non-existence of infectious diseases according to the instructions of the Chief Veterinary Officer of Iceland at any given time".

The present report was commissioned by the Ministry of Industries and Innovation of Iceland (MII) in October 2017 with the following tasks:

"…. a review of the current Icelandic legislation and administrative regulation concerning the import of dogs and cats to Iceland, with special attention to possible modifications pertaining to the import of guide dogs, all with a view to preserve and maintain the current high level of protection of Icelandic animal health".

Also, "The report will be based on internationally accepted science-based guidelines for qualitative animal health risk analyses, including risk assessment, risk management and risk communication. The work will be conducted in close collaboration with Icelandic animal health specialists".

A visit to Iceland was carried out by the author on 11 – 15 December 2017, during which a series of meetings was held with staff members of MII, Keldur, MAST and the Quarantine Station at Keflavik to review and discuss the current import conditions for dogs and cats to Iceland, and to reveal past experience with introductions of potentially pathogenic agents.

An additional visit took place on 16 – 19 September 2018 where various technical issues of importance and preliminary draft sections were discusses with the Icelandic specialists. Furthermore, a Skype session was conducted on 15 November 2018, and on a few additional occasions, meetings with Dr. Hreinsson took place in Copenhagen. The author gratefully acknowledges all of the Icelandic specialists for their significant contributions to this report.

CHAPTER 2. RISK ANALYSIS METHODOLOGY

The internationally accepted methodology of Risk Analysis is to be applied to this assignment, and its contents can be illustrated in Figure 1:

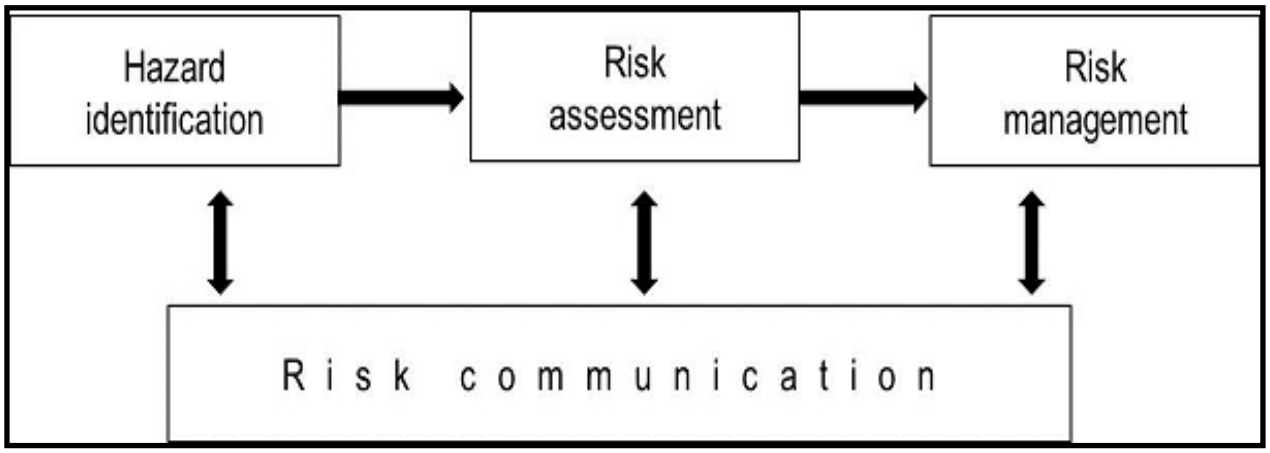


Figure 1. The four components of Risk Analysis (From OIE TAHC, 2017)

- Hazard Identification: the identification of the relevant potential diseases/infections to consider
- Risk Assessment: the scientific evaluation of the risks, i.e. the likelihood and potential consequences of those diseases/infections identified;
- Risk Management: alternative appropriate measures to reduce the risks identified
- Risk Communication: the sharing of relevant risk information with all stakeholders

In connection with export-import of animals, the principles of Risk Analysis have been endorsed and applied by the relevant international standard setting organizations, such as the World Trade Organization (WTO) and the World Animal Health Organization (OIE), both of which Iceland is a member, and these principles are implemented in their respective professional guidelines for trade and animal health protection, i.e. the WTO-SPS Agreement and the OIE Terrestrial Animal Health Code (TAHC).

Risk Analysis should:

- **Identify hazards**
- **Characterize risks**
- **Recognize uncertainty**
- **Summarize conclusions**
- **Recommend management options**
- **Document the basis for decisions**

Benefits of Risk Analysis:

- **Justify and defend decisions**
- **Evaluate decisions of others**
- **Prioritize resources**
- **View risk objectively and realistically**
- **Identify research and information needs**
- **Identify technical points of difference**

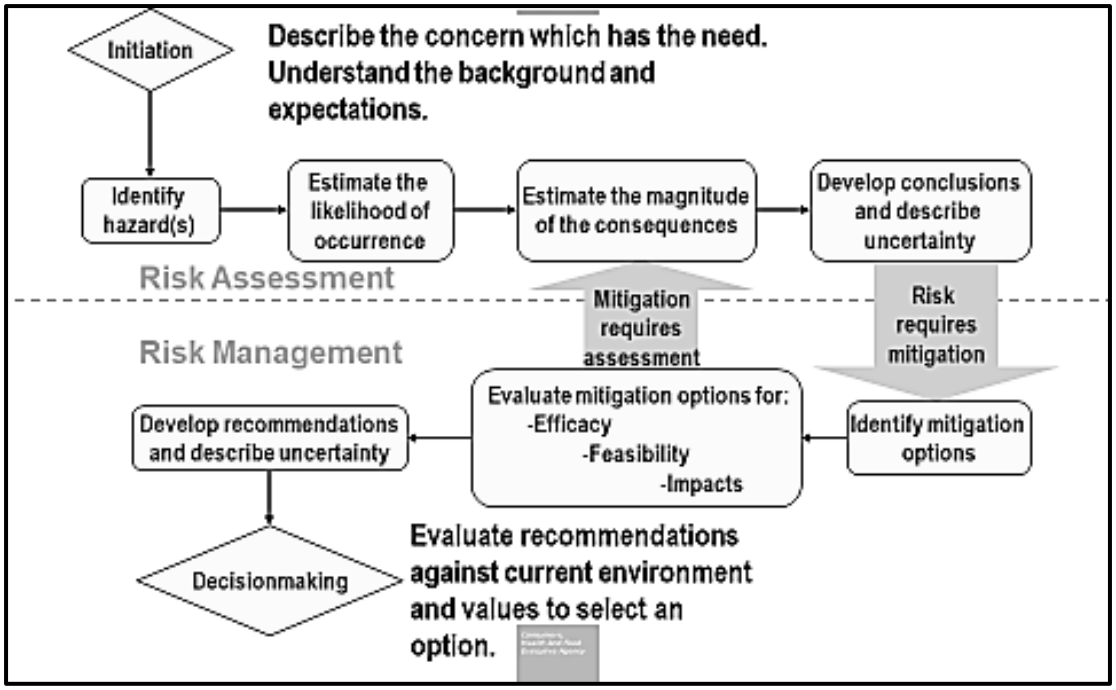


Figure 2. Flow-chart of the processes and interactions of Risk Assessment and Risk Management

Risk assessment methodology

Risk assessments may be conducted and their results presented either qualitatively or quantitatively. Qualitative assessment does not require mathematical modelling skills and is often the type of assessment used for routine decision making (TAHC, 2017). Simple interpretations and conversions between the results of the two types of assessment can be found in Table 1.

Table 1. Probability/likelihood conversion table Modified from: Generic Import Risk Analysis (IRA) for Pig Meat - Final Import Risk Analysis Report. Department of Agriculture, Fisheries and Forestry, Australian Government, 2004. Available at: *http://www.daff.gov.au/_data/assets/pdf_file/0018/18081/2004-01b.pdf*

Qualitative interpretation	Quantitative interpretation		
Likelihood	Description	Probability	Percentage
Very low	Event very unlikely to occur	< 0.05	< 5%
Low	Event unlikely to occur	0.05 - 0.3	5 - 30%
Moderate	Event likely to occur	0.3 - 0.7	30 - 70%
High	Event very likely to occur	0.7 – 1.0	70 - 100%

For the evaluation of the risk associated with the potential importation of diseases and infections with dogs and cats to Iceland, both qualitative and quantitative risk assessments was used, mainly depending on the availability of data to enable specific quantitative or semi-quantitative risk-estimates.

The components of risk assessment according to the procedures described in the OIE Handbook (2004):

A. Entry assessment
B. Exposure assessment
C. Consequence assessment
D. Risk estimation

Where a single exposure group has been identified, the risk assessment would yield the following (qualitative, semi-quantitative or quantitative) results:

- the likelihood of entry
- the likelihood of exposure
- the likely magnitude of consequences, for dogs and/or cats, as well as for humans.

In addition, where possible, import volume should also be investigated and should be included in the process of risk estimation. Trade volume can be included in the release and exposure assessments or examined at the completion of an assessment. If trade volume is to be included at the completion of an assessment, it will be necessary to carry out the release and exposure assessments using a suitable 'basic unit'. When the commodity is 'live animals' then an individual animal would be a suitable basic unit.

After the most appropriate basic unit has been determined, the release and exposure assessments should be multiplied to give the likelihood of entry and exposure. Where both of the components have been estimated semi-quantitatively or quantitatively, this will be a mathematical procedure and can be incorporated in the spreadsheet model. Where one or other components has been evaluated qualitatively, then it will be necessary to combine them by using the approaches described in the subsequent discussion of qualitative and semi-quantitative release and exposure assessment.

In a qualitative risk assessment, each of these elements makes use of combining categories through the use of tables, e.g. to assess the overall probability of entry by estimating the number of infected animals based on the number of animals imported and the likelihood of an imported animal being infected (Table 2).

Table 2. Entry assessment: The likelihood of entry of one or more infected animals from a given exporting country per time unit

Probability of an imported animal being infected	Number of animals imported			
	Very low	**Low**	**Moderate**	**High**
Very low	Negligible	Very low	Very low	Low
Low	Very low	Low	Low	Moderate
Moderate	Very low	Low	Moderate	Moderate
High	Low	Moderate	Moderate	High

If data are available to perform a quantitative estimation of the likelihood of entry, the following procedure can be applied (AUS 2001):

The likelihood of entry, once obtained, should be modified by considering trade volume. The appropriate result of this procedure will be a likelihood phrased as 'the likelihood that a given disease will be introduced at least once as a result of importing a given commodity for 1 year'. Algebraically, this probability can be expressed as:

$$LI_{annual} = 1-(1-LI)^{VT}$$

where:

- LI_{annual} is the annual Likelihood of Introduction, i.e. of entry and subsequent exposure — that is, the likelihood that a given disease will be introduced to Icelandic dogs and/or cats as a result of importing dogs and/or cats for a period of one year;
- LI is the Likelihood of Introduction, expressed in terms of the chosen 'basic unit';
- VT is the volume of trade, expressed as the number of basic units imported during one year;

To enable a proper combination of qualitative likelihood estimates, e.g. from the entry and the subsequent exposure assessments, Table 3 was used**.**

Table 3. Combination matrix used to jointly evaluate two likelihood estimates, based on the assumption that the second event (exposure) is conditioned on the first event (entry). Modified from: EFSA Panel on Animal Health and Welfare (AHAW 2013). Scientific Opinion on the risk of entry of *Aethina tumida* and *Tropilaelaps spp*. in the EU.

Likelihood of entry	**Likelihood of exposure**			
	Very low	**Low**	**Moderate**	**High**
Very low	**Very low**	**Very low**	**Very low**	**Very low**
Low	**Very low**	**Low**	**Low**	**Low**
Moderate	**Low**	**Low**	**Moderate**	**Moderate**
High	**Low**	**Moderate**	**Moderate**	**High**

After an estimate of the likelihood of introduction has been obtained and expressed in units that reflect the likely trade volume, this can be combined with the assessment of consequences to derive a risk estimate. Where all components of the risk assessment are quantitative, this will simply be a mathematical procedure. In the more common situation where there are one or more qualitative elements, then a set of 'decision rules' will be required.

The qualitative impact categories of direct and indirect consequences of exposure, establishment and spread of an infection and/or disease to the Icelandic small animal populations are presented in Table 4.

Table 4. Impact of direct and indirect consequences

Impact of consequences[1]	Direct consequences				Indirect consequences
	Infection	Disease	Production loss	Public health	Control costs
Very low	Few cases Asymptomatic	Few cases, short duration	Temporary, slight decrease	Few cases, short duration	None
Low	Low incidence, Asymptomatic	Low incidence short duration, no mortality	Temporary decrease, short duration	Few cases, temporary illness	Low
Moderate	Moderate incidence, symptoms	Moderate incidence, moderate duration, low mortality	Moderate decrease, moderate duration	Moderate case numbers, moderate illness, moderate duration	Moderate
High	High incidence and/or rapid spread, carriers, latent infections	High incidence, treatment required, long duration mortality, poor welfare	Severe decrease, long duration, treatment costs, mortality/ culling	High incidence, long duration, hospital treatment, mortality	High

[1]The highest impact score among the five columns determines the overall impact level

To combine the joint likelihood-estimate from Table 3 with the impact of the consequences from Table 4, the matrix in Table 5 is used to estimate the overall risk.

Table 5. Risk characterization matrix combining the joint likelihood of entry and exposure from Table 3 with the consequence impacts in Table 4. (Modified from Generic Import Risk Analysis (IRA) for Pig Meat - Final Import Risk Analysis Report. Department of Agriculture, Fisheries and Forestry, Australian Government, 2004. Available at: *http://www.daff.gov.au/__data/assets/pdf_file/0018/18081/2004-01b.pdf)*

Combined, conditional likelihood	Consequence impacts			
	Very low	Low	Moderate	High
Very low	Very low risk	Very low risk	Very low risk	Very low risk
Low	Very low risk	Low risk	Low risk	Low risk
Moderate	Very low risk	Low risk	Moderate risk	Moderate risk
High	Very low risk	Low risk	Moderate risk	High risk

Risk Management

When a risk-estimate according to Table 5 for a given disease/infection is above the chosen threshold, risk management procedures should be considered. According to the WTO-SPS Agreement this threshold value is called the Appropriate Level of Protection (ALOP). Apparently, an official ALOP level has not been defined for import of dogs and cats to Iceland.

Assuming, however, that Iceland would accept only a "very low risk", i.e. < 5% probability of importing one or more infected dogs and cats per year among the pets for all of the listed diseases - as Australia and New Zealand have chosen, while those diseases/infected animals covered by the relevant OIE TAHC chapters should be dealt with accordingly.

- Optional management tools to be used to reduce the hazard to a very low level of risk:
    - Quarantine? If so, Station or home?
    - Vaccination?
    - Testing?
    - Prophylactic treatment? Pre- and/or post-arrival?
    - If needed, specify management by country/region of origin, when geographical variations in incidence and prevalence exist?

- How effective are the options?
- How feasible are the options?
- What impacts do the options have?
- What is the level and type of uncertainty?
- What is the best option?

Risk Communication

Risk communication is an interactive process among individuals, groups and institutions with open, multiple exchanges of information and opinions, which through consulting, informing, explaining and justifying lead to better understanding and decisions, e.g. about risk management measures.

Stakeholders in risk communication:

- Animal owners, veterinarians
- The public, consumers, merchants
- Special interest-groups
- The media
- The industries, organizations
- State and federal governments, departments
- The parliament, politicians
- International trade partners

CHAPTER 3. HAZARD IDENTIFICATION OF RELEVANT CANINE AND FELINE TRANSMISSIBLE PATHOGENS

According the WTO-SPS agreement in the context of import of dogs and cats to Iceland, the potential hazards are all transmissible diseases and infections that may affect - but do not currently occur among - the domestic dog and cat populations nor other indigenous animal species, as well as indigenous diseases and infections that are covered by current official eradication or control programs. It is not a requirement that all such diseases and infections are defined as notifiable, reportable or in other ways listed by the Icelandic authorities.

It is however implicit, that these potentially hazardous conditions should be capable of entering, exposing, transmitting, spreading and causing negative consequences among the indigenous animal and/or human populations of Iceland.

Consequently, the list of potential hazards is not a static list, but a dynamic one, which might compose novel pathogens, changes in susceptibility, pathogenicity, etc. of existing pathogens

Furthermore, since each country can define its Appropriate Level of Protection (ALOP), the competent national authority may decide which pathogens to define as hazardous in this context. Normally, a list of the relevant hazards is published as “notifiable” or “reportable” in a formal piece of regulatory act or alike, specifying when and how to report suspected occurrence of such a disease to the proper authorities. In Iceland, however, this is slightly different, in that only some of the relevant diseases are in fact listed in formal regulations, as follows:

LISTED BY THE ICELANDIC REGULATION

Regulation No. 52, 24 January 2014: Regulation on notifiable and reportable animal diseases

Article 2. Serious notifiable diseases, including:

Multiple species

B052. Aujeszky’s disease

A010 FMD

B352 Tularemia

B058 Rabies

B051 Anthrax

B103/B253 Brucellosis

Dogs, cats

I501 *Sarcoptes* mange (Scapies)

I504 *Echinococcus multilocularis*

Article 3. Other notifiable diseases

Multiple species

I002 Ringworm

B056 Leptospirosis

I003 Neosporosis

C619/C855 Intestinal *Salmonella* infections

B053 *Echinococcus granulosis*

Dogs, cats

I505 Canine distemper

B501 Canine leishmaniosis

Article 5. Reportable diseases

Dogs, cats

I509 Ear mites

I510 Feline leukemia virus

I511 Cheyletiellosis

I512 Canine parvovirus

I513 *Hepatitis contagiosa canis* (HCC)

All of the above sources of identified transmissible pathogens are represented in the list of infections mentioned here in Annex 3, which lists all 54 diseases/infections considered in the Icelandic 2002 report.

Regulation 935/2004 on the importation of pets and dog semen gives the Chief Veterinary Officer of Iceland the authority to instruct at any given time sampling at the Isolation Facility to determine the existence or non-existence of infectious diseases. The 2002 report listing is presumably consistent with this authorization.

In addition, international regulations have classified certain diseases and agents as sufficiently pathogenic to be reported, prevented and controlled according to internationally accepted principles and procedures, including Importation of Animals Act no. 54, 16 March 1990 and trade and import regulations to limit the transfer and spread of pathogens and diseases.

COVERED BY INTERNATIONAL REGULATIONS (OIE)

OIE:

Iceland is a member of the OIE.

The OIE Terrestrial Animal Health Code 2017 (TAHC) contains chapters on the following pathogenic agents, for which the dog may be a potential major transmitter and for which risk management measures in import situations are provided:

Rabies: TAHC Chapter 8.14

Article 8.14.4. Recommendations for importation of domestic animals from rabies free countries: An International veterinary certificate* is required, attesting that the animals:

1: showed no clinical sign of rabies the day prior to or on the day of shipment;

2: and either:

A: have been captured at a distance that precludes any contact with animals in an infected country. The distance should be defined in accordance with the biology of the species exported, including home range and long- distance movements;

Or

B: have been kept in captivity for the six months prior to shipment in a rabies free country.

Rabies-free countries: (see reference to this in Icelandic Regulation 935/2004)

Article 8.14.6. Recommendations for importation of dogs, cats and ferrets from countries considered infected with rabies:

An International veterinary certificate is required, attesting that the animals:

1: showed no clinical sign of rabies the day prior to or on the day of shipment;

and

2: were permanently identified and their identification number stated in the certificate;

AND EITHER:

3: were vaccinated or revaccinated, in accordance with the recommendations of the manufacturer. The vaccine should have been produced and used in accordance with the Terrestrial Manual;

AND

4: were subjected not less than 3 months and not more than 12 months prior to shipment to an antibody titration test as prescribed in the Terrestrial Manual with a positive result of at least 0.5IU/ml;

OR

5: were kept in a quarantine station for six months prior to export.

Echinococcus granulosus*: *TAHC Chapter 8.5

Article 8.5.5. Recommendations for the importation of dogs and wild canids from an infected country

Veterinary Authorities of importing countries should require the presentation of an international veterinary certificate attesting that:

1: the animal has been treated between 24 and 72 hours prior to embarkation with praziquantel (5 mg/kg), or another cestocidal product with comparable efficacy against intestinal forms of *E. granulosus;*

2: adequate precautions have been taken to avoid reinfection of the animal between treatment and embarkation.

Echinococcus mulltilocularis: TAHC Chapter 8.6

Article 8.6.2. Safe commodities

When authorising import or transit of any commodities of livestock, Veterinary Authorities should not require any related conditions regardless of the status of the animal population of the exporting country or zone. NB: See below on EU's regulation on EM.

Article 8.6.5. Recommendations for the importation of dogs and wild canids from an infected country

Veterinary Authorities of importing countries should require the presentation of an international veterinary certificate attesting that:

1: the animal has been treated between 24 and 72 hours prior to embarkation with praziquantel (5 mg/kg), or another cestocidal product with a comparable efficacy against intestinal forms of *E. multilocularis;*

2: adequate precautions have been taken to avoid reinfection of the animal between treatment and embarkation.

CHAPTER 4 RISK ASSESSMENT OF RELEVANT CANINE AND FELINE TRANSMISSIBLE PATHOGENS

Strategies to assess the risk for categories of transmissible pathogens and susceptible animals

Depending on the basic information available for each of the relevant diseases/infections, different strategies for estimating the risk to Iceland of introduction, establishment and spread have been or may be pursued.

1. For infections that have been detected during quarantine testing in the past in Iceland and for which numerical frequencies of positive reactors have been published, a quantitative assessment of the likelihood of introduction may be attempted. It has furthermore been possible with the use of the annual statistics from MAST of the number of imported dogs and cats to update the estimations to the current levels of imported dogs and cats. This has been possible for several parasitological infestations of dogs and cats, as will be shown below (Tables 7 – 9).
2. For some of the other infections for which numerical historic data are not available, it is possible to estimate whether or not the risk to the Icelandic dogs and/or cats is above or below any ALOP assumed for Iceland. This is based on the fact, that if published information on the consequences of an introduction is available and is found to be below the ALOP threshold of risk assumed for Iceland, that infection is of no concern irrespective of its likelihoods of introduction and exposure-establishment.
3. Risk assessments for many of the relevant infections of dogs and cats have been recently performed elsewhere, and are available as working documents, e.g. from Nordic veterinary institutions (e.g. Høgåsen et al. 2012, Lind, 2014), from the European Food Safety Authority (EFSA) at the request of the EU Commission (e.g. EFSA VBD Storymaps), as well as from the Australian and New Zealand governmental bodies as preparations for updating their respective rules or imports of dogs and cats since 2000. The classifications proposed in these documents may function as a guide to what might be considered reasonably relevant for Icelandic conditions as well. Both Australia and New Zealand have produced recent guidelines on their respective import requirements for dogs and cats, and it seems reasonable to review these guidelines for the respective diseases/infections, especially when it comes to the questions of consequences to dogs/cats and to humans concerned.
4. All risk assessments are affected by uncertainty and variability, which need to be taken into account when interpreting the outcomes. Variability affects e.g. the numbers of dogs and cats imported per year, the distribution of countries of origin, the ages of the imported animals, etc., all of which may influence the probabilities of infections occurring. Also, available literature estimates of relevant disease occurrence vary. The prevalence of canine vector-borne infections is considered as rather low among dogs imported to Germany from various Mediterranean countries (Hamel et al. 2011), and that there is a low risk for such infections during a limited single stay in endemic countries (Hamel et al. 2013). However, Menn et al. (2010) concluded, that travelling with dogs to such regions carries a significant risk of acquiring a vector-borne infection.

Risks associated with the import of Assistance dogs to Iceland

Assistance dogs not only provide a specific service to their handlers, but also greatly enhance the quality of their lives with a new sense of freedom and independence.

There are three types of Assistance Dogs (use the links below to read more about the three categories):

- **Guide Dogs – for the blind and the visually impaired**
- **Hearing Dogs – for the deaf and hard of hearing**
- **Service Dogs – for people with disabilities other than those related to vision or hearing**

The issues associated with the import of Assistance dogs to Iceland were dealt with in a document in Icelandic from MAST dated the 2 of March 2012, parts of which was later translated into English. The document text was produced by a working group consisting of three vets (from MAST?).

It is important to notice, that "Assistance dogs" cover a number of different uses of these dogs, e.g. on the one hand individually owned or assigned dogs to assist individuals with reduced vision, hearing or other debiliating conditions for the owner/user to better manage getting around safely, but on the other hand also contain rescue- or service- dogs, handled by the police, military or emergency crews in case of physical or environmental emergencies, including localization and rescue operations, etc. A discussion of the often confusing and conflicting glossary used for these different types of assistance dogs can be found in Yamamoto et al. (2015).

For the category of Guide dogs, which is the subject of the 2012 document, the underlying theme is, to what extent it might be feasible to limit the general requirements for import quarantine procedures, so as to minimize the duration and/or the physical restraining required for imported dogs in general, and thereby reducing the physical and emotional stress for the service dog owner/user, hampered by reduced abilities or other significant handicaps for the duration of the quarantine period. One possible solution mentioned in the MAST document might be the use of home quarantine as a replacement of the regular physical quarantine conditions for 28 days or more. Both Australia and New Zealand regulation in place to allow documented guide-dogs to go through home quarantine after arrival.

One of the mitigating facts mentioned in the 2012 document is, that the annual number of these service dogs expected for import to Iceland is likely small compared to the total number of dogs imported to Iceland per year. Therefore the annual risk of disease introduction caused by imported service dogs would be much lower than the annual total risk estimated for the imported group of companion dogs (pets, farm and hunting dogs), everything else being equal.

Another factor which should be considered is the potential differences between guide dogs and pet- or hunting-dogs in their exposure to various disease agents.

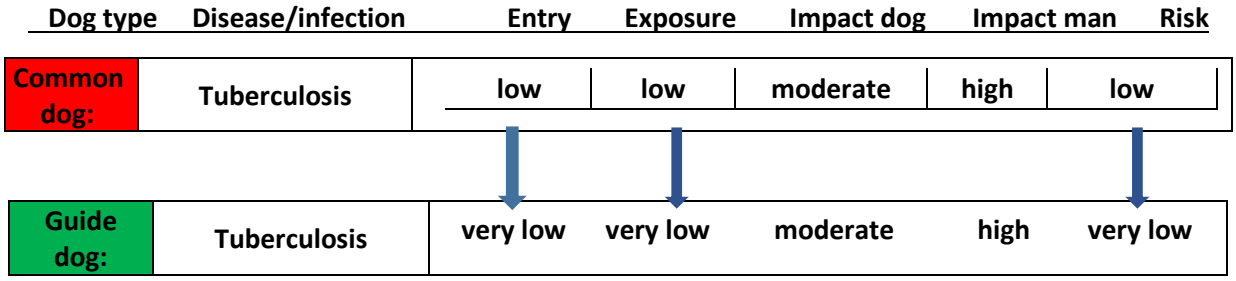
Typically, guide dogs are highly trained with respect to remaining in close physical contact with their owner/user at all times outside of the home environment. Guide dogs are most often also spayed/neutered, on a leash and not likely to stray around outside of the owners/users close physical location. Thus, typical conditions under which ordinary non-restricted dogs might become affected by environmental-, wild animal- or dog-to-dog-transferred disease agents are much less likely to affect guide dogs. An exemption might be vector-borne infections, assumng that guide dogs and the other types of dogs spend at least some time outdoors.

In other words, the probabilities of entry and of exposure to/establishment of disease agents via imported dogs are likely to be somewhat reduced for guide dogs compared to the average pet-, farm- or hunting-dog, since a lowered probability for guide dogs of exposure to/establishment of infectious agents can reasonably be assumed to exist, both in the country of origin/departure before the travel as well as In Iceland after the importation. Estimates of the numerical differences in the probabilities of entry of and of exposure to disease agents between guide dogs and ordinary pet- and working dogs are not likely to be quantified and recorded, but at a common scale of qualitative risk assessment, a fair and conservative estimate would be, that both the likelihoods of the entry of and the subsequent exposure to the non-vector-borne disease agents considered here, are at least one category-step lower than for

free-roaming dogs. If this model is accepted, it becomes feasible to assess the risks associated with the import of guide dogs, using the lowered estimate of both entry and exposure/establishment to assess the risk for the disease/infection categories considered, except vector-borne diseases/infections. It should be noted, that the risk estimates of vector-borne diseases/infections already tend to be low due to low levels of exposure due to the scarcity of vector organisms in Iceland caused by the climatic conditions.

EXAMPLE:

Using the scale of: ”negligible”, ”very low”, ” low”, ”moderate” and ”high” with results from the RA Summary:



Assuming that ”very low” may be considered as the Icelandic ALOP value (as for Australia and New Zealand), then the risk of importing a guide dog would be acceptable without any risk management procedures to deal with a possible infection with tuberculosis, whereas dealing with the import of a common dog, i.e. a pet-, farm- or hunting-dog, would require some kind of risk management, e.g. a quarantine period with testing for tuberculosis.

Appendix 1 contains all the detailed information and classifications of the 54 listed diseases/infections, and Risk assessment Summary Tables 1 – 5 (see end of Appendix 1) have been included, with Summary Tables 4 and 5 using the above methodology for the Guide dogs. An overview of the results is given in Table 6, where It can be seen, that when using the above-mentioned assumption of Guide dogs being less exposed to the environment, wild-life and other dogs potentially carrying and transmitting pathogenic organisms, before as well as after the import to Iceland, the risk categorizations of ”Acceptable risk”, covering the ”very low”, ”very low/negligible” and ”negligible” risk categories, changes from an estimated 15 (37%) conditions among All dogs to 22 (54%) among Guide dogs. This expected lower risk for Guide dogs could motivate less strict import risk management procedures for Guide dogs imported to Iceland, at least for the 22 conditions designated with with ”Acceptable risk”.

Table 6. Comparisons of risk categories distributions proposed for the 54 listed diseases/infections among All dogs and Guide dogs

Dog categories	Risk categories										
	High	High/ Mode -rate	Mode -rate	Mode -rate/ Low	Low	Low/ Very low	Very low	Very low/ Negligible	Negligible	Accept -able risk*	Total
All dogs											
Diseases per risk category	5	1	11	1	7	1	11	0	4	15	41
% of all diseases	12%	2%	27%	2%	17%	2%	27%	0%	10%	37%	100%
Cumulative number of diseases	5	6	17	18	25	26	37	37	41		
Cumulative % of all diseases	12%	15%	41%	44%	61%	63%	90%	90%	100%		
Service dogs											
Diseases per risk category	0	0	6	0	12	1	6	1	15	22	41
% of all diseases	0%	0%	15%	0%	29%	2%	15%	2%	37%	54%	100%
Cumulative number of diseases	0	0	6	6	18	19	25	26	41		
Cumulative % of all diseases	0%	0%	15%	15%	44%	46%	61%	63%	100%		

**Acceptable risk according to the assumption of ALOP for Iceland being "very low"*

Risk assessment: Quantitative methodology

Two publications by the group of parasitological experts at Keldur provide data on findings from dogs and cats during quarantine after arrival to Iceland (Eydal et al. 2001; Eydal & Skirnisson 2016). The 2001 publication summarizes the endoparasites found in fecal samples taken during the quarantine periods for 608 dogs and 236 cats arriving during 1989 – 2000. The results are shown in Table 7.

Table 7.

Eydal et al. 2001: Parasites of imported dogs and cats in Iceland 1989 - 2000				
	Dogs		Cats	
Origin	12 years	Mean	12 years	Mean
Europe	447	37	137	11
N. America	141	12	92	8
Africa	3	0	0	0
Australia	1	0	0	0
S. America	0	0	1	0
Asia	0	0	1	0
Unknown	16	1	5	0
Total	608	51	236	20

Table 8 contains more recent data to be used to account for the increasing number of imported dogs and cats since 2000. The data are extracted from the MAST Annual Reports for 2012 – 2016.

Table 8.

Recent imports (MAST Annual Reports)		
Year	Dogs	Cats
2012	151	47
2013	167	36
2014	165	29
2015	169	43
2016	217	49
Mean	174	41
Total	869	204

Table 9. Data on endoparasites detected in imported and quarantined dogs and cats 1989 – 2000 published by Eydal et al. (2001).

Dogs

Endoparasites	Cases		Mean prevalence	Recent entry prob. per year[1]	Qualitative entry prob.	Exposure	Consequence	Qualitative risk
	Total	Per year						
Anchylostoma sp.	3	0,25	0,0049	0,58	Moderate	Low/moderate[2]	Low/moderate[2]	Low/moderate
Isospora canis	2	0,17	0,0033	0,44	Moderate	n.a.		
I. bahiensis	2	0,17	0,0033	0,44	Moderate	n.a.		
I. rivolta	4	0,33	0,0066	0,68	Moderate	n.a.		
*Strongyloides stercoralis**	20	0,74	0,0062	0,66	Moderate	Low/moderate[2]	Low[2]	Low
Trichuris vulpis	4	0,33	0,0066	0,68	Moderate	n.a.		
Digenea	2	0,17	0,0033	0,44	Moderate	n.a.		

Cats

Endoparasites	Cases		Mean prevalence	Recent entry prob. per year[1]	Qualitative entry prob.	Exposure	Consequence	Qualitative risk
	Total	Per year						
Anchylostoma sp.	1	0,08	0,0042	0,16	Low	Low/moderate[2]	Low/moderate[2]	Low
Digenea	1	0,08	0,0042	0,16	Low	n.a.		

***Data for 1989 - 2016, see M. Eydal & K. Skirnirsson (2016): Strongyloides stercoralis found in imported dogs, household dogs and kennel dogs in Iceland.Icel. Agric. Sci.,29,39-51.**

[1] See explanatory note below

[2] Helga R. Høgåsen, Inger Sofie Hamnes, Rebecca Davidson & Arve Lund (2012): Importrisikovurdering av gatehunder fra Øst-Europa. Veterinærinstituttets rapportserie (Norwegian Veterinary Institute Report Series), Rapport 11 - see also Fig. 1.

Explanatory notes to Table 9: **Cases per year = 'Cases 1989 – 2000'/12**

Mean prevalence = Cases per year/ mean annual no. of imported dogs or cats (Table 7)

Recent import probability per year = 1 - (1 - mean prevalence) $^{\text{mean recent imports (Table 8)}}$

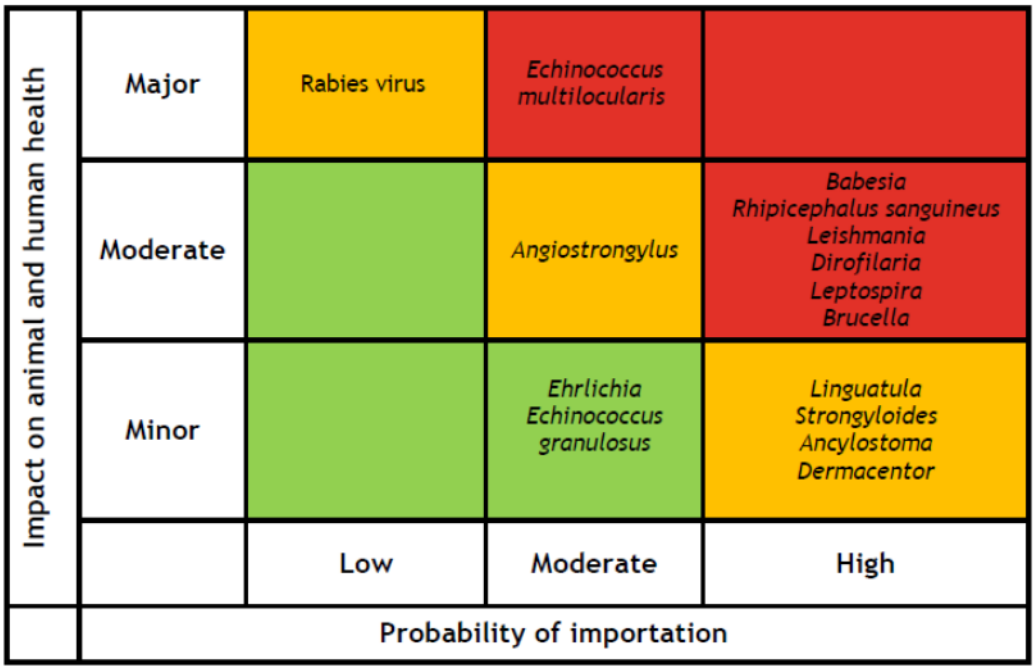


Figure 1. From Høgåsen et al. 2012

In Table 9, *Anchylostoma sp*. and *Strongyloides stercoralis* are listed as being of interest as potentially relevant agents for risk reducing measures when importing dogs and cats, since these endoparasites appear with a high probability of importation, but with estimated minor consequences, indicating an overall "low risk".

If one assumes that Iceland might concur to its ALOP to be "very low" - as for Australia and New Zealand (see below) -, there would be a need to implement risk management measures for these two conditions to decrease their estimated import risks to move from "low" to - or below - the "very low" risk level.

CHAPTER 5. RISK MANAGEMENT

Annex A of the SPS Agreement defines the concept of an 'appropriate level of protection' (ALOP) as the level of protection deemed appropriate by a WTO Member establishing a sanitary or phytosanitary measure to protect human, animal or plant life or health within its territory. Among number of obligations, a WTO Member should take into account the objective of minimizing negative trade effects in setting its ALOP, cf. Fig. 2.

Perceived ALOP for importing dogs and cats to Iceland

Countries have the right according to the WTO-SPS agreement to set their own Appropriate Level of Protection (ALOP) when importing animals and animal products, e.g. dogs and cats. In practice, this means that for diseases and infections not covered by the TAHC of the OIE (Rabies, *Echinococcus multilocularis* and *E. granulosus* are covered by the TAHC*),* countries can decide their respective ALOP, as long as it does not infringe on WTO-SPS principles.

The current situation of a relative absence of most transmissible diseases in Icelandic dogs and cats is partly due to the location of the country as an island surrounded by the sea, and partly due to a long-

standing policy of strict regulation on importation of animals and animal products, including dogs and cats accompanying visiting tourists, as well as such animals introduced by the Icelandic people. There are a few similar situations elsewhere, as exemplified by Australia and New Zealand, both of which also maintain strict regulation on the import of dogs and cats. Both of these countries have detailed and updated rules and regulations for imports of dogs and cats, including special provisions for assistance-/service-dogs. It would therefore seem reasonable to review the current Icelandic conditions for import of dogs and cats with a view to those adopted by Australia and New Zealand.

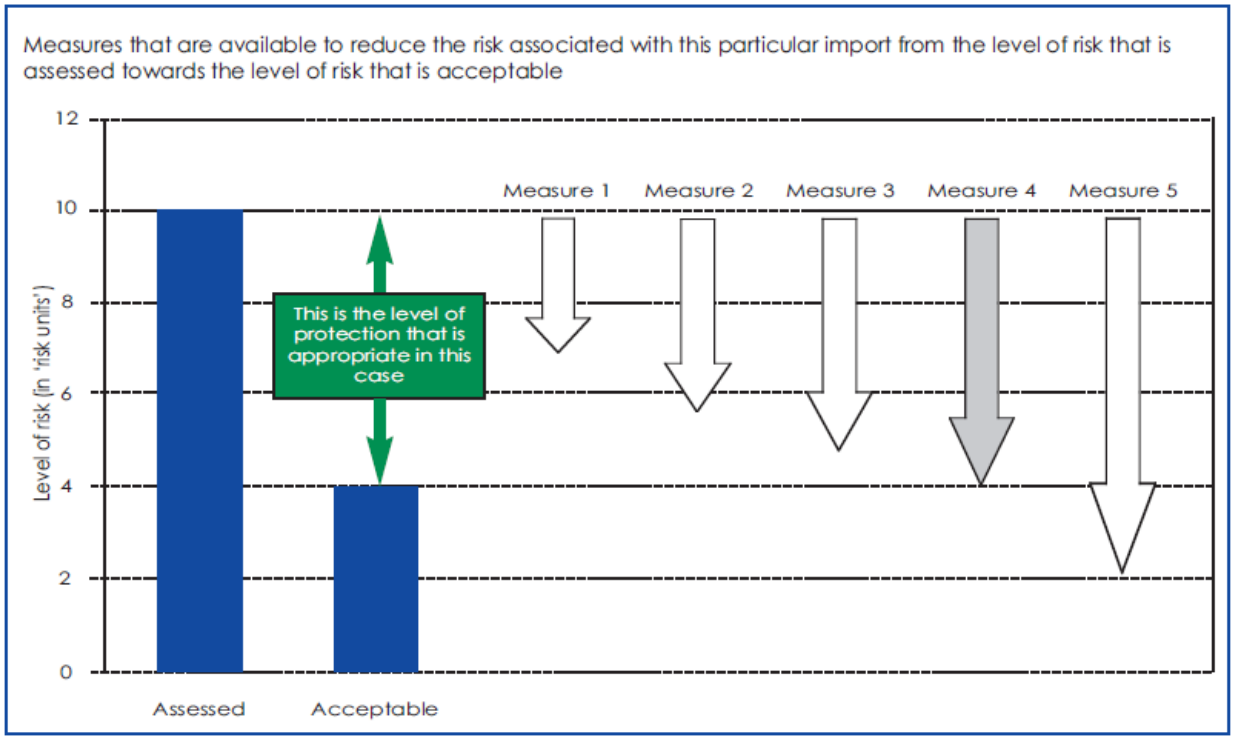


Figure 2. Relationship between assessed risk, acceptable level of risk, appropriate level of protection and alternative sanitary measures. From: MacDiarmid, S.C. & Pharo, H.J. (2003).

Risk management principles

The aim of a given risk management procedure is to reduce the estimated level of risk for any given disease/infection to the appropriate ALOP for Iceland. However, the countries of origin for imported dogs and cats do not present similar levels of risk for any given disease, primarily because the prevalence of pathogens are different in different countries and regions of the world, and because the numbers of animals presented in a given year at the Icelandic border varies greatly among countries and regions (e.g. see Table 7). Therefore, some importing countries, e.g. Australia, have grouped the exporting countries/regions into categories, which have been assigned different import risk management procedures, e.g. in the duration of quarantine, required testing procedures, etc., according to their presumed likelihood of presenting affected animals and/or the associated level of consequences.

The broad classification used in Table 7 may not suffice to discriminate high-risk countries/regions from less critical places of origin. Table 10 gives a more detailed break-down of the regions in Table 7 to illustrate both the uneven distribution of number of imported animals by countries of origin within regions listed in Table 7, and also some of the known geographical differences in the prevalence levels of relevant infections among the European countries.

Table 10. Regional differences in entry assessment for selected pathogen groups

Export region	Mean annual import of dogs and cats, 2012 - 2016	Entry assessment: The likelihood of entry of one or more infected animals from a given exporting region per year		
		Vector-borne diseases	Rabies	Helminth infections
Northern Europe	85	Very low	Very low	Low/Moderate
Central Europe	22	Moderate	Low	Moderate
The British Islands	20	Low	Very low	Low/Moderate
Southern Europe	17	High	Low	High
Eastern Europe	27	Moderate	Moderate	High
Unknown	3	n.a.	n.a.	n.a.
Total	174			

In order to account for such regional differences in entry assessments, differential risk management might be considered, as Australia and New Zealand have done in their respective import risk management procedures by grouping the countries of origin in different risk categories and assign different risk management procedures.

Risk management, however, is not purely science-based, but also greatly influenced and dependent on the national political, cultural and historical background and experience. It can be seen from the presentations of the risk management procedures for imports of dogs and cats described by Australia and New Zealand, that e.g. their rules for import of Guide Dogs and the choice of home quarantine as the chosen management measure to cope with the list of diseases and infections, has not been supported by any objective scientific facts, e.g. on sensitivity, specificity, safety and security. On the contrary, the home quarantine alternative is motivated as follows by New Zealand under: "**Assistance & guide dogs":** "Assistance dogs are highly trained animals that help people with special needs. The process for importing them to New Zealand is similar to importing other dogs, but special quarantine arrangements can be made **to minimise disruption for handlers**".

Obviously, this appears to be primarily a socio-political decision, and in order to allow the Icelandic authorities to consider similar non-science-based judgements, the present report will not recommend specific risk management procedures to be applied in connection with the import of dogs and cats to Iceland. However, the procedures decided upon by the Icelandic authorities should be consistent with whatever Iceland chooses to aim for in terms of ALOP and with the detailed risk assessment classifications presented in the former section on Risks associated with the import of Assistance dogs to Iceland, and with the comparisons of "Guide dogs" and "All dogs" (see Table 6 and the Summary Tables in Appendix 1).

APPENDIX 1. RISK ASSESSMENT OF INDIVIDUAL DISEASES/INFECTIONS

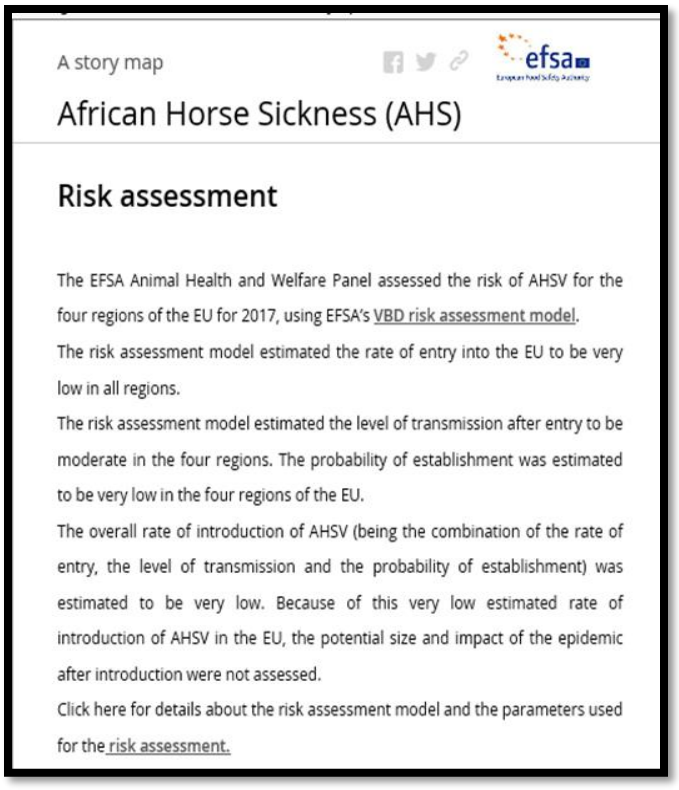
Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
African Horse Sickness (AHS)	**5.1.1**	EFSA VBD maps 2017* Merck Veterinary Manual OIE Technical Disease Card	Very low	Very low	Very low	Negligible	Very low

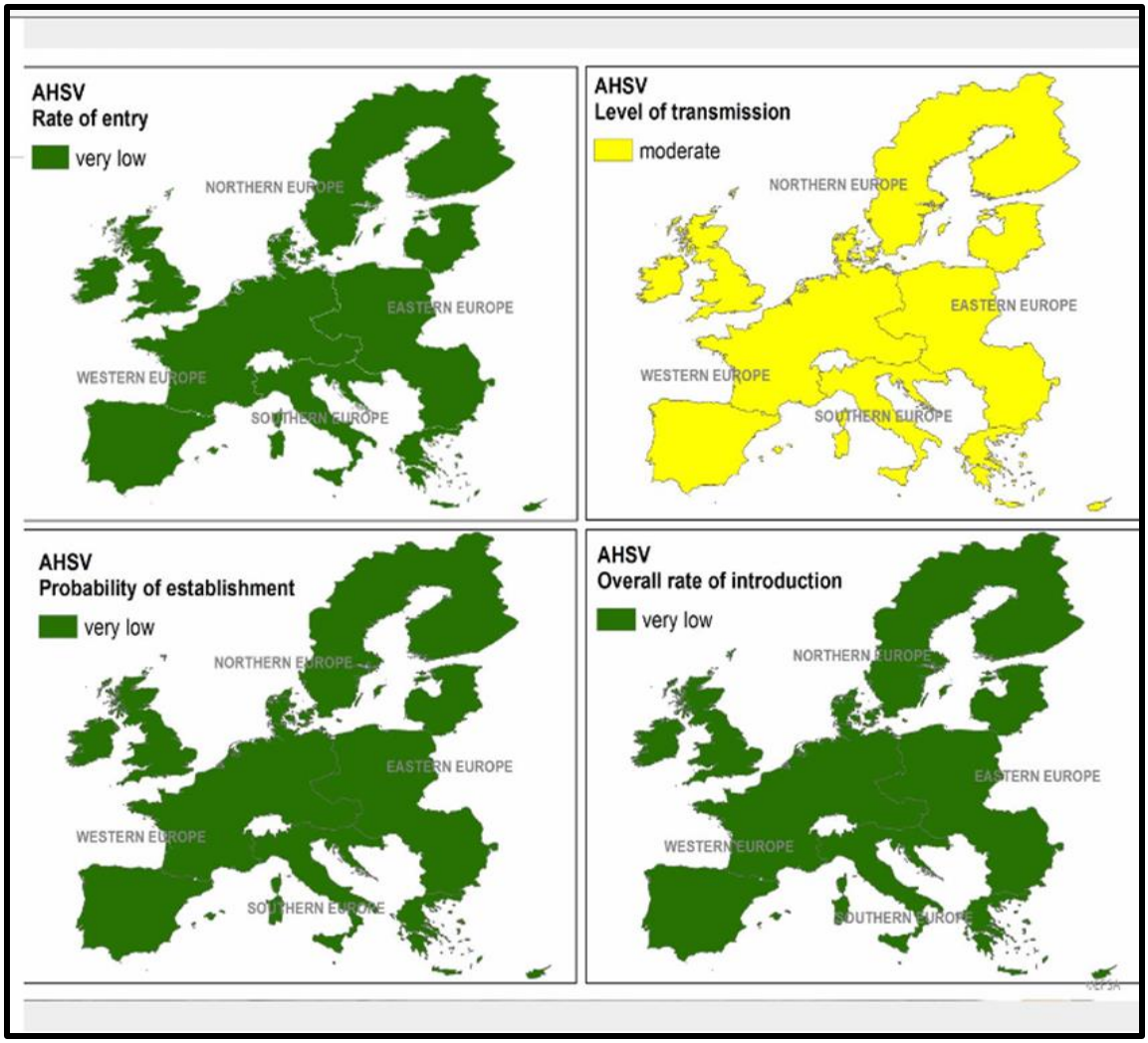
*https://efsa.maps.arcgis.com/apps/MapJournal/index.html?appid=df1fa0739f144112957d2557f9e60a58#

Transmission: *Culicoides* spp are the principal vectors of all nine serotypes of AHSV, with *C. imicola* usually considered to be the most important. Consequently, AHS is seen during warm, rainy seasons, which favor propagation of the vectors, and disappears when cold weather stops or significantly reduces vector activity. The virus also has been isolated from the dog tick *Rhipicephalus sanguineus*, and the camel tick *Hyalomma dromedarii* during the winter in southern Egypt, where the disease is endemic. Countries affected: 2017: 6 African countries

AHSV has apparently been transmitted between dogs by infected mosquitoes. Furthermore, dogs, and possibly large African carnivores such as lions and leopards, can be infected by ingestion of meat from AHSV-infected equids. Dogs have per-acute fatal infection after eating infected horsemeat, but are not a preferred host by *Culicoides* spp. and unlikely to play a role in transmission. It is generally considered that dogs and other large carnivores, ticks, and mosquitoes play little part in the epidemiology of AHS.

EFSA Vector- Borne Disease Maps (VBD):




AHSV
Rate of entry
very low
NORTHERN EUROPE
EASTERN EUROPE
WESTERN EUROPE
SOUTHERN EUROPE
AHSV
Level of transmission
moderate
NORTHERN EUROPE
EASTERN EUROPE
WESTERN EUROPE
SOUTHERN EUROPE
AHSV
Probability of establishment
very low
NORTHERN EUROPE
EASTERN EUROPE
WESTERN EUROPE
SOUTHERN EUROPE
AHSV
Overall rate of introduction
very low
NORTHERN EUROPE
EASTERN EUROPE
WESTERN EUROPE
SOUTHERN EUROPE

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Aujeszky's Disease	**5.1.2**	Merck Veterinary Manual, Lind 2014	Neglig.	Neglig.	High	Neglig.	Neglig.

Lind 2014	Zoonotisk risiko (Hund-> Homo)	Impact hos hund	Impact hos homo	Forekomst (endemicitet)
Aujeszky's	Ingen	Alvorlig	-	Sporadic (swine)

Dead-end hosts, such as dogs, cats, or wildlife, can transmit the virus between farms, but these animals survive only 2–3 days after becoming infected.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Feline calicivirus (FCV)	**5.1.3**	Merck Veterinary Manual Wikipedia	High	High	Mod.	Neglig.	Mod.

Feline respiratory disease complex includes those illnesses typified by rhinosinusitis, conjunctivitis, lacrimation, salivation, and oral ulcerations. The principal diseases, feline viral rhinotracheitis (FVR; feline herpesvirus type 1), **feline calicivirus (FCV),** *Chlamydia felis*, *Mycoplasma felis*, or combinations of these infections, affect exotic as well as domestic species.

Most acute feline upper respiratory infections are caused by FVR virus, although FCV may be more prevalent in some populations. Dual infections with these viruses may occur. FVR and caliciviruses are host-specific and pose no known human risk.

FCV is very contagious, and latently infected cats will continue to shed viruses, so complete control is difficult. The prevalence of FCV varies depending on the environment. In private households, FCV is present in about 10% of cats (either in active or carrier state), while the prevalence in shelters or catteries is 25 to 40%.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Canine Distemper	**5.1.4**	Merck Veterinary Manual, Wikipedia, Lind 2014, AHAC8	High	High	High	Neglig.	High

Lind 2014	Zoonotisk risiko (Hund-> Homo)	Impact hos hund	Impact hos homo	Forekomst (endemicitet)
Distemper	Ingen	Alvorlig	-	Globalt

Canine distemper is a highly contagious, systemic, viral disease of dogs seen worldwide. Clinically, it is characterized by a diphasic fever, leukopenia, GI and respiratory catarrh, and frequently pneumonic and neurologic complications. Its epidemiology is complicated by the large number of species susceptible to infection. Domestic dogs (including feral populations) are considered to be the reservoir species in most, if not all, locations. Antigenic drift and strain diversity are increasingly documented in association with outbreaks in wild species, domestic dogs, and exotic animals held in zoos and parks.

In canines, distemper impacts several body systems, including the gastrointestinal and respiratory tracts and the spinal cord and brain, with common symptoms that include high fever, eye inflammation and eye/nose discharge, labored breathing and coughing, vomiting and diarrhea, loss of appetite and lethargy, and hardening of nose and footpads. The viral infection can be accompanied by secondary bacterial infections and can present eventual serious neurological symptoms.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Feline poxvirus (Cowpox Virus Infections in Cats)	**5.1.5**	Merck Veterinary Manual, Reperant et al., 2016 ecdc: *https://ecdc.europa.eu/en/cowpox/about-disease-cowpox*	Mod.	Mod.	Low	Low	Low

Many orthopox-virus infections of domestic animals and people result from "spill over" from rodent reservoir hosts. Although traditionally described as infecting cattle, infections of cattle with cowpox virus are now very rare. Domestic cats in Europe are now the most commonly recognized species clinically affected by cowpox virus. Many cats show no signs other than skin lesions, but ~20% may develop mild coryza or conjunctivitis.

Sporadic human cases of cowpox have been reported in Europe, mostly linked to handling of infected animal, usually rodents and cats. Human infection results from direct contact with an infected animal. The disease in humans presents as localized lesions mainly on fingers, hands or the face, in the form of red blisters. The disease is self-limiting in immunocompetent persons. No specific treatment is available. There is no known evidence of human to human transmission of the virus.

. Cat-to-cat transmission can also occur but usually results in only subclinical infection.

The significance of the disease and its relatively recent recognition in cats is an enigma. It may have always been present in the feline population, but not recognized. Alternatively, the disease may be increasing in importance as a result of a change either in the epidemiology of the disease, in the reservoir host, or in the nature of the dominant biotype of the virus itself.

However, most domestic cats recover uneventfully. More severe pulmonary disease is uncommon in domestic cats. In domestic cats, supportive treatment (broad-spectrum antibiotics, fluid therapy) is generally successful, and mortality is low.

Because it seems that infection in domestic cats is mainly sporadic and acquired from chance contact with an infected wildlife reservoir, it is difficult to control the exposure of outdoor cats to this virus.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Feline leukemia virus	**5.1.6**	Merck Veterinary Manual, Hansen & Holm 2001	sero-reactors*	High	High	Neglig.	n.a.*

*Two among 115 randomly selected Icelandic cats (1.7%) were found to be sero-positive for FeLV. It is possible, however, that the result is due to false positive reactions (Hansen & Holm 2001).

Feline leukemia virus (FeLV) remains one of the most important infectious diseases of cats globally. It manifests primarily through profound anemia, malignancies, and immunosuppression and infects domestic cats and other species of Felidae. In the laboratory, cells from a much wider range of species can be infected by some strains of the virus.

FeLV is considered to be an age-dependent disease; young kittens are at higher risk of progressive infection and more rapid disease progression, whereas adults display some degree of age resistance. However, transmission can occur at any age, and factors affecting clinical course of disease are complex and incompletely understood. Persistently infected, healthy cats serve as reservoirs of FeLV for both vertical and horizontal viral transmission. Oronasal contact with infectious saliva or urine represents the most likely mode of horizontal transmission; vertical transmission in utero or through nursing is also common.

Management of infected cats:

Infected cats may have a good quality of life. Although many cats succumb within 3 yrs. of diagnosis, others remain clinically healthy for multiple years.

Preventive veterinary care, including frequent physical examination, laboratory monitoring, core vaccination, spay/neuter surgery, dental prophylaxis, and parasite control, is essential.
Avoid transmission to other cats by preventing access to outdoors and other uninfected cats in household.

Many antiviral and immunotherapeutic treatments are described in early trials or anecdotal use, but none has both wide availability and demonstrated clinical efficacy in controlled field studies.

Zoonotic Risk:

Some strains of FeLV can be experimentally grown in human tissue cultures, leading to concerns of potential for transmission to people. Studies addressing this concern have shown no evidence that any zoonotic risk exists, and there are no known cases of zoonotic transmission.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Feline infectious peritonitis	**5.1.7**	Merck Veterinary Manual	High	Mod.	Mod.	Neglig.	Mod.

Feline infectious peritonitis (FIP) is an immune-mediated disease triggered by infection with a feline coronavirus (FCoV). However, <5% of FCoV-infected cats develop FIP in multi-cat households.

Epidemiology and Transmission:

FCoV and FIP are a major problem in multi-cat households. The virus is endemic in environments in which many cats are kept together in a confined space (e. g. catteries, shelters, pet stores). FCoV is found less commonly in free-roaming community cats, because they do not typically use the same locations to bury their feces; shared litter boxes are a major source of transmission in multi-cat households.

Zoonotic Risk:

Although coronaviruses shared with animals, such as severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS), are responsible for severe respiratory disease outbreaks in people, there is no indication that FCoV is infectious to people.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Feline Immunodeficiency Virus (FIV)	**5.1.8**	Merck Veterinary Manual Wikipedia Hansen & Holm 2001	Sero-reactors*	High	Mod.	Neglig.	n.a.*

*Three among 115 randomly selected Icelandic cats (2.6%) were found to be sero-positive for FIV. It is possible, however, that the result is due to false positive reactions. Already in 1997, however, an FIV-positive cat had been found at Keldur (Hansen & Holm 2001).

FIV has been identified in domestic and wild felids. The infection is endemic in cats throughout the world. Virus is shed in the saliva, and biting is the principal mode of transmission. As a result, free-roaming, male, and aged cats are at greatest risk of infection. FIV infection is uncommon in closed purebred catteries. Cats with acquired immunodeficiency induced by FIV develop chronic secondary and opportunistic infections of the respiratory, GI (including mouth), and urinary tracts, as well as the skin.

Feline immunodeficiency virus is spread from cat to cat, primarily by biting. Once infected, cats remain infected for life and most eventually have a deterioration of immune function and increased risk of infections.

FIV and HIV are both lentiviruses. However, humans cannot be infected by FIV, nor can cats be infected by HIV. FIV is transmitted primarily through deep bite wounds, where the virus present in the infected cat's saliva enters the body tissues of another cat. FIV+ cats can share water bowls, pellet bowls, eat from the same bowl of wet food, and use the same litter box with low danger of transmitting the disease. A vigilant pet owner who treats secondary infections can allow an infected cat to live a reasonably long life. The chance that an FIV-infected cat will pass the virus to other cats within a household is low, unless there is fighting between cats, or wounds present that could allow entry of the virus from infected to non-infected cat.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Canine Herpesviral Infection	**5.1.9**	Merck Veterinary Manual Lind 2014	High	High	High	Neglig.	High

Lind 2014	Zoonotisk risiko (Hund-> Homo)	Impact hos hund	Impact hos homo	Forekomst (endemicitet)
Herpesvirus	Ingen	Alvorlig	-	Globalt

Etiology and Pathogenesis:

The most significant systemic disease occurs in fetal or neonatal puppies from in utero infection, or infection in the first 3 wks. of life. After this time, natural resistance to infection improves as puppies mature and maintain a higher body temperature. CHV is relatively unstable outside the host, so close contact is required for transmission.

Only canids (dogs, wolves, coyotes) are known to be susceptible. The seroprevalence in dog populations worldwide ranges from 20% to 98% depending on the region. Because latently infected animals may transiently convert to seronegative status, any seroprevalence study likely underestimates the true rate of exposure and carriage.

Clinical Findings:

CHV causes serious disease only in very young puppies. The rapid death and characteristic lesions distinguish it from canine distemper. Deaths due to CHV infection usually occur in puppies 1–3 wks. old, occasionally in puppies up to 1 mo. old, and rarely in pups as old as 6 mo. Typically, onset is sudden, and death occurs after an illness of ≤24 hr. Older dogs exposed to or experimentally inoculated with CHV may develop a mild rhinitis, which may be part of the “kennel cough” syndrome (infectious tracheobronchitis) or a vesicular vaginitis or posthitis. Acutely infected pregnant bitches may abort a litter, or deliver a partially stillborn litter; however, they seldom exhibit other clinical signs, and future breedings are likely to be successful.

Prevention:

Because of the high seroprevalence among adult dogs and because virus may be shed by asymptomatic individuals, complete avoidance of exposure is not a reasonable management strategy for most dogs.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Feline Parvovirus (Panleukopenia)	**5.1.10**	Merck Veterinary Manual	High	High	High	Neglig.	High

Feline Panleukopenia (Feline infectious enteritis, Feline parvoviral enteritis) is a highly contagious, often fatal, viral disease of cats that is seen worldwide. Kittens are affected most severely. The causative parvovirus is very resistant; it can persist for 1 yr. at room temperature in the environment, if protected in organic material. Feline panleukopenia is now diagnosed infrequently by veterinarians, presumably as a consequence of widespread vaccine use. However, infection rates remain high in some unvaccinated cat populations, and the disease occasionally is seen in vaccinated, pedigreed kittens that have been exposed to a high virus challenge.

Etiology, Transmission, and Pathogenesis:

Cats are infected oro-nasally by exposure to infected animals, their feces, secretions, or contaminated fomites. Most free-roaming cats are thought to be exposed to the virus during their first year of life. Those that develop subclinical infection or survive acute illness mount a robust, long-lasting, protective immune response.

Clinical Findings:

Most infections are subclinical, as evidenced by the high seroprevalence of anti-FPV antibodies among unvaccinated, healthy cats. Those cats that become ill are usually <1 yr. old. Per-acute cases may die suddenly with little or no warning (fading kittens). Acute cases show fever (104°–107°F [40°–41.7°C]), depression, and anorexia after an incubation period of 2–7 days. Vomiting usually develops 1–2 days after the onset of fever; it is typically bilious and unrelated to eating. Diarrhea may begin a little later but is not always present. Extreme dehydration develops rapidly. The duration of this self-limiting illness is seldom >5–7 days. Mortality is highest in young kittens <5 mo. old.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Infectious Tracheobronchitis of Dogs (Kennel Cough)	**5.1.11**	Merck Veterinary Manual AHAC8 Lind 2014	High	High	Mod.	Neglig.	Mod.

Lind 2014	**Zoonotisk risiko (Hund-> Homo)**	**Impact hos hund**	**Impact hos homo**	**Forekomst (endemicitet)**
Bordetella bronchiseptica	Lav	Moderat (kennel cough)	Lav	Pan-europæisk

Infectious tracheobronchitis results from inflammation of the upper airways. It is a mild disease that normally improves on its own. However, it can progress to fatal bronchopneumonia in puppies or to chronic bronchitis in weakened, ill, or aged dogs. The disease spreads rapidly among susceptible dogs housed in close confinement such as veterinary hospitals or kennels.

A number of viral and bacterial organisms can cause kennel cough. It is common to have infections with more than one of these organisms at the same time. Stress and environmental changes such as extremes of ventilation, temperature, and humidity appear to increase the dog's susceptibility to disease as well as its severity.

The most common sign is spasms of harsh, dry coughing, which may be followed by retching and gagging. The severity of the cough usually diminishes during the first 5 days, but the disease persists for 10 to 20 days. Affected dogs have few if any additional signs except for some loss of appetite. Body temperature and white blood cell counts usually remain normal. Development of more severe signs, including fever, pus-containing nasal discharge, depression, loss of appetite, and a productive cough, especially in puppies, usually indicates the presence of an additional infection such as distemper or bronchopneumonia. Stress, particularly from adverse environmental conditions and improper nutrition, may contribute to a relapse during recovery.

Tracheobronchitis is usually suspected whenever a dog demonstrates the distinctive harsh cough and has a history of exposure to other susceptible or affected dogs. Laboratory tests are usually normal.

In most cases, affected dogs should not be hospitalized because the disease is highly contagious and because it generally improves on its own.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
***Hepatitis contagiosa canis* (HCC)**	**5.1.12**	Merck Veterinary Manual AHAC8 Lind 2014	High	High	High	Negl.	High

Lind 2014	**Zoonotisk risiko (Hund-> Homo)**	**Impact hos hund**	**Impact hos homo**	**Forekomst (endemicitet)**
Infektiøs hepatitis	Ingen	Alvorlig	-	Globalt

Infectious canine hepatitis is a worldwide, contagious disease of dogs with signs that vary from a slight fever and congestion of the mucous membranes to severe depression, severe reduction in white blood cells, and deficiency of blood clotting. In recent years, the disease has become uncommon in areas where routine vaccination is used.

Infectious canine hepatitis is caused by a virus, canine adenovirus 1. Consumption of urine, feces, or saliva from infected dogs is the most common route of infection. Recovered dogs shed virus in their urine for at least 6 months. The virus targets the liver, kidneys, spleen, and lungs, though other organs are occasionally involved. Long-term kidney damage and clouding of the cornea of the eye ("blue eye") result from immune-complex reactions after recovery from the disease.

Clinical Findings:

It may be difficult to get an infected dog's blood to clot. Respiratory signs may be seen in a few dogs with infectious canine hepatitis. Although central nervous system involvement is unusual, severely infected dogs may develop convulsions from brain damage. Slight paralysis, caused by bleeding in the brain, may also occur. After recovery, dogs eat well but regain weight slowly.

Signs vary from a slight fever to death. The mortality rate ranges from 10%–30% and is typically highest in very young dogs. Concurrent parvoviral or distemper infection worsens the prognosis. The incubation period is 4–9 days. The first sign is a fever of >104°F (40°C), which lasts 1–6 days and is usually biphasic. If the fever is of short duration, leukopenia may be the only other sign, but if it persists for >1 day, acute illness develops.

Signs are apathy, anorexia, thirst, conjunctivitis, serous discharge from the eyes and nose, and occasionally abdominal pain and vomiting. Intense hyperemia or petechiae of the oral mucosa, as well as enlarged tonsils, may be seen. Tachycardia out of proportion to the fever may occur. There may be subcutaneous edema of the head, neck, and trunk. Despite hepatic involvement, there is a notable absence of icterus in most acute clinical cases. On recovery, dogs eat well but regain weight slowly.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Rabies	**5.1.13**	Merck Veterinary Manual, Lind 2014, Høgåsen et al. 2012, Rijks et al. 2016 OIE TAHC 2018, Chapter 8.14 AUS 2012 Corrin & MacDiarmid (1997) MacDiarmid & Corrin (1998).	Low	High	High	High	Low

Lind 2014	Zoonotisk risiko (Hund-> Homo)	Impact hos hund	Impact hos homo	Forekomst (endemicitet)
Rabies	Høj	Alvorlig	Alvorlig	Globalt

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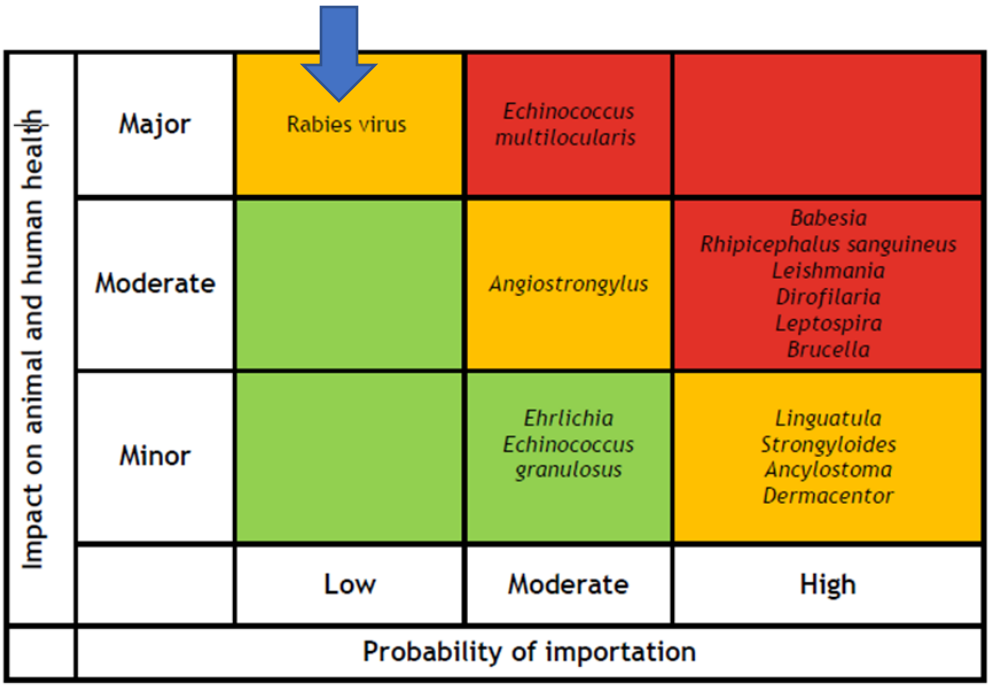


From Høgåsen et al. 2012

AUS 2012 Risk review

This section documents the review of the risk of rabies introduction associated with importing domestic dogs and cats under Australia's existing requirements, and considers whether those risks have changed significantly since the introduction of the existing requirements in 1995. Under Australia's current quarantine policy, many thousands of dogs and cats have been imported without the introduction of rabies virus.

Release

Rabies is widely distributed in the world; few countries are rabies free.

In countries where rabies is endemic in the domestic and wild dog populations, prevalence varies and depends largely on the efficiency, effectiveness and management of the rabies control programs.

In countries and regions where rabies is endemic only in wildlife maintenance hosts (i.e. United States, Canada, western Europe), effective rabies management and control programs including vaccination of domestic dogs and cats, minimise the occurrence of urban rabies. However, cases of urban rabies in unvaccinated dogs and cats occur sporadically due to spillover infections from wildlife.

Cats are less susceptible to rabies than dogs, but all unvaccinated dogs and cats are susceptible to rabies virus. Dogs are recognised as the principal vector for human rabies virus infection in many parts of the world.

Data on the international movement of dogs is limited, but an estimated 25% of dogs imported into the United States in 2006 were either too young to be vaccinated or were not current for rabies vaccination.

The incubation period for rabies generally ranges between three to twelve weeks and in rare cases has exceeded six months. Six months is the internationally recognised incubation period (OIE 2018).

Infected dogs and cats can excrete rabies virus from up to 15 days before clinical signs start to appear, and can continue to excrete virus until they die.

Increased availability of effective vaccines against rabies has facilitated an increase in the international movement of companion animals from rabies-affected countries (Goddard et al. 2010)

Reports of vaccine failure are rare, but instances of vaccine failure should be anticipated for a variety of reasons (e.g. inappropriate vaccine storage, such as 'cold chain failure', incorrect administration of the vaccine and/or immunocompromised vaccine recipients).

Rabies virus neutralising antibody titres are a reliable indicator of postvaccinal immunity; postvaccinal neutralising antibody titres of > 0.5 IU/mL are accepted internationally as an indicator of effective vaccination (OIE TAHC art. 8.14.6).

Taking the preceding factors into consideration, the review concluded that:

- rabies virus control and management has improved in many developed countries, especially in Europe and North America
- canine rabies continues to be a serious problem in many countries in Asia, Africa, the Middle East, and Central and South America
- increased availability of effective vaccines against rabies has facilitated an increase in the international movement of domestic dogs and cats from rabies-affected countries

• the likelihood of rabies virus being present in a dog or cat imported from an approved country has not changed significantly since the introduction of existing quarantine requirements in 1995.

Exposure

Rabies virus is generally transmitted in saliva via the bite of an infected animal or by licking abrasions.

The incubation period for rabies virus is defined as six months (OIE 2011b), but generally ranges from a few days to several months.

Dogs and cats can excrete rabies virus for up to 15 days before they start to show clinical signs; therefore, infected animals may appear healthy and transmit rabies virus to other animals.

All mammals, including humans, are susceptible to infection. Australian exposure groups considered susceptible to rabies virus are mammals including:

- humans
- domestic dogs and cats
- feral canids—foxes, feral dogs
- feral felids—feral cats
- native canids—dingoes
- dasyurids—Tasmanian devils, antechinus, dunnarts and quolls.

The majority of dogs and cats imported into Australia are companion animals kept in relatively secure premises with limited but controlled access to other pets and minimal exposure to feral animals and wildlife. Humans and other companion animals living in the same household as a rabies virus–infected dog or cat are most at risk of exposure.

Inquisitive species, such as cattle or humans, are more likely to be bitten by a rabid animal than timid species.

Local governments (councils) in Australia generally have effective animal control programs to minimise the stray dog population. This reduces uncontrolled contacts that a rabies virus infected dog or cat may have with stray dogs.

Taking the preceding factors into consideration, the review concluded that:

- the likelihood of an Australian exposure group being exposed to an imported dog or cat infected with rabies virus has not changed significantly since the introduction of existing quarantine requirements in 1995.

Consequences—outbreak scenario of establishment and/or spread

In the event that an infected dog or cat incubating rabies virus is imported into Australia, is released from post-arrival quarantine and bites other dogs—especially local stray dogs, wild dogs or dingoes—rabies virus infection may establish and be maintained within the exposure group. Spillover into non-native canid and felid species (foxes, wild dogs and feral cats) may lead to the establishment and spread of rabies virus infection in feral and wild animal populations.

Dogs are the main vector for interspecies rabies virus transmission With 3.41 million pet dogs in Australia), the vast majority of which are not vaccinated against rabies virus, it is plausible that if exposed to a rabies virus–infected dog or cat, rabies virus would establish and spread within Australia's dog population.

Local governments (councils) in Australia generally have animal control programs to control and minimise stray dogs, thus reducing but not eliminating the likelihood of establishment and/or spread via stray dogs.

Dingoes and dasyurids (Tasmanian devil, quoll, antechinus, dunnart) are less abundant than feral canids and felids, but are considered susceptible to spillover infection and may contribute to disease agent establishment and spread. Native wildlife on which canid species occasionally prey (possums, wombats, wallabies, kangaroos) may also be infected but are unlikely to maintain a wildlife rabies virus cycle.
Australia has effective veterinary and human the review concluded that:

- the likelihood of establishment and/or spread of rabies virus in Australia via imported dogs and cats has not changed significantly since the introduction of current biosecurity requirements in 1995.

Consequences—effects of establishment and/or spread

Under Australia's Emergency Animal Disease Response Agreement, variation no. 10/ 02—26.10.10, rabies virus is listed as a Category 1 disease. Category 1 diseases are emergency animal diseases that seriously affect human health and/or the environment (such as depletion of native fauna), but may only have minimal direct consequences to livestock industries.
If rabies virus infection was to establish in free-ranging feral and wild animal populations, the disease may be difficult to eradicate and may become endemic. Vaccination via oral baits has proven to be an effective method of rabies virus control in reservoir populations of some wildlife and feral animals. For example, ongoing implementation of oral-bait vaccination programs has enabled the eradication of rabies virus from the red fox population throughout most of Europe.
Resource intensive, ongoing management and control programs are necessary for managing the threats to public health, animal health (including native fauna) and social amenity associated with endemic rabies virus infection in feral and wild animals.
Taking the preceding factors into consideration, the review concluded that:

- the consequences of establishment and/or spread of rabies virus in Australia have not changed significantly since the introduction of current biosecurity requirements in 1995.

Conclusion

Based on the preceding factors, it was concluded that the overall risk of rabies virus infection associated with the importation of dogs and cats has not changed significantly since the introduction of current biosecurity requirements in 1995.
Due to the significant animal health and public health consequences associated with the introduction and establishment of rabies virus, it was concluded that risk management for rabies virus continues to be warranted for both dogs and cats. It was also concluded that risk management for rabies virus was not warranted for dog or cat semen.

Rabies is an acute, progressive viral encephalomyelitis that principally affects carnivores and bats, although any mammal can be affected. The disease is fatal once clinical signs appear. Rabies is found throughout the world, but a few countries claim to be free of the disease because of either successful elimination programs or their island status and enforcement of rigorous quarantine regulations. Globally, the dog is the most important reservoir, particularly in developing countries. Integrated veterinary management of local animal populations, by mass vaccination of dogs and community promotion of

responsible pet ownership, is the most cost-effective, humane, long-term solution toward eliminating regional canine rabies in a One Health context.

Epidemiology:

From an epidemiologic perspective, the name of the mammalian species acting as the reservoir and vector is used as an adjective to describe involvement in the infection process. For example, rabies maintained by dog-to-dog transmission is termed canine rabies, whereas rabies in a dog as a result of infection with a variant from a different reservoir mammal, e.g., skunk (or raccoon or fox), would be referred to as skunk (or raccoon or fox, etc.) rabies in a dog.

Rabies emergence may be affected by changes in virus-host dynamics or human translocation of infected species.

In western Europe, red fox rabies predominated before its elimination by oral vaccination. In parts of eastern Europe, rabies in raccoon dogs is of increasing concern. Bat rabies, maintained by several different lyssaviruses in insectivorous *Chiroptera*, appears to be widely distributed throughout Europe.

All rabies reservoirs are also vectors of the virus, but not all vectors are reservoirs. For example, cats can effectively transmit the virus, but no cat-to-cat transmission of rabies perpetuates in lieu of a predominant reservoir (such as infected dogs), and no unique feline rabies virus variant has been documented. However, cats are the most commonly reported rabid domestic animal in the USA. Virus is present in the saliva of rabid cats, and people have developed rabies after being bitten by rabid cats. Reported cases in domestic cats have outnumbered those in dogs in the USA every year since 1990.

Transmission and Pathogenesis:

Lyssaviruses are highly neurotropic. Transmission almost always occurs via introduction of virus-laden saliva into tissues, usually by the bite of a rabid animal. Although much less likely, virus from saliva, salivary glands, or brain can cause infection by entering the body through fresh wounds or intact mucous membranes. Usually, saliva is infectious at the time clinical signs occur, but domestic dogs, cats, and ferrets may shed virus for several days before onset of clinical signs. Viral shedding in skunks has been reported for up to 8 days before onset of signs.

The incubation period is both prolonged and variable. Typically, the virus remains at the inoculation site for a considerable time. The unusual length of the incubation period helps to explain the effective action of local infiltration of rabies immune globulin during human postexposure prophylaxis, even days after exposure. Most rabies cases in dogs develop within 21–80 days after exposure, but the incubation period may be shorter or considerably longer. One recorded case of rabies in a person in the USA had an incubation period estimated reliably of >8 yr.

The virus travels via the peripheral nerves to the spinal cord and ascends to the brain. After reaching the brain, the virus travels via peripheral nerves to the salivary glands. If an animal is capable of transmitting rabies via its saliva, virus will be detectable in the brain. Virus is shed intermittently in the saliva.

Near the end of the clinical phase, after replication in the CNS, virus may be found in nearly every innervated organ. Rabies has been transmitted by transplantation of tissues and organs from infected people.

Clinical Findings:

Clinical signs of rabies are rarely definitive. Rabid animals of all species usually exhibit typical signs of CNS disturbance, with minor variations among species. The most reliable signs, regardless of species, are acute behavioral changes and unexplained progressive paralysis. Behavioral changes may include sudden anorexia, signs of apprehension or nervousness, irritability, and hyperexcitability (including priapism). The animal may seek solitude. Ataxia, altered phonation, and changes in temperament are apparent. Uncharacteristic aggressiveness may develop—a normally docile animal may suddenly become vicious. Commonly, rabid wild animals may lose their fear of people, and normally nocturnal species may be seen wandering about during the daytime.

Diagnosis:

Clinical diagnosis is difficult, especially in areas where rabies is uncommon, and should not be relied on when making public health decisions. In the early stages, rabies can easily be confused with other diseases or with normal aggressive tendencies. Therefore, when rabies is suspected and definitive diagnosis is required, laboratory confirmation is indicated. Suspect animals should be euthanized, and the head removed for laboratory shipment.

Rabies diagnosis should be done by a qualified laboratory, designated by the local or state health department in accordance with established standardized national protocols for rabies testing. Immunofluorescence microscopy on fresh brain tissue, which allows direct visual observation of a specific antigen-antibody reaction, is the current test of choice. When properly used, it can establish a highly specific diagnosis within a few hours. Brain tissues examined must include medulla oblongata and cerebellum (and should be preserved by refrigeration with wet ice or cold packs). Virus isolation by the mouse inoculation test or tissue culture techniques using mouse neuroblastoma cells may be used for confirmation of indeterminate fluorescent antibody results, but it is no longer in common use in the USA.

Prevention and Control:

Comprehensive guidelines for control in dogs have been prepared internationally by the World Health Organization and in the USA by the National Association of State Public Health Veterinarians (NASPHV). They include the following: 1) notification of suspected cases, and euthanasia of dogs with clinical signs and dogs bitten by a suspected rabid animal; 2) reduction of contact rates between susceptible dogs by leash laws, dog movement control, and quarantine; 3) mass immunization of dogs by campaigns and by continuing vaccination of young dogs; 4) stray dog control and euthanasia of unvaccinated dogs with low levels of dependency on, or restriction by, people; and 5) dog registration.

The OIE TAHC chapter 8.14, articles 4 and 6 contain recommendations to veterinary authorities on how to import dogs and cats from countries considered free from and infected with rabies, respectively. The latter recommendations include either vaccination and subsequent testing and titration for antibodies, or

quarantine for 6 months before shipment. These procedures have been endorsed e.g. by New Zealand, as discussed and documented by Corrin & MacDiarmid (1997) and MacDiarmid & Corrin (1998).

Management of Suspected Rabies Cases—Exposure of Pets:

Where terrestrial wildlife or bat rabies is known to occur, any animal bitten or otherwise exposed by a wild, carnivorous mammal (or a bat) not available for testing should be regarded as having been exposed to rabies. The NASPHV recommends that any unvaccinated dog, cat, or ferret exposed to rabies be euthanized immediately. If the owner is unwilling to do this, the animal should be placed in strict isolation (i.e., no human or animal contact) for 6 mo. and vaccinated against rabies 1 mo. before release. If an exposed domestic animal is currently vaccinated, it should be revaccinated immediately and closely observed for 45 days.

Zoonotic Risk:

Rabies has the highest case fatality of any infectious disease. When a person is exposed to an animal suspected of having rabies, the risk of rabies virus transmission should be evaluated carefully. Risk assessment should include consideration of the species of animal involved, the prevalence of rabies in the area, whether exposure sufficient to transmit rabies virus occurred, and the current status of the animal and its availability for diagnostic testing. Wild carnivores and bats present a considerable risk where the disease is found, regardless of whether abnormal behavior has been observed. Insectivorous bats, though small, can inflict wounds with their teeth and should never be caught or handled with bare hands. Bat bites may be ignored or go unnoticed, so direct contact with bats could be considered a risk of virus exposure. Any wild carnivore or bat suspected of exposing a person to rabies should be considered rabid unless proved otherwise by laboratory diagnosis; ideally, this includes bats in direct contact with people, such as those found in rooms with sleeping or otherwise unaware persons. Wildlife, including wolf hybrids, should never be kept as pets; if one of those animals exposes a person or domestic animal, the wild animal should be managed like free-ranging wildlife.

Any healthy domestic dog, cat, or ferret, whether vaccinated against rabies or not, that exposes (bites or deposits saliva in a fresh wound or on a mucous membrane) a person should be confined for 10 days; if the animal develops any signs of rabies during that period, it should be euthanized and its brain promptly submitted for rabies diagnosis. If the dog, cat, or ferret responsible for the exposure is stray or unwanted, it may be euthanized as soon as possible and submitted for rabies diagnosis. Since the advent of testing by immunofluorescence microscopy, there is no value in holding such animals to “let the disease progress” as an aid to diagnosis.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Feline rhinotracheitis	**5.1.14**	Merck Veterinary Manual	High	High	Mod.	Negl.	Mod.

The majority of feline upper respiratory infections are caused by feline herpesviral rhinotracheitis, although the incidence of feline calicivirus may be higher in some populations of cats. Infection with both these viruses at once may occur. Natural transmission of these agents occurs through small droplets in the air (such as from a sneeze) and contaminated objects, which can be carried to a susceptible cat by a handler. Recovering cats may harbor the virus for many months. Stress may trigger a relapse.

The onset of **feline herpesviral rhinotracheitis** is marked by fever, frequent sneezing, inflamed eyes (conjunctivitis), rhinitis, and often salivation. Excitement or movement may cause sneezing. The fever may reach 105°F (40.5°C) but subsides and then may come and go. Initially, the disease causes a clear discharge from the nose and eyes; it soon increases in amount and contains mucus and pus. At this point, depression and loss of appetite become evident. Severely affected cats may develop mouth inflammation with sores, and inflammation of the cornea occurs in some cats.

Signs may persist for 5 to 10 days in milder cases and up to 6 weeks in severe cases. The outlook is generally good except for young kittens and older cats. When the illness is prolonged, weight loss may be marked. Bacteria often infect cats that are already ill with feline herpes-viral rhinotracheitis.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Canine parvovirus	**5.1.15**	Merck Veterinary Manual Lind 2014 AHAC8	High	High	High	Neglig.	High

Lind 2014	Zoonotisk risiko (Hund-> Homo)	Impact hos hund	Impact hos homo	Forekomst (endemicitet)
Parvovirus	Ingen	Alvorlig	-	Globalt

Canine parvovirus (CPV) is a highly contagious and relatively common cause of acute, infectious GI illness in young dogs. Although its exact origin is unknown, it is believed to have arisen from feline panleukopenia virus or a related parvovirus of nondomestic animals. Infectious CPV can persist indoors at room temperature for at least 2 mo.; outdoors, if protected from sunlight and desiccation, it can persist for many months and possibly years.

Young (6 wks. to 6 mo.), unvaccinated or incompletely vaccinated dogs are most susceptible. Rottweilers, Doberman Pinschers, American Pit Bull Terriers, English Springer Spaniels, and German Shepherds have been described to be at increased risk of disease. Among dogs >6 mo. old, intact male dogs are more likely than intact female dogs to develop CPV enteritis.

Clinical Findings:

Clinical signs of parvoviral enteritis generally develop within 5–7 days of infection but can range from 2–14 days. Initial clinical signs may be nonspecific (e.g., lethargy, anorexia, fever) with progression to vomiting and hemorrhagic small-bowel diarrhea within 24–48 hr. Although CPV-associated leukoencephalomalacia has been reported, CNS signs are more commonly attributable to hypoglycemia, sepsis, or acid-base and electrolyte abnormalities. Inapparent or subclinical infection is common.

Diagnosis:

Most clinically ill dogs shed large quantities of virus in the feces. However, false-negative results can be seen early in the course of the disease (before peak viral shedding), because of the dilutional effect of large volume diarrhea, or after the rapid decline in viral shedding that tends to occur within 10–12 days of infection. False-positive results can be seen within 4–10 days of vaccination with modified-live CPV vaccine. Alternative ways to detect CPV antigen in feces include PCR testing, electron microscopy, and virus isolation. Serodiagnosis of CPV infection requires demonstration of a 4-fold increase in serum IgG titer throughout a 14-day period or detection of IgM antibodies in the absence of recent (within 4 wks.) vaccination.

Treatment and Prognosis:

The main goals of treatment for CPV enteritis include restoration of fluid, electrolyte, and metabolic abnormalities and prevention of secondary bacterial infection. Most puppies that survive the first 3–4

days of illness make a full recovery, usually within 1 wk. With appropriate supportive care, 68%–92% of dogs with CPV enteritis will survive. Dogs that recover develop long-term, possibly lifelong immunity.

CPV can remain viable in the environment for an extended period. In a kennel, shelter, or hospital situation, cages and equipment should be cleaned, disinfected, and dried twice before reuse. The same concepts can be applied to a home situation. Removal of contaminated organic material is important in outdoor situations where complete disinfection is not practical. Disinfectants can be applied outdoors with spray hoses, but disinfection will be less effective than when applied to clean, indoor surfaces. In a home situation, only fully vaccinated puppies (at 6, 8, and 12 wks.) or fully vaccinated adult dogs should be introduced into the home of a dog recently diagnosed with CPV enteritis. Booster vaccination of in-contact healthy dogs that are up-to-date on parvovirus vaccination is reasonable but potentially unnecessary given the extended duration of immunity to CPV.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Foot-and-Mouth Disease	**5.1.16**	Merck Veterinary Manual AHAC8	Very low	Very low	Neglig.	Neglig.	Neglig.

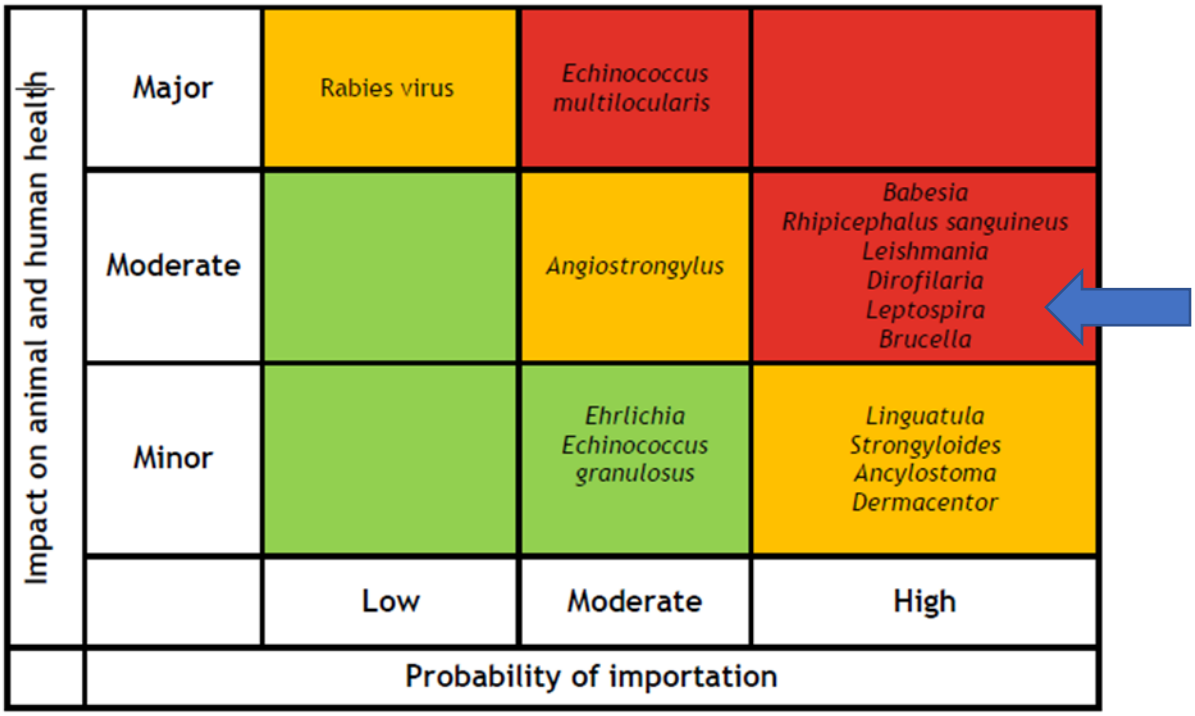
The virus is transmitted via direct or indirect contact with infected secretions and excretions (including semen and milk), mechanical vectors (people, horses, dogs, cats, birds, vehicles), and air currents over land or water.

FootAndMouthDiseaseInfo.org

Q: Can my cat or dog contract FMD?
A: No, dogs and cats cannot become infected with FMD; however, they are capable of spreading the disease

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Canine leptospirosis	**5.2.1.**	Merck Veterinary Manual, AHAC8, AUS 2012, Lind 2014, Høgåsen et al. 2012, Rijks et al. 2016	High	Moderate	Moderate /High	Moderate	Moderate /High

Lind 2014	**Zoonotisk risiko (Hund-> Homo)**	**Impact hos hund**	**Impact hos homo**	**Forekomst (endemicitet)**
Canine leptospirosis	Ingen	Alvorlig	-	Globalt



From Høgåsen et al. 2012

AUS 2012 Risk review

Regarding the importation of dogs and cats, the following key factors were considered relevant to the biosecurity risk presented by *L. interrogans* sv. Canicola:

• Due to the widespread international distribution of leptospires, leptospirosis was removed from the OIE list of diseases in 2012. Leptospirosis is not a nationally notifiable animal disease in Australia.
• Canicola serovars occur in Australia. Hence, it is not certain that *L. interrogans* sv. Canicola is exotic, although it has not been detected in dogs in Australia.
• *L. interrogans* sv. Canicola is typically associated with renal dysfunction. Severe disease, including fatal infection, is more likely to occur in young dogs.
• Dogs are the recognised maintenance host for *L. interrogans* sv. Canicola.
• There are a number of leptospiral serovars that are endemic in Australia that cause severe and sometimes fatal infection in dogs.
• Vaccination against *L. interrogans* sv. Canicola is of moderate efficacy in preventing clinical disease, but does not provide a sterile immunity. Vaccinated dogs may carry and shed *L. interrogans* sv. Canicola.
• Vaccination of toy breeds against *L. interrogans* sv. Canicola is not recommended, because these breeds are reported to be at an increased risk of adverse vaccination reactions.
• Serology is not a highly sensitive diagnostic tool for determining infection status. Dogs that are not vaccinated against *L. interrogans* sv. Canicola and return negative serology results may still carry and shed *L. interrogans* sv. Canicola.
• Antibiotic treatment of clinically healthy dogs with positive serology for *L. interrogans* sv. Canicola is not reliable in eliminating a carrier status.

Conclusion Based on the preceding factors, it was concluded that risk management for *L. interrogans* sv. Canicola continues to be warranted for dogs.

The following measures were proposed as appropriate risk management options.

Pre-export measures (dogs)

Vaccination

• Dogs must be fully vaccinated with an approved vaccine against *L. interrogans* sv. Canicola. Full vaccination requires:
– initial vaccination comprising two vaccinations a minimum of four weeks apart and
– annual re-vaccination.
• The annual revaccination or final vaccination of the initial course must be administered between 12 months and 14 days before export to Australia.

OR

Serology

• Within 30 days of export, dogs must be tested for *L. interrogans* sv. Canicola infection by the MAT and have a negative result.
• A negative result is defined as less than 50% agglutination at a serum dilution of 1:100.

It is extremely important for veterinarians to maintain a high index of suspicion for leptospirosis, because this is a zoonotic disease and has a wide range of clinical presentations in dogs. Any age, breed, or sex of dog is susceptible to leptospirosis, and the diagnosis should not be excluded on the basis of signalment or

lifestyle. Canine leptospirosis is not restricted to large-breed dogs, male dogs, or dogs with a predominantly outdoor lifestyle.

Dogs are the maintenance host for *Leptospira interrogans* serovar *Canicola*, and before widespread vaccination programs, serovars *Canicola* and *Icterohaemorrhagiae* were the most common serovars in dogs. The serovars that cause disease in dogs are likely to vary with geographic region and the presence of reservoir hosts. Unfortunately, current understanding of the serovars that cause natural disease in dogs is limited by the fact that isolation of leptospires is rarely performed; thus, studies to date have relied on serologic data. Experimental infections and isolation of organisms from a small number of sick dogs have shown that serovars *Icterohaemorrhagiae, Canicola, Autumnalis, Pomona, Bratislava, Sejroe, and Ballum* are capable of causing disease in dogs. Knowledge of the infecting serovar in dogs is essential for epidemiologic studies and vaccine development; it is less important for clinicians managing individual cases.

Prevention:

Avoidance of exposure to free-ranging wildlife and domestic animals that may be maintenance hosts for *Leptospira* is difficult because rodents, raccoons, opossums, and skunks are frequently found in rural and urban environments (Prevalence in Iceland ???). The cornerstone of leptospirosis prevention is vaccination with polyvalent inactivated vaccines. Immunity to leptospirosis is believed to be serovar specific and, therefore, vaccines are formulated for various species to include the relevant serovars. There are currently no leptospiral vaccines for horses. Leptospiral vaccines are generally designed and evaluated for the ability to prevent clinical signs of disease, although some vaccines have also been shown to significantly reduce renal colonization and urine shedding.

Zoonotic Risk:

People are susceptible to infection with most of the pathogenic serovars of *Leptospira* but are incidental hosts and, therefore, not important reservoirs of infection. Occupational exposure is a risk factor, and veterinarians, veterinary staff, livestock producers, and dairy workers are at increased risk. In addition, recreational exposure to waters contaminated with urine of domestic animals or wildlife presents a risk. Animal owners have contracted leptospirosis via contact with infected companion animals and livestock.

Because leptospirosis is a zoonotic disease, all veterinary personnel should take appropriate precautions when handling known or suspected infected animals. Such dogs do not need to be placed in isolation but should be nursed with barrier precautions, paying particular attention to avoiding exposure of skin or mucous membranes to urine or blood. Infected dogs should be allowed to urinate in designated areas that can subsequently be cleaned and disinfected. The organisms are killed by all commonly used disinfectants. Owners of dogs recently diagnosed with leptospirosis should be advised of the zoonotic nature of the disease and contact their physicians with any health concerns. Owners should wear gloves when cleaning up urine and should wash their hands after handling the dog, at least until the course of antibiotic therapy is completed.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Melioidosis	**5.2.2.**	Merck Veterinary Manual	Very low	Low	Moderate	Moderate	Very low

Melioidosis is an uncommon bacterial infection of humans and animals. The disease-causing agent is *Burkholderia pseudomallei,* which occurs in the soil throughout southeast Asia, northern Australia, and the South Pacific. Melioidosis has been diagnosed in many animals, including cats and humans. Cats may succumb to infection due to a weakened immune system. The most common routes of infection are via skin inoculation, contamination of wounds, ingestion of soil or contaminated carcasses, or inhalation.

Infection may be associated with single or multiple curd-like nodules or abscesses, which can be located in any organ. Pneumonia is the most common form of the disease in both animals and humans. Lameness can occur. It is possible for an infection to lie dormant before becoming apparent. Death may result in animals with sudden and intense infections or when vital organs are affected. Treatment with antibiotics can be expensive and prolonged.

Host susceptibility and disease manifestations vary between species. The introduction of naive livestock to endemic regions may predispose them to disease, as seen with sheep, goats, pigs, and camelids. Other species (e.g. dogs and cats) may succumb to infection due to immunocompromising conditions.

Transmission:

Infection is thought to be opportunistic and primarily a result of transmission from the environment (e.g., contaminated soil and surface waters) rather than from animal to animal. The most common routes of infection are via percutaneous inoculation, contamination of wounds, ingestion of soil or contaminated carcasses, or inhalation.

Treatment and Prevention:

Treatment with the appropriate antibiotics should be based on culture and sensitivity results. Treatment may be expensive, prolonged, and possibly unsuccessful, with the risk of recrudescence once treatment is discontinued. The possibility of underlying immunosuppressive conditions should be investigated in less susceptible species. Minimization of environmental contamination by diseased animals is also an important control measure. There is no effective vaccine.

Zoonotic Risk:

Melioidosis has zoonotic potential. Infected animals can shed the organism in wound exudates and, depending on the site of infection, from other sources, including nasal secretions, milk, feces, and urine.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Murine typhus	**5.2.3.**	Merck Veterinary Manual	Low	Very low	Moderate	Moderate	Very low

Rickettsia typhi, the causative agent of murine typhus, and *R felis* are zoonotic pathogens maintained primarily in rodent reservoirs (rats, mice) that may also be associated with enzootic cycles involving opossums and domestic cats. Infection is transmitted to people and other animals through contact with infected fleas. The cat flea is considered absent from Iceland.

Epidemiology:

Infection in people is primarily thought to occur through exposure of abraded skin with infectious flea feces; aerosolization of infectious materials may occur in limited settings. Dogs and cats are presumably exposed in a similar fashion. Although known to occur worldwide, currently fewer than several hundred human cases of murine typhus are reported in the USA each year.

Clinical Findings:

Clinical illness associated with canine and feline infection with R typhi and R felis is not well documented, but evidence of exposure based on presence of anti-rickettsial antibodies has been noted, particularly in association with outbreaks of human disease. Although a role as a possible reservoir for infection has been suggested, particularly for cats, the importance of domestic animals in maintenance of enzootic cycles has not been well elucidated. Nonetheless, dogs and cats may, at a minimum, serve as a source of fleas that may pose a transmission risk to people. Regular flea control is recommended to reduce risk of flea-associated transmission to people.

Zoonotic Risk:

R typhi is considered a zoonotic pathogen. Serologic evidence of exposure or past infection in dogs or cats indicates a heightened risk of human infections in a given area, and flea control for pets is an essential component of disease control.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Plague	**5.2.4.**	AUS 2012	Low	Very low	Moderate	Moderate	Very low

AUS 2012 Risk review

Regarding the importation of dogs and cats, the following key factors were considered relevant to the biosecurity risk presented by *Y. pestis*:

- Dogs are relatively resistant to infection by *Y. pestis*. Clinically healthy dogs are considered to pose a negligible risk of harbouring and transmitting *Y. pestis*.
- Cats are susceptible and there is a short incubation period between infection and expression of clinical disease (1–3 days).
- Cats are more likely than dogs to be severely affected and infection may be fatal in cats.
- Cats that are clinically healthy at pre-export inspection and remain clinically healthy during post-arrival quarantine are considered to pose a negligible risk of harbouring and transmitting *Y. pestis*.

Conclusion

Based on the preceding factors, it was concluded that risk management for *Y. pestis* continues to be warranted for dogs and cats. It was also concluded that risk management for *Y. pestis* is not warranted for dog or cat semen.

The following measures were proposed as an appropriate risk management option for dogs and cats.

Pre-export measures

Vector management

- As per pre-export measures proposed for external parasite control

Post-arrival measures

Vector management

- As per post-arrival measures proposed for external parasite control

Clinical signs

Flea bites allow for the bacteria to pass the skin barrier, but fleas are considered not present in Iceland, yet they have been found on imported dogs and cats. Dogs can develop mild to moderate pyrexia shortly after infection and recover clinically without treatment within about a week. Cats develop pyrexia, lethargy, anorexia, lymphadenopathy, abscesses, bacteraemia, septicaemia or pneumonia. The mortality rate in affected cats can be 50%.

Diagnosis

Y. pestis is usually found in large numbers in infected tissues. Aseptically collected specimens of fluids, tissues, lymph node aspirates or blood should be submitted for culture. Examination via direct fluorescent antibody technique of air-dried impression smears of affected tissues provides a rapid, presumptive diagnosis with good reliability. Serology is often unreliable in detecting the presence of infection early in the course of disease. A passive haemagglutination assay is used to detect antibody specific to the F1 antigen of *Y. pestis.* A four-fold rise in serial titres from specimens collected 10–14 days apart should be

used to enable active infection to be differentiated from previous exposure because dogs and cats in endemic areas frequently have high titres that persist for more than 12 months.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Rocky Mountain spotted fever	**5.2.5.**	Merck Veterinary Manual AUS 2012	High	Very low	Low/ Moderate	Moderate/ High	Very low

AUS 2012 Risk review

Removed. Dogs and cats do not have a significant role in disease epidemiology. The main vector of RMSF (*Dermacentor* ticks) is not present in Australia and, although a vector (*Rhipicephalus sanguineus*) for RMSF and MSF is present in Australia, it is not regarded as the primary vector for RMSF in the US.

Rocky Mountain spotted fever (RMSF) is a disease of people and dogs caused by *Rickettsia rickettsii*. *R rickettsii* and closely related members of the spotted fever group of rickettsiae are considered endemic throughout much of North, South, and Central America. These pathogens are transmitted primarily through the bites of infected ticks. The ability of genetically similar rickettsial organisms, such as *R parkeri*, to cause clinically similar disease in dogs is unknown. Because of their susceptibility to *R rickettsii* and relatively higher rates of tick exposure, dogs may serve as excellent sentinels of risk for *R rickettsii* infection in people. Clusters of disease are frequently reported in defined geographic areas, and temporally associated infections may be seen in both dogs and their owners.

Epidemiology:

In the USA, *Dermacentor variabilis* (the American dog tick) and *D andersoni* (the Rocky Mountain wood tick) are considered the primary vectors for *R rickettsii*. In South America, several *Amblyomma* spp of ticks have been implicated in transmission. The organism has also been isolated from *Rhipicephalus sanguineus* ticks (the brown dog tick), which appear to be the primary vector in some focal areas of Arizona, particularly on American Indian tribal lands, and may also play an as-yet unappreciated role in outbreaks elsewhere in the USA. *R sanguineus* ticks are also associated with transmission of *R rickettsii* in Central America and with large city-based outbreaks in Mexico, and also with *R. conorii* in the Mediteranian region, which may cause Mediterranean Spotted Fever. The pathogen is acquired by larval and nymph stages of ticks while feeding on infected vertebrate hosts and is also passed from female ticks to progeny through transovarial transmission. An estimated <1% of *Dermacentor* spp ticks carry *R rickettsii*, even in areas considered highly endemic. In highly enzootic regions of Arizona where *R rickettsii* is transmitted by the brown dog tick, as many as 5% of ticks may be infected.

Seroprevalence in dogs from endemic areas ranges from 4.3%–77%, but these values do not accurately reflect infection rates because of the detection of cross-reacting antibodies to other genetically similar rickettsiae. RMSF transmission through blood transfusion has been documented in a single human case and should be considered when selecting canine blood donors. Direct transmission from dogs to people

has not been reported, although human infection may occur after contact of abraded skin or conjunctiva with tick hemolymph or excreta during removal of engorged ticks from pets.

Treatment:

Antibiotic treatment should be administered based on clinical suspicion without waiting for results of serologic tests, because delayed administration of antibiotics may result in higher rates of severe or fatal outcome. Supportive care for dehydration and hemorrhagic diathesis may be necessary.

Precautions should be taken for the safe removal and control of ticks. In settings in which *R rickettsii* transmission from *R sanquineus* is suspected, medications and products with proven efficacy against this tick species are important to use. Because *R sanguineus* infestations can be problematic in kennels and around homes, and long-term tick control is needed for outbreak control, use of effective long-acting tick collars on all susceptible dogs might be considered.

Zoonotic Risk:

R rickettsii is considered a zoonotic pathogen. The potential for household clustering and large urban outbreaks, particularly in areas with transmission by brown dog ticks, makes RMSF a disease of significant public health concern. Although clinical disease occurs in both animals and people, the involvement of a required intermediate tick vector for transmission means dogs and other infected animals do not pose a direct transmission risk in normal circumstances. Infection in dogs indicates a heightened risk of human infections related to tick exposure in a given area, and serologic studies of dogs in emerging areas may help predict human risk of infection. Particularly in areas where transmission occurs via *R sanguineus*, close cooperation between veterinary, medical, and public health officials is important to achieve control.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Salmonellosis	**5.2.6.**	Merck Veterinary Manual Lind 2014	High	Low	Moderate	Moderate	Low

Lind 2014	**Zoonotisk risiko (Hund-> Homo)**	**Impact hos hund**	**Impact hos homo**	**Forekomst (endemicitet)**
Salmonella	Lav	Moderat	Moderat	Pan-europæisk

Salmonellosis in warm-blooded vertebrates is in most cases associated with serovars of *Salmonella enterica*. The most common type of infection is the carrier state, in which infected animals carry the pathogen for a variable period of time without showing any clinical signs. Clinical disease is characterized by two major syndromes: a systemic septicemia (also termed as typhoid) and an enteritis. Other less common clinical presentations include abortion, arthritis, respiratory disease, necrosis of extremities, and meningitis.

Many **dogs** and **cats** are asymptomatic carriers of salmonellae. Clinical disease is uncommon, but when it is seen, it is often associated with hospitalization, another infection or debilitating condition in adults, or exposure to large numbers of the bacteria in puppies and kittens, in which enteritis may be common.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Tularemia	**5.2.7.**	Merck Veterinary Manual AUS 2012	Low	Low	Moderate	High	Low

AUS 2012 Risk review

Regarding the importation of dogs and cats, the following key factors were considered relevant to the biosecurity risk presented by *F. tularensis*:

- *F. tularensis* is widespread in the Northern Hemisphere.
- *F. tularensis* is OIE-listed and nationally notifiable in Australia.
- Both dogs and cats may be infected, but are not considered significant in the transmission of disease.
- Infected ticks are a known source of transmission of *F. tularensis* to dogs and cats.

Conclusion

Based on the preceding factors, it was concluded that risk management for *F. tularensis* continues to be warranted in dogs and cats. It was also concluded that risk management for *F. tularensis* is not warranted for dog or cat semen.

The following measures were proposed as an appropriate risk management option for dogs and cats.

Pre-export measures

Vector management

- As for pre-export measures proposed for external parasite control.

Post-arrival measures

Vector management

- As for post-arrival measures proposed for external parasite control.

Tularemia is a bacterial septicemia that affects >250 species of wild and domestic mammals, birds, reptiles, fish, and people. *Francisella tularensis* is listed as a category A bioterrorism agent because of the potential for fatality, airborne dissemination, and societal disruption if released.

Epidemiology and Transmission:

Tularemia is a relatively rare bacterial disease of birds, animals, and people and is found throughout the Northern Hemisphere. It is also called 'rabbit fever. Among domestic animals, sheep are the most common host, but clinical infection has also been reported in cats, dogs, pigs, and horses. Cats are at increased risk because of predatory behavior and appear to have an increased susceptibility, whereas cattle appear to be resistant. Little is known of the true incidence and spectrum of clinical disease in domesticated animals. Important wild animal hosts for *F tularensis tularensis* include cottontail and jackrabbits, whereas the

most common vectors are the ticks *Dermacentor andersoni* (the wood tick), *Amblyomma americanum* (the lone star tick), *D variabilis* (the American dog tick), and *Chrysops discalis* (the deer fly). Animal hosts of *F tularensis holarctica* are lagomorphs, beaver, muskrat, voles, and sheep. Ticks, flies, fleas, and exposure to contaminated water sources are all associated with transmission of this subspecies, which has also been found to persist naturally in a water-associated amoeba.

Tularemia can be transmitted by aerosol, direct contact, ingestion, or arthropods. Inhalation of aerosolized organisms (in the laboratory or as an airborne agent in an act of bioterrorism) can produce a pneumonic form. Direct contact with, or ingestion of, infected carcasses of wild animals (e.g., cottontail rabbit) can produce the ulcero-glandular, oculo-glandular, oropharyngeal (local lesion with regional lymphadenitis), or typhoidal form. Immersion in or ingestion of contaminated water can result in infection in aquatic animals. Ticks can maintain infection trans-stadially and trans-ovarially, making them efficient reservoirs and vectors.

The most common source of infection for people and herbivores is the bite of an infected tick, but people who prepare or eat improperly cooked wild game are also at increased risk. Dogs, cats, and other carnivores may acquire infection from ingestion of an infected carcass. Case reports have implicated cats as a source of infection in people.

Tularemia must be differentiated from other septicemic diseases (especially plague and pseudotuberculosis) or acute pneumonia. When large numbers of sheep show typical signs during periods of heavy tick infestation, tularemia or tick paralysis should be suspected. Tularemia should be considered in cats with signs of acute lymphadenopathy, malaise, oral ulcers, and history of recent ingestion of wild prey.

Diagnosis of acute infection is confirmed by culture and identification of the bacterium, direct or indirect fluorescent antibody test, or a 4-fold increase in antibody titer between acute and convalescent serum specimens. A single titer of ≥1:80 by the tube agglutination test is presumptive evidence of prior infection. When tularemia is suspected, laboratory personnel should be alerted as a precaution to reduce the risk of laboratory-acquired infection. In some jurisdictions, tularemia in animals is reportable to public health authorities.

Treatment and Control:

Streptomycin, gentamicin, and tetracyclines are effective at recommended dose levels. Gentamicin should be continued for 10 days. Because tetracycline and chloramphenicol are bacteriostatic, they should be continued for 14 days to minimize the risk of relapse. Early treatment is important to minimize risk of fatality. Because of the substantial sylvatic (wildlife and tick) component of the *Francisella* life cycle, control is limited to reducing arthropod infestation and to rapid diagnosis and treatment.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Tuberculosis	**5.2.8**	Merck Veterinary Manual	Low	Low	Moderate	High	Low

Tuberculosis is an infectious disease caused by bacteria of the genus *Mycobacterium*. The disease affects practically all species of vertebrates, and, before control measures were adopted, was a major disease of humans and domestic animals. Signs and lesions are generally similar in the various species.

Three main types of tubercle bacilli are recognized: human (*Mycobacterium tuberculosis*), bovine (*Mycobacterium bovis*), and avian (*Mycobacterium avium*). The 2 mammalian types are more closely related to each other than to the avian type. Each of the types may produce infection in other host species.

Cats: Tuberculosis is uncommon in cats. Cats are quite resistant to infection with *Mycobacterium tuberculosis* but are susceptible to *M bovis*, *M avium* complex, or *M microti*. *M lepraemurium* has been isolated from granulomatous lesions in the skin. Some unclassified acid-fast bacilli have also been isolated. Contaminated milk causing GI tract lesions, typically in the mesenteric lymph nodes, is the most common circumstance, and historically this was responsible for a very high percentage of tuberculous cats in Europe. Rapid, hematogenous dissemination to other organs, including the lungs and regional lymph nodes, can occur. Infected skin or deeper wounds sometimes give rise to tuberculous sinuses. Lesions have a central area of necrosis, usually without calcification. The tuberculin skin test is considered unreliable in cats. Diagnosis may be assisted by radiography and ELISA. Identification of the organism is necessary to confirm a diagnosis. Efficacious treatment protocols are not available. Therefore, it is recommended that cats infected with *M bovis* be euthanized because of public health concerns.

Dogs: Most infected dogs do not have any signs, as the canine immune system actively suppresses the bacteria. When disease does occur, signs generally include chronic coughing with difficulty breathing or quick, shallow breaths. Other generalized signs include progressive emaciation, lethargy, weakness, poor appetite, and a low-grade, fluctuating fever. The disease is easily transmitted to humans and other animals and represents a public health risk. Therefore, treatment of tuberculosis in dogs should be discussed with your veterinarian. If a dog is suspected of having advanced tuberculous lesions, it must be reported to the appropriate public health authorities, and the dog should be euthanized.

Dogs may be infected with *Mycobacterium tuberculosis*, *M bovis*, and occasionally with *M avium* complex or *M fortuitum*, commonly from a human or bovine source. Tuberculous lesions are usually found in the lungs, liver, kidney, pleura, and peritoneum; they have a gray appearance, usually with a noncalcified, necrotic center. Lesions are often exudative and can produce a large quantity of straw-colored fluid in the thorax. False-negative tuberculin tests are often seen in dogs. Radiographs and a thorough history are useful in diagnosis. Treatment is not often recommended. Affected dogs in close contact with people should be euthanized because of public health concerns.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Anthrax	**5.2.9**	Merck Veterinary Manual	Very low	Very low	High	High	Very low

Anthrax is an often-fatal infectious disease that may infect all warm-blooded animals, including dogs, cats and humans. Anthrax is caused by infection with bacteria known as *Bacillus anthracis*. This bacterium forms spores, which make it extremely resistant to environmental conditions such as heating, freezing, chemical disinfection, or dehydration that typically destroy other types of bacteria. Thus, it can persist for a long time within or on a contaminated environment or object. Livestock may consume the spores while grazing; however, the most common source of infection in cats is from raw or poorly cooked contaminated meat or contact with the blood, tissues, or body fluids of infected animals that harbor spores.

Anthrax is not directly communicable by usual social contact from one infected animal to another animal, between animal and human, or between human and human, even in the case of anthrax pneumonia. For infection to occur, spores must gain access to the new victim by ingestion, inhalation, or through open wounds. When transmission occurs between individuals it is usually through exposure to infected tissue or body fluids. Human cases of anthrax may follow contact with contaminated animals or animal products. Therefore, humans should use strict precautions (wearing gloves, protective clothing, goggles, and masks) when handling potentially infected animals or their remains.
Under-diagnosis and unreliable reporting make it difficult to estimate the true frequency of anthrax worldwide; however, anthrax has been reported from nearly every continent. Under normal circumstances, anthrax outbreaks in the United States are extremely rare. Anthrax received much attention in 2001 in relation to the terrorist attacks on the United States because of its potential use as a biological weapon.

Dogs and cats may develop sudden, severe (acute) blood poisoning after ingesting *Bacillus anthracis* bacteria. This may lead to a rapid swelling of the throat and sudden death. More often, a mild, chronic form is seen, in which cats show generalized signs of illness and gradually recover with treatment. Intestinal involvement is seldom recognized because the signs (such as loss of appetite, vomiting, diarrhea or constipation) are so nonspecific.
A diagnosis based on signs is difficult because many infections and other conditions (such as poisoning) may have signs similar to anthrax. Diagnosis thus requires laboratory analysis of blood samples from the potentially infected cat to confirm the presence of the bacteria.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Borreliosis	**5.2.10**	Merck Veterinary Manual AUS 2012 Bjarnadottir 2007 Lind 2014	High	Moderate	Moderate	High	Moderate

Lind 2014	Zoonotisk risiko (Hund-> Homo)	Impact hos hund	Impact hos homo	Forekomst (endemicitet)
Borrelia burgdoferi	Moderat	Moderat	Alvorlig	Pan-europæisk (Ixodes)

<u>AUS 2012</u> Risk review

Regarding the importation of dogs and cats, the following key factors were considered relevant to the biosecurity risk presented by Lyme disease:

- Lyme disease is the most commonly reported tick-borne disease in humans in Asia, Europe and the United States.
- In endemic areas, dogs have an equal or greater likelihood of becoming infected as humans.
- The incubation period is 2–5 months, most infections are subclinical and diagnosis may not be straightforward.
- Exotic ticks infected with *B. burgdorferi* s.l may be present on imported dogs and cats.
- Local ticks feeding on infected animals may become infected and act as vectors.
- If potential tick vectors in Iceland became infected, they may spread infection to other animals, which may then act as reservoir hosts.
- Birds and small mammals may disseminate the organism across a wide area.

Conclusion

Based on the preceding factors, it was concluded that risk management for Lyme disease continues to be warranted for dogs and cats. In addition, it was concluded that risk management for Lyme disease is not warranted for dog or cat semen.

The following measures were proposed as an appropriate risk management option.

Pre-export measures (dogs and cats)

Vector management

- As per pre-export measures proposed for external parasite control

Post-arrival measures (dogs and cats)

Vector management

- As per post-arrival measures proposed for external parasite control

Lyme borreliosis is a bacterial, tick-transmitted disease of animals (dogs, horses, probably cats) and people. Areas of greatest incidence in the USA are regions in the northeast (particularly the New England states), the upper Midwest, and the Pacific coast. Lyme borreliosis also occurs in moderate climatic regions of Europe and Asia. It appears, however, not to be endemic in Iceland (Bjarnadottir, 2007).

Zoonotic Risk:

Lyme borreliosis is an important zoonotic disease. Animals and people are infected during the blood meal of hard-shelled ticks (*Ixodes* spp). Companion and farm animals are not the source of infection in people. Pets may bring unattached infected ticks into the household and subsequently the vectors may be passed on to other animals or people during close contact.

Etiology and Transmission:

Tick vectors of *B burgdorferi sensu lato* are hard-shelled *Ixodes* ticks. *I ricinus* and *I persulcatus* are the primary vectors in Europe and Asia. *B miyamotoi* is another member of the genus *Borrelia*. Although this spirochete is transmitted by ixodid ticks and may cause infections in mammals characterized by clinical signs such as fever, headache, fatigue, and muscle aches, the bacterium is a relapsing, fever-causing organism.

Ixodid ticks hatch from eggs as uninfected larvae. Both larvae and nymphs may acquire spirochetes from *Borrelia*-carrying hosts. Small mammals, especially rodents, play a major role as reservoir hosts. Birds and lizards may also harbor certain *Borrelia* species and serve as reservoir hosts. Infection rates of the vectors vary according to region and season and can be as high as 50% in adult ticks. After tick attachment, >24 hrs. elapse before the first *B burgdorferi sensu lato* organisms are transmitted into the host's skin. Stable infection of the host occurs at >53 hrs. into the blood meal. Therefore, early removal of attached ticks reduces the potential for spirochete transmission. *B burgdorferi sensu lato* organisms are not transmitted by insects, body fluids (urine, saliva, semen), or bite wounds. Experimental studies have shown that dams infected before gestation may transmit spirochetes to their pups in utero.

Clinical Findings:

Numerous clinical syndromes have been attributed to Lyme borreliosis in domestic animals, including limb and joint disease and renal, neurologic, and cardiac abnormalities. In dogs, intermittent, recurrent lameness; fever; anorexia; lethargy; and lymphadenopathy with or without swollen, painful joints are the most common clinical signs. The second most common syndrome associated with Lyme borreliosis is renal failure, which is generally fatal.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Boutonneuse fever	**5.2.11**	Merck Veterinary Manual Lind, 2014	High	Very low	Low	High	Very low

Lind 2014	Zoonotisk risiko (Hund-> Homo)	Impact hos hund	Impact hos homo	Forekomst (endemicitet)
Rickettsia conorii	Høj	Lav	Høj	Pan-europæisk (Rhipicephalus)

Boutonneuse fever (also called Mediterranean spotted fever, *fièvre boutonneuse*, Kenya tick typhus, Indian tick typhus, Marseilles fever, or African tick-bite fever, or Astrakhan Fever) is a fever as a result of a rickettsial infection caused by the bacterium *Rickettsia conorii* and transmitted by the dog tick *Rhipicephalus sanguineus*. Boutonneuse fever can be seen in many places around the world, although it is endemic in countries surrounding the Mediterranean Sea. This disease was first described in Tunisia in 1910 by Conor and Bruch and was named *boutonneuse* (French for "spotty") due to its papular skin rash characteristics in humans.
High seroprevalence is found among dogs in the Mediterranean countries (reservoir animals), but rarely with any clinical symptoms.

Nonspecific febrile illness; eschar (typically single) may or may not be present; rash, often maculopapular, in most; life-threatening disseminated disease or neurologic signs possible but uncommon; case fatality rate 1% –18% if untreated.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Campylobacter	**5.2.12**	Merck Veterinary Manual Lind, 2014 Rijks et al. 2016	Moderate	Moderate	Moderate	Moderate	Moderate

Lind, 2014	**Zoonotisk risiko (Hund-> Homo)**	**Impact hos hund**	**Impact hos homo**	**Forekomst (endemicitet)**
Campylobacter	Moderat	Moderat	Moderat	Pan-europæisk

Gastrointestinal campylobacteriosis is a bacterial disease. It is caused by 2 related bacteria of the *Campylobacter* genus. These organisms, along with a number of other species of *Campylobacter*, can be isolated from infected dogs and cats that do not show signs of infection (carriers), as well as from animals that show signs of the illness. This disease can be transmitted to humans. Animals, including dogs and cats (especially those recently adopted from shelters), and wild animals maintained in captivity can serve as sources of human infection.

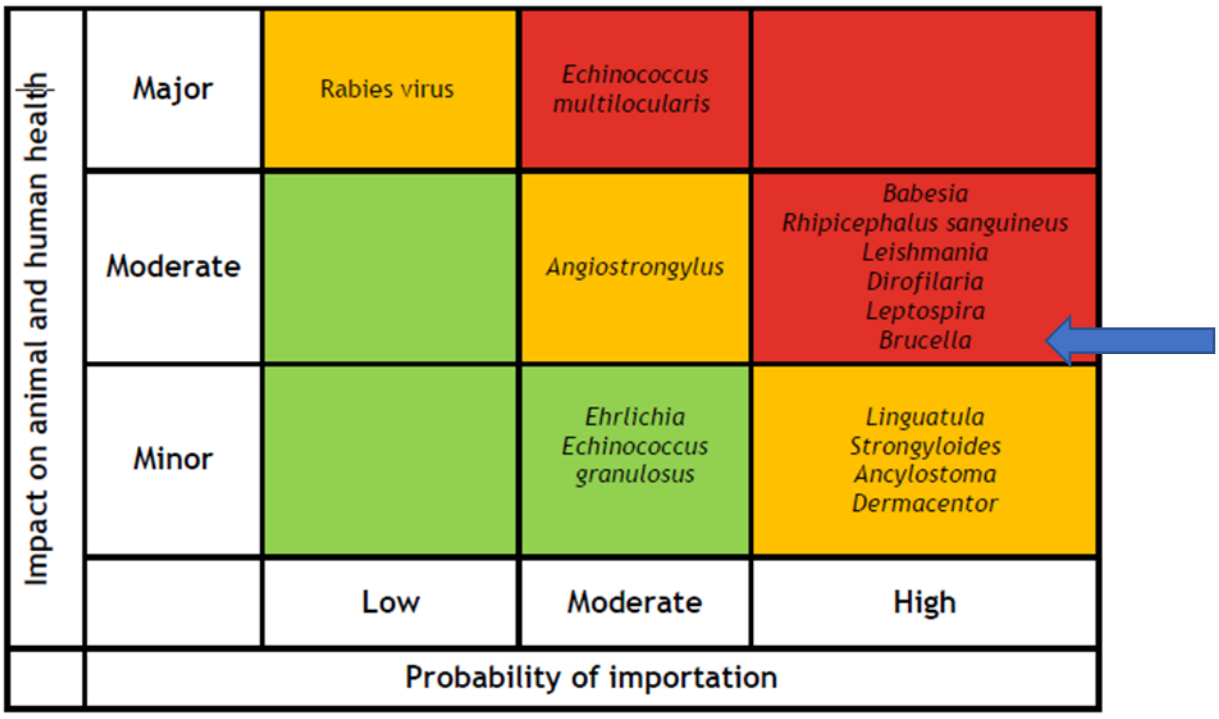
Exposure to feces of infected animals and food or waterborne transmission appear to be the most common routes of infection. One suspected source of infection for pets is eating undercooked poultry and other raw meat products. Wild birds also may be important sources of water contamination.

The diarrhea appears to be most severe in young animals. Typical signs include mucus-laden, watery, or bile-streaked diarrhea (with or without blood) that lasts 3 to 7 days; reduced appetite; and occasional vomiting. Fever may also be present. Intermittent diarrhea may persist for more than 2 weeks; in some, the intermittent diarrhea may continue for months. To diagnose campylobacteriosis, a veterinarian will test the animal's feces and blood for evidence of infection.

Antibiotic treatment for pets found to carry these bacteria is usually reserved for those that are young, severely affected, or a potential source of human infection. This is because other organisms are likely to be involved and antibiotic treatment is often not effective.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Canine brucellosis	**5.2.13**	Holst et al. 2012, AUS 2012, Høgåsen et al. 2012 Lind 2014	High	Moderate	Moderate/ High	Moderate/ High	Moderate

Lind 2014	Zoonotisk risiko (Hund-> Homo)	Impact hos hund	Impact hos homo	Forekomst (endemicitet)
Brucella canis	Moderat	Alvorlig	Alvorlig	Sporadisk



From Høgåsen et al. 2012

AUS 2012 Risk review

Regarding the importation of dogs and cats, the following key factors were considered relevant to the biosecurity risk presented by *B. canis*:

- Canine brucellosis is a nationally notifiable disease in Australia.
- Canine brucellosis is primarily a sexually transmissible disease of breeding dogs and is also transmissible by artificial insemination with infected semen.

• Although transmission of *B. canis* has been associated with vaginoscopy, blood transfusion and the use of contaminated syringes, these modes of transmission are of minor epidemiological importance.

• Chronic infections are common and treatment with antibiotics is often ineffective at eliminating infection because of the persistent intracellular location of the organism.

• Although desexed animals may occasionally harbour infection, shedding of the organism by desexed animals is not considered to be a significant risk factor for establishment or spread.

Conclusion

Based on the preceding factors, it was concluded that risk management for canine brucellosis continues to be warranted for dogs and their semen.

The following measures were proposed as an appropriate risk management option.

Pre-export measures (dogs)

For entire dogs:

• The dog must be tested for *Brucella canis* during the 21 days before export using a serum agglutination test (SAT)2 with a negative result. If test results by SAT are positive or inconclusive, the dog will be eligible for export if serum is tested within the 21 days immediately before export with a negative result by CPAg-AGID.

• If mated or artificially inseminated within the 30 days before export, serological testing must be conducted not less than 14 days after the date of last mating or insemination.3

• The laboratory report, or a copy endorsed by an official veterinarian, must be attached to the veterinary certificate.

NOTE: Any entire dog diagnosed with *B. canis* infection based on serological test results is not eligible for import, regardless of treatment.

OR

For dogs that are desexed:

• The desexing certificate, or a copy endorsed by an official veterinarian, must be attached to the veterinary certificate.

OR

• Serological testing must be conducted as for entire dogs, with a negative result within the 21 days immediately before export.

Recommendations to avoid introduction of B. canis into non-endemic areas (Holst et al. 2012):

- To avoid introducing brucellosis into a kennel, the introduction of untested dogs from endemic areas should be avoided.
- Dogs from kennels in which B. canis has been diagnosed should not be used for breeding.
- Dogs from endemic areas should be kept isolated until tested free of B. canis to avoid further spread of the disease. This is recommended for natural mating, artificial insemination with fresh, chilled or frozen semen, and when introducing new dogs into the kennels.
- For kennels in endemic areas it is often recommended that breeding animals are tested annually, and that all new dogs are tested before being introduced into the kennel.

- Serologic tests can be negative up to 4 weeks after infection, and at least 12 weeks must pass to be sure of detecting antibodies in an infected animal. Therefore, two negative tests 4-6 weeks apart are needed in case the dog is incubating the disease. At least one of the sampling occasions should be no earlier than 12 weeks after suspected contact with an infected animal.
- Bitches that might be chronically infected should be tested during oestrus, pregnancy or at abortion, as they may have low antibody titres at other times.
- Chronically infected male dogs can also be difficult to detect, as they too can be serologically negative. In addition to serologic methods, bacterial culture or PCR analysis can be performed to detect the bacterium.
- Only dogs tested free from B. canis should be used for breeding, and recommended tests depend on the level of risk.

If B. canis is detected in a group of dogs, regular testing and removal of infected dogs and strict hygiene is necessary to eradicate the infection. Common disinfectants are effective against B. canis.
B canis is a cause of abortion at 45–55 days of gestation in kenneled dogs. Dogs are the only definitive host of this organism. Infection has caused a reduction of 75% in the number of pups weaned in some breeding kennels. The disease disseminates rapidly among closely confined dogs, especially at time of breeding or when abortions occur. Transmission occurs via ingestion of contaminated materials or venereal routes. Urine transmission has been reported but seems to be unusual. Both sexes appear to be equally susceptible.

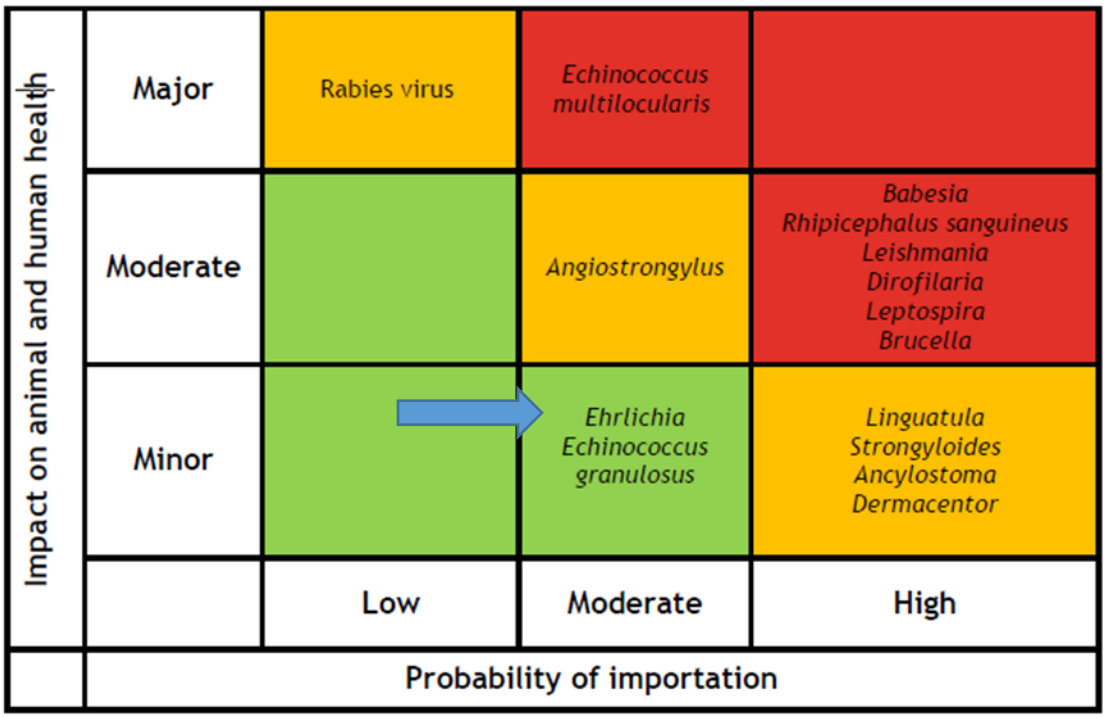
Infection with *Brucella canis* in dogs leads to abortion, infection of the sexual organs in males, and infertility. The disease occurs throughout the world and primarily affects dogs. Both sexes appear to be equally susceptible. The primary sign of the infection in females is abortion during the last trimester of pregnancy without previous signs of abnormality. Abortion may occur during subsequent pregnancies. Prolonged vaginal discharge usually follows abortion. Abortions may occur during subsequent pregnancies. Infected dogs may develop generalized lymphadenitis and frequently epididymitis, peri-orchitis, and prostatitis. Spondylitis and uveitis are occasional complications. Bacteremia is frequent and persists for ~18 mo. after exposure. Fever is not characteristic.

In males, the primary signs of infection are inflammation of the epididymites or testicles and reluctance to mate because of this inflammation. Transmission of brucellosis from dogs to humans occurs, but is quite rare. In humans, the disease can be very serious.

Brucellosis caused by *Brucella abortus, B. suis*, or *B. melitensis* is relatively rare in dogs. In cases that do occur, the dogs are usually around livestock, as they are the primary source of those strains of the bacteria. The disease is diagnosed through laboratory tests. Spread of infection is controlled through isolation of infected dogs. Brucellosis is very difficult to treat successfully, and euthanasia of infected dogs is often recommended.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Canine ehrlichiosis	**5.2.14**	AUS 2012, Hamel et al. 2013, Lind 2014, Høgåsen et al. 2012	Moderate	Low	Moderate	Low/ Moderate	Low

Lind 2014	Zoonotisk risiko (Hund-> Homo)	Impact hos hund	Impact hos homo	Forekomst (endemicitet)
Ehrlichia canis	Moderat	Moderat	Moderat	Syd-Europa (Rhipicephalus)



From Høgåsen et al. 2012

AUS 2012 Risk review

Regarding the importation of dogs and cats, the following key factors were considered relevant to the biosecurity risk presented by Canine monocytic ehrlichiosis (CME):

- *E. canis* and *E. chaffeensis* are exotic to Australia.
- CME caused by *E. canis* may be associated with high morbidity and mortality rates in dogs.
- Dogs are considered the primary reservoir host for *E. canis*, and white-tailed deer are considered the primary reservoir host for *E. chaffeensis*.
- *Rhipicephalus sanguineus*, the primary tick vector for *E. canis*, is present in Australia.
- Species of *Ehrlichia* that naturally infect cats (and the significance of such infection) have not been determined.
- Persistent subclinical infection is a feature of *E. canis* infection in dogs and may last for years. Ticks are able to acquire *E. canis* infection from subclinically infected dogs post-treatment.
- Treatment of subclinically infected dogs with antibiotics (e.g. doxycycline) has not been shown to be reliable in eliminating *E. canis* infection. Antibody (IgG) titres following exposure to *E. canis* may persist for months to years. Persistently high IgG titres have been detected in subclinically infected dogs.
- PCR is a sensitive technique that is useful in detecting active infection. However, the short and variable time that *E. canis* is present in the plasma of an infected dog limits its value in detecting subclinical infection.
- PCR is most sensitive when performed on spleen samples. Collection of spleen samples is invasive and not practical for routine pre-export testing.
- Diagnostic serology using an IFAT is reliable for detecting the presence of IgG antibodies to *E. canis*.
- False positive serology (IFAT) results may occur due to cross-reactivity with other ehrlichial species (e.g. *E. chaffeensis* and *E. ewingii*).
- False negative serology results may occur in the acute phase if blood is collected before seroconversion has occurred.
- The incubation period for CME is 8–20 days with seroconversion reported to occur 3–28 days after experimental exposure to *E. canis*; seroconversion may be influenced by the dose of infectious organisms.
- For disease screening, prophylactic treatment against tick vectors before serological testing may reduce the occurrence of false negative serological results as well as manage the risk that animals may be in the incubation phase of infection at the time of physical examination.
- Vector management is important to prevent the entry of CME.

Conclusion

Based on the preceding factors, it was concluded that risk management for CME continues to be warranted for dogs. In addition, it was concluded that risk management for CME is not warranted for dog semen.

The following measures were proposed as an appropriate risk management option.

Pre-export measures (dogs)

Serology

- Before export, a blood sample must be obtained from the dog by a government-approved veterinarian and be submitted for serology testing for *E. canis* by IFAT. The test must produce a negative result at a dilution of 1:40 (or as otherwise agreed by DAFF and the testing laboratory).

AND

Vector management

- At least 21 days before blood sample collection, the dog must be treated by a government-approved veterinarian with a parasiticide effective against ticks on contact, in accordance with the manufacurer's recommendations. At the time of treatment, the dog must be thoroughly examined for ticks and any ticks detected must be removed.
- The dog must be re-treated with the parasiticide by a government-approved veterinarian, in accordance with the manufacturer's recommendations, such that the animal is continuously protected against ticks for the period extending from 21 days before blood sampling to export. A final treatment must be administered within the four days immediately before export.
- At each parasiticide re-treatment and at the time of blood sampling, the dog must be subjected to a thorough physical examination by a government-approved veterinarian and found to be free of ticks.
- The dog must be subjected to a thorough physical examination by a government-approved veterinarian within the four days immediately before export and found to be free of ticks.
- If a viable tick is detected at any examination, it must be removed and the dog must be re-treated with a parasiticide effective against ticks on contact. A blood sample for *E. canis* serology must be collected at least 21 days after re-treatment.

Post-arrival measures (dogs)

Vector management

- As per post-arrival measures proposed for external parasite control.

The *Ehrlichia* and *Anaplasma* rickettsiae are present in many parts of the world, including the United States. They are transmitted by ticks (including the brown dog tick, lone star tick, and black-legged tick) that become infected after feeding on infected animals. People, dogs, cats, and other domestic animals are accidental hosts of these disease-causing organisms.

Canine monocytic ehrlichiosis (CME) is usually caused by the rickettsia *Ehrlichia canis*, although other types of *Ehrlichia* are sometimes involved. (Rickettsiae are a specialized type of bacteria that live only inside other cells.) Carried by ticks, the organism infects a certain type of white blood cell and causes fever

and other signs. In infections caused by *Ehrlichia canis*, signs commonly progress from short to long-term, depending on the strain of the organism and the immune status of the host. In short-term cases, there is fever, widespread inflammation of the lymph nodes, enlargement of the spleen, and a decrease in the number of platelets in the bloodstream. In addition, there may be loss of appetite, depression, loss of stamina, stiffness and reluctance to walk, swelling of the limbs or scrotum, and coughing or difficulty in breathing. Most short-term cases are seen in the warmer months, when ticks are active. During this phase of infection, death is rare and the infected animal may recover spontaneously. The recovered dog may remain free of signs thereafter, or long-term disease may develop.

Long-term ehrlichiosis caused by *Ehrlichia canis* may develop in any breed of dog, but certain breeds (such as German Shepherds) may be predisposed. Long-term infection does not vary with the seasons. Signs depend on which organs are affected and may include enlargement of the spleen, kidney failure, and inflammation of the lungs, eye, brain and spinal cord. If the brain and spinal cord are involved, there may be problems with the nervous system, such as lack of coordination, depression, partial paralysis, and increased sensitivity to a normally painless touch. Severe weight loss is common.

The most important preventive steps are those that control ticks, the most common source of the disease. Keeping your dog away from areas known to harbor ticks is a step you can take. Preventive medications that will keep your dog from being infested with ticks are also available from your veterinarian. Any ticks found on your dog should be promptly and properly removed to prevent the spread of disease.

Some types of *Ehrlichia* bacteria can be transmitted to people. Despite the occurrence of disease in both animals and people, a tick is required for transmission, so dogs and other infected animals do not pose a direct transmission risk in normal circumstances. Infection in dogs may indicate a heightened risk of human infections related to tick exposure in a given area.

Epidemiology:

E canis is transmitted by the brown dog tick, *Rhipicephalus sanguineus*, which is found worldwide; accordingly, canine monocytic ehrlichiosis also has a worldwide enzootic distribution. Acute *E canis* cases may resemble infection with *Rickettsia rickettsii* (the agent of Rocky Mountain spotted fever, which can also be transmitted by the brown dog tick). *Rhipicephalus* ticks become infected with *E canis* after feeding on infected dogs, and ticks transmit infection to other dogs during blood meals taken in successive life stages.

Prevention:

Prevention is enhanced by controlling ticks on dogs, through use of reliable methods. In particular, medications and products with proven efficacy against *R sanguineus* are important to use. Because *R sanguineus* infestations can be problematic in kennels and around homes, and long-term tick control is needed for management, use of effective long-acting collars on all susceptible dogs might be considered; collars containing propoxur, amitraz, or flumethrin have proven activity against *R sanguineus.*

Zoonotic Risk:

E chaffeensis, *E ewingii*, and *A phagocytophilum* are considered zoonoses. Despite the occurrence of disease in both animals and people, the involvement of a required intermediate tick vector for transmission means dogs and other infected animals do not pose a direct transmission risk in normal circumstances. Infection in dogs may indicate a heightened risk of human infections related to tick exposure in a given area.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Glanders	**5.2.15**	Merck Veterinary Manual	Very low	Low	High	High	Very low

Glanders is a contagious, acute or chronic, usually fatal disease of Equidae caused by *Burkholderia mallei* and characterized by serial development of ulcerating nodules that are most commonly found in the upper respiratory tract, lungs, and skin. Felidae and other species are susceptible, and infections are usually fatal. The organism is infectious for people, with a 95% fatality rate in untreated septicemia cases, and is considered a potential bioterrorism agent. Glanders is one of the oldest diseases known and once was prevalent worldwide. It has now been eradicated or effectively controlled in many countries, including the USA. In recent years, the disease has been reported in the Middle East, Pakistan, India, Mongolia, China, Brazil, and Africa.

Etiology:

Burkholderia mallei, a clonal gram-negative facultative intracellular obligate pathogen, is present in nasal exudates and discharges from ulcerated skin of infected animals.

Dogs and Cats:

The disease is characterized by the development of a series of ulcerating nodules. The nodules are most commonly found in the upper respiratory tract, lungs, and skin. Humans, cats, dogs, and other species are susceptible, but infections in dogs and cats are uncommon. Infections in humans are often fatal.

There is no vaccine for glanders. Prevention and control depend on early detection and elimination of affected animals. Complete quarantine and rigorous disinfection are required for all housing and objects that have been in contact with the infected animal. Euthanasia is usually recommended for infected animals.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Ringworm	**5.3.1**	Merck Veterinary Manual	Moderate	Moderate	High	Moderate	Moderate

Ringworm is an infection of skin, hair, or claws caused by a type of fungus. In dogs, about 70% of ringworm cases are caused by the fungus *Microsporum canis*, 20% by *Microsporum gypseum*, and 10% by *Trichophyton mentagrophytes*. In cats, 98% are caused by *M canis*. The infecting fungus is spread easily in the environment. People can easily be infected with these fungi.

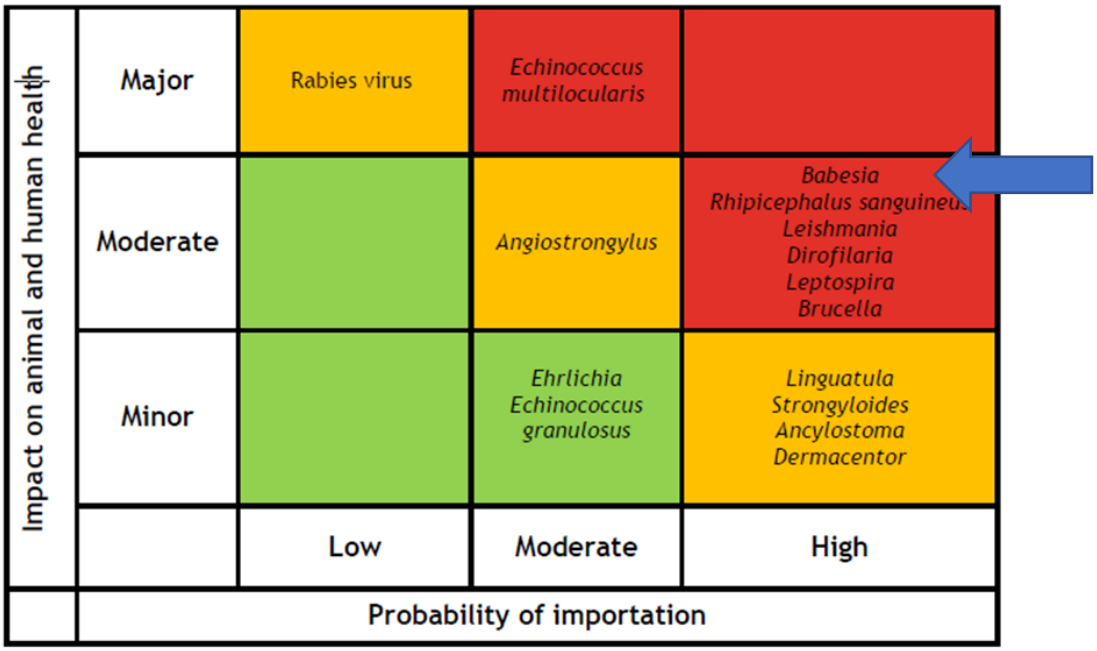
The clinical appearance of ringworm in cats is quite variable. Kittens are affected most commonly. Typical lesions consist of focal alopecia, scaling, and crusting; most are located around the ears and face or on the extremities. Cats with clinically inapparent infections can serve as a source of infection to other cats or people. Occasionally, dermatophytosis in cats causes feline miliary dermatitis and is pruritic. Cats with generalized dermatophytosis occasionally develop cutaneous ulcerated nodules, known as dermatophyte granulomas or pseudomycetomas. Devon Rex cats can have a maculopapular hyperpigmented, crusted disease that histopathologically is an eosinophilic/mastocytic dermatitis.

Lesions in dogs are classically alopecic, scaly patches with broken hairs. Dogs may also develop regional or generalized folliculitis and furunculosis with papules and pustules. A focal nodular form of dermatophytosis in dogs is the kerion reaction. Generalized ringworm in adult dogs is uncommon and is usually accompanied by immunodeficiency, especially endogenous or iatrogenic hyperadrenocorticism. Differential diagnoses for classic ringworm lesions in dogs include demodicosis, bacterial folliculitis, and seborrheic dermatitis.

Dermatophytosis in dogs and shorthaired cats may be self-limiting, but resolution can be hastened by treatment. Another primary objective of therapy is to decrease environmental contamination and prevent spread of infection to other animals and people.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Babesiosis	**5.4.1**	Merck Veterinary Manual Hamel et al. 2012 Høgåsen et al. 2012 Lind 2014	High	Very low	Moderate	Low/ Moderate	Very low

Lind 2014	**Zoonotisk risiko (Hund-> Homo)**	**Impact hos hund**	**Impact hos homo**	**Forekomst (endemicitet)**
Babesia canis	Lav	Moderat-alvorlig	Lav	Syd- og centralEuropa (*Dermacentor*)
Babesia gibsoni	Lav	Moderat-alvorlig	Lav	Syd- og centralEuropa (*Dermacentor*)
Babesia vogeli	Lav	Lav-moderat	Lav	Syd-Europa (*Rhipicephalus*)



From Høgåsen et al. 2012

Babesiosis is the most important and widely distributed tick-borne disease of dogs and wild canines (wolves, foxes, jackals). Babesiosis is transmitted by D*ermacentor spp*. and *R. sanguineus*. It is caused by protozoan parasites of the genus *Babesia,* which infect the red blood cells. Babesiosis affects a wide range of domestic and wild animals and, occasionally, humans. While the major economic impact of babesiosis is on the cattle industry, infections in dogs and cats occur at various rates throughout the world.
Signs of infection vary from a mild illness that passes quickly to a severe disease that rapidly results in death. In some cases, the parasite causes a long-term disease with severe and progressive anemia as the main symptom. Babesiosis can be confused with other conditions that cause fever, anemia, destruction of red blood cells, jaundice, or red urine. Therefore, laboratory tests should be performed to confirm the diagnosis.

A vaccine based on some types of *Babesia* is available, but it does not protect against all types. Preventing exposure to ticks by using appropriate tick control products and removing any ticks promptly will help keep your dog from being exposed to this parasite.

A small number of cases of human babesiosis have been reported, but it is unclear whether the species of *Babesia* that infect dogs (*Babesia canis ssp.*) are the same as those that cause infection in people. Fatal cases have been reported in people whose spleen had been removed or who had a weakened immune system. Human *Babesia* infections are acquired by way of bites from infected ticks or through contaminated blood transfusions.

Illness of varying severity due to *Babesia felis* has been reported in domestic cats in Africa and India. An unusual feature is its lack of response to the normal medicines used to destroy *Babesia* parasites.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Hepatozoonosis	**5.4.2**	EFSA VBD 2017, Menn et al. 2010, AUS 2012, Lind 2014	Low/ Moderate	Very low/Low	Low/ Moderate	Very low	Very low/ Low

Lind 2014	Zoonotisk risiko (Hund-> Homo)	Impact hos hund	Impact hos homo	Forekomst (endemicitet)
Hepatozoon canis	Ingen-lav	Lav-moderat	-	Syd-Europa (*Rhipicephalus*)

AUS 2012: Risk review

Regarding the importation of dogs and cats, the following key factors were considered relevant to the biosecurity risk presented by hepatozoonosis:

- Hepatozoonosis is not an OIE-listed disease and is not a nationally notifiable disease in Australia.
- Canine hepatozoonosis is exotic to Australia.
- The dog is a vertebrate host for *H. canis* and *H. americanum*.
- *R. sanguineus*, the tick vector for *H. canis*, is present in Australia.
- Prevention of infection of dogs is by treatment with acaricides to prevent infestation by ticks.
- Treatment does not eliminate either *H. canis* or *H. americanum* parasites.
- Available diagnostic tests are not reliable as screening tests for hepatozoonosis.

Conclusion Based on the preceding factors, it was concluded that risk management for hepatozoonosis is warranted for dogs only. It was also concluded that risk management for hepatozoonosis is not warranted for dog semen.

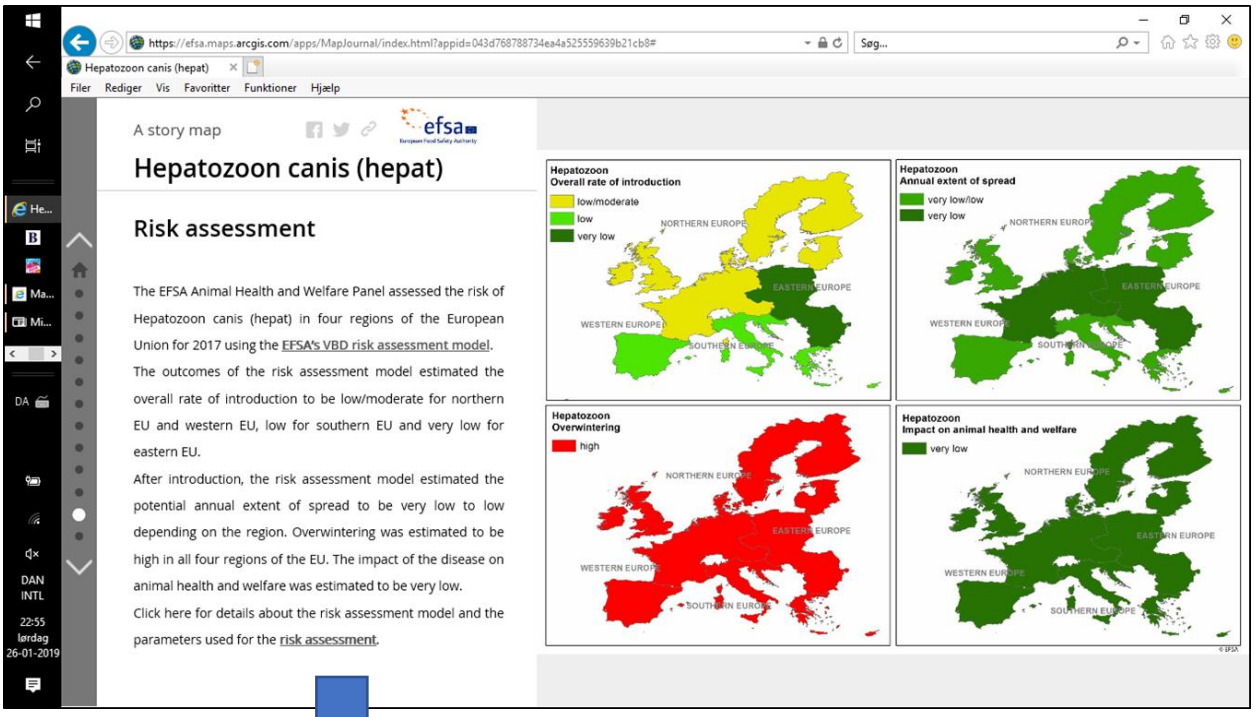
The following measures were proposed as an appropriate risk management option.

Pre-export measures (dogs)

- As per pre-export measures proposed for external parasite control.

Post-arrival measures (dogs)

- As per post-arrival measures proposed for external parasite control.



A story map
efsa
Hepatozoon canis (hepat)
Risk assessment
The EFSA Animal Health and Welfare Panel assessed the risk of Hepatozoon canis (hepat) in four regions of the European Union for 2017 using the EFSA's VBD risk assessment model.
The outcomes of the risk assessment model estimated the overall rate of introduction to be low/moderate for northern EU and western EU, low for southern EU and very low for eastern EU.
After introduction, the risk assessment model estimated the potential annual extent of spread to be very low to low depending on the region. Overwintering was estimated to be high in all four regions of the EU. The impact of the disease on animal health and welfare was estimated to be very low.
Click here for details about the risk assessment model and the parameters used for the risk assessment.

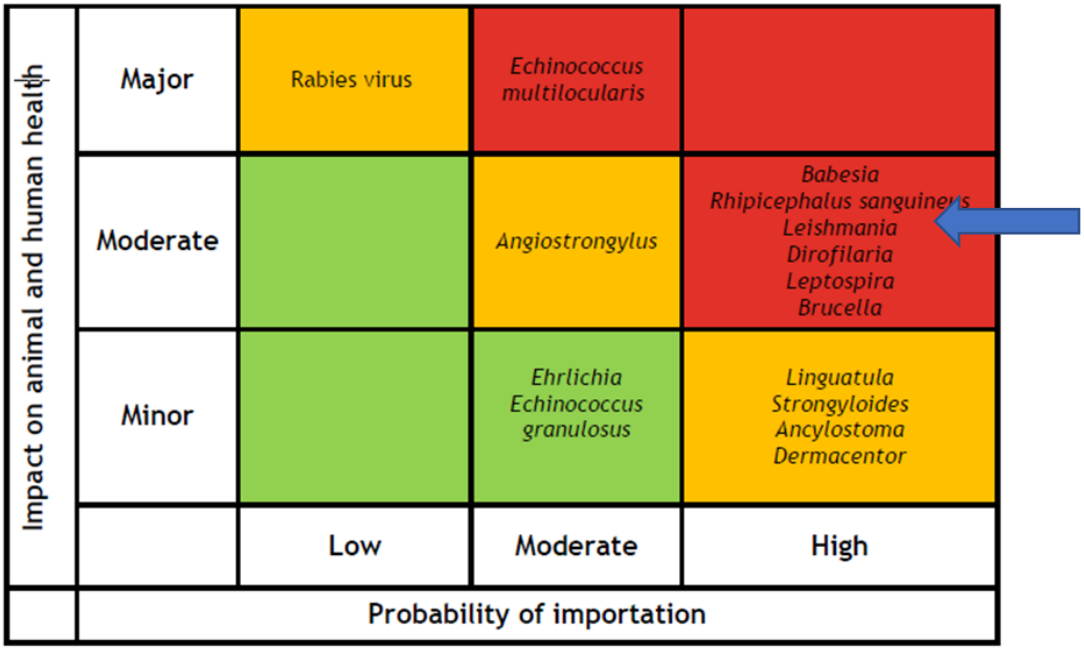
Hepatozoonosis is a disease of wild and domestic carnivores (meat-eating animals) caused by a *Hepatozoon* protozoan. This organism is transmitted by ticks, but its mode of transmission is unusual. The tick picks up the organism from an infected host while biting the animal. An uninfected animal gets the disease by eating the tick (or animals that ate a tick), not from being bitten by the tick. In most of the world, the protozoan *Hepatozoon canis* is transmitted by the brown dog tick (*R. sanguineus*), causing Old World hepatozoonosis. There is no known risk of transferring this disease to humans.

In much of the world (India, Africa, southeast Asia, the Middle East, southern Europe, and islands in the Pacific and Indian Oceans), infected dogs have no signs of infection or only mild signs. Having a suppressed immune system due to another disease appears to play an important role in the development of significant signs. In the United States, more severe signs may occur, even in dogs that do not have a suppressed immune system.

Dogs older than 4 to 6 months old are usually resistant to infection with *H. canis*. Hepatozoonosis is a life-long infection in dogs. No known treatment completely clears the body of the organism. In the past, most dogs showed only temporary improvement, with frequent relapses within 3 to 6 months and death within 2 years of diagnosis. However, remission can now usually be achieved by using new drug combinations. These new therapies have resulted in a marked improvement in the outlook for dogs with hepatozoonosis. Prevention of access to ticks and preventing dogs from catching and eating prey are the most effective ways to control this disease. Affected dogs should not be bred.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Leishmaniosis	**5.4.3**	Høgåsen et al. 2012 AUS 2012 EFSA VBD 2017, Menn et al. 2010, Hamel et al. 2013, Lind 2014 Ready 2010	Variable	Very low	High	High	Very low

Lind 2014	**Zoonotisk risiko (Hund-> Homo)**	**Impact hos hund**	**Impact hos homo**	**Forekomst (endemicitet)**
Leishmania infantum	Høj	Alvorlig	Alvorlig	Syd-Europa (Rhinland)



From Høgåsen et al. 2012

EFSA VBD Risk assessment

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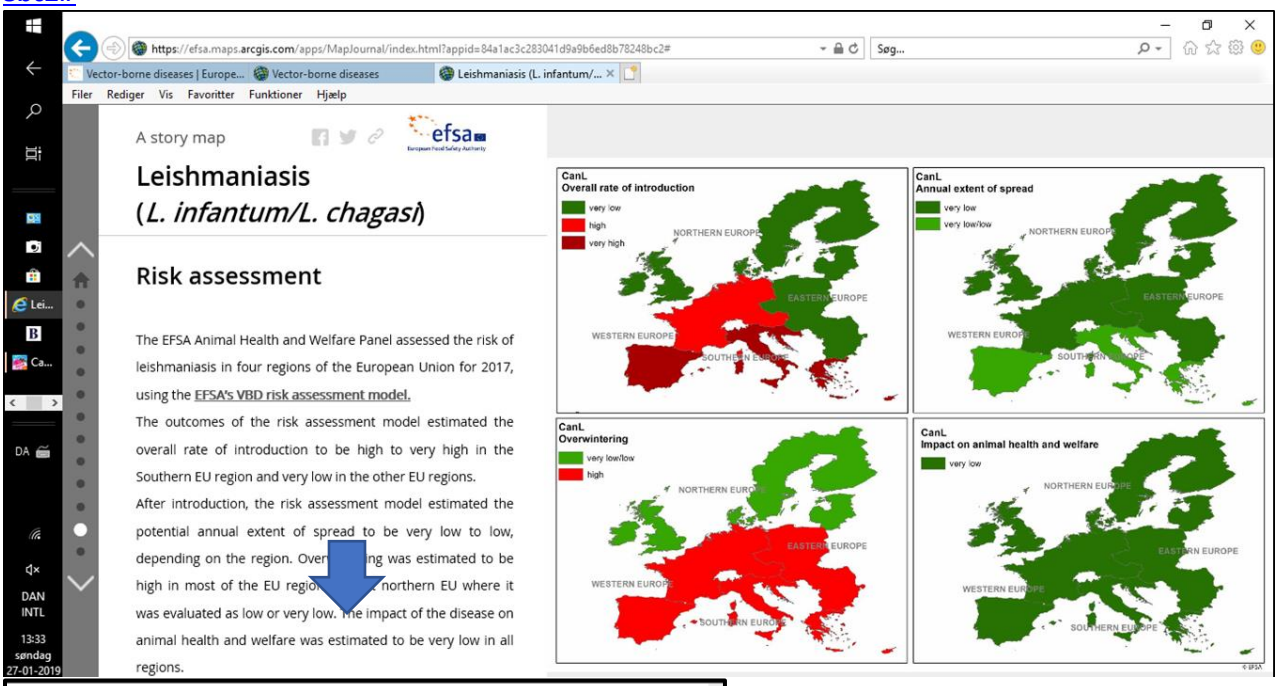
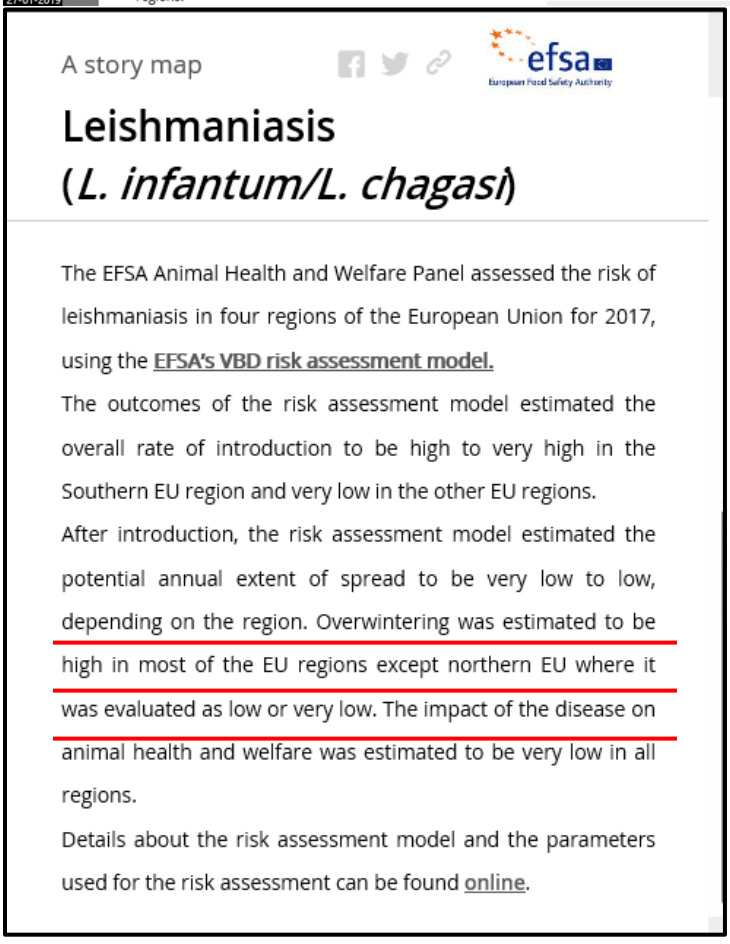




A story map

Leishmaniasis
(*L. infantum/L. chagasi*)

The EFSA Animal Health and Welfare Panel assessed the risk of leishmaniasis in four regions of the European Union for 2017, using the **EFSA's VBD risk assessment model.**

The outcomes of the risk assessment model estimated the overall rate of introduction to be high to very high in the Southern EU region and very low in the other EU regions.

After introduction, the risk assessment model estimated the potential annual extent of spread to be very low to low, depending on the region. Overwintering was estimated to be high in most of the EU regions except northern EU where it was evaluated as low or very low. The impact of the disease on animal health and welfare was estimated to be very low in all regions.

Details about the risk assessment model and the parameters used for the risk assessment can be found online.

AUS 2012 Risk review

Regarding the importation of dogs and cats, the following key factors were considered relevant to the biosecurity risk presented by *L. infantum*:

- Leishmaniasis is an OIE-listed disease and is a nationally notifiable disease in Australia.
- *L. infantum* is endemic in many countries and causes severe viscerocutaneous disease in dogs and humans.
- Dogs and humans are a recognised reservoir host of *L. infantum*.
- *Leishmania* spp. have been isolated from the skin lesions of a group of captive red kangaroos (*Macropus rufus*) in Australia.
- There is evidence that biting midges may be transmitting Australian *Leishmania* spp. in the Northern Territory.
- It remains uncertain whether a competent vector capable of transmitting *L. infantum* is present in Australia.
- Treatment does not eliminate infection.

Conclusion

Based on the preceding factors, it was concluded that risk management for *L. infantum* continues to be warranted for dogs. It was also concluded that risk management for *L. infantum* is warranted for dog semen.

The current biosecurity measure (pre-export serology with a negative result) was considered an appropriate risk management option for dogs.

Leishmaniosis is a disease caused by protozoan parasites of the genus *Leishmania* and transmitted through the bites of female phlebotomine sand flies. More than 23 species of *Leishmania* have been described, most of which are zoonotic. The most important *Leishmania* parasite to affect domestic animals is *L infantum*, also known as *L chagasi* in Latin America. Dogs are the main reservoir host for human visceral leishmaniosis caused by *L infantum*, and the disease is potentially fatal in dogs and people. Because the internal organs and skin of the dog are affected, the canine disease is termed viscero-cutaneous or canine leishmaniosis. Cats, horses, and other mammals can be infected by *L infantum* or other *Leishmania* species. The disease in cats is rarer than in dogs and may manifest in cutaneous or visceral organs. *L braziliensis*, the cause of tegumentary canine leishmaniosis, is widespread in regions of South America and may geographically overlap with *L chagasi*.

Canine leishmaniosis is a major zoonosis endemic in >70 countries. It is prevalent in southern Europe, Africa, Asia, South and Central America, and sporadically in the USA.

Transmission:

Leishmania is a diphasic parasite that completes its life cycle in two hosts: a sand fly that harbors the flagellated extracellular promastigote form and a mammal in which the intracellular amastigote parasite form develops.

Transmission is a complex process that requires special adaptation between the sand fly host and the particular *Leishmania* species transmitted. There are numerous species of sand flies, only a minority of which are competent vectors of *Leishmania*. Dogs with or without clinical signs are infectious to sand flies

and may transmit leishmaniosis. Congenital vertical transmission of canine leishmaniosis from an infected dam to its offspring has been reported but appears to be uncommon. Transmission by transfusion of blood products from infected dogs has been shown to cause infection in recipients. Direct dog-to-dog transmission by contact has been suggested as a mode of disease transmission in an effort to explain the spread of infection among kenneled Foxhounds in the USA in the absence of proven sand fly vectors. At present, the validity of direct transmission is unknown.

Cats and other domestic animals are rarely infected and usually only develop skin ulcers, without showing other signs of disease.

Zoonotic Risk:

Human visceral leishmaniosis caused by *L infantum* is a serious public health problem in areas where canine leishmaniosis is endemic and dogs are the main reservoir of infection. It is mostly a disease of young children. Malnutrition has been recognized as a risk factor and may explain why this disease is more prevalent among children in poor countries than among those in affluent ones, despite high prevalence rates in the canine populations. Human disease is also prevalent in immunosuppressed individuals; HIV patients are the predominant risk group for human leishmaniosis in southern Europe. Efforts to control canine leishmaniosis and the human disease in endemic areas focus on disrupting the transmission of infection and preventing canine infection at the population level.

Leishmaniosis is a chronic, severe disease of humans, dogs, and certain rodents caused by single-celled protozoa of the genus *Leishmania*. Visceral leishmaniosis is characterized by skin lesions, disease of the lymph nodes, weight loss, anemia, lameness, and kidney failure. Occasionally, there may be bleeding from the nose or eye lesions.

Leishmaniosis can be transmitted from dogs to people. Humans most frequently catch this disease when they are bitten by a sand fly or other insect that has previously bitten an infected animal or human. While there are only a very few human or animal cases in the US each year, worldwide there are about 1.5 million cases of cutaneous leishmaniosis and 500,000 cases of visceral leishmaniosis a year. Most human cases of visceral leishmaniosis are reported in India, Bangladesh, Nepal, Sudan, and Brazil.

The most reliable diagnostic test for canine leishmaniasis is direct observation of the parasite in bone marrow or lymph node smears. If your veterinarian suspects leishmaniosis, samples of bone marrow or fluid from the lymph nodes will be taken to confirm the diagnosis.

Drug treatment is available for dogs with visceral leishmaniosis and may last up to 6 months. Relapses after treatment are common. In areas where the disease is common, rapid treatment of infected dogs, control of stray and homeless dogs, and control of sand flies are recommended. At present, there is no effective vaccine.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Chaga's disease	**5.4.4**	Merck Veterinary Manual AUS 2012	Low	Negligible	Moderate	Moderate	Negligible

Chagas' Disease (*Trypanosoma cruzi* infection)

AUS 2012 Risk review

Regarding the importation of dogs and cats, the following key factors were considered relevant to the biosecurity risk presented by Chagas' disease:

- Chagas' disease is not OIE-listed, but is a nationally notifiable disease in Australia.
- Chagas' disease is endemic in Central and South America where it is a significant human health problem; it also occurs in the southern United States.
- Dogs and cats are recognized reservoir hosts of *T. cruzi*, as are humans.
- Treatment does not reliably eliminate infection.
- At least one triatomine (*Tr. leopoldi*) has been identified in Australia; however, its remote location and habitat preference make it an unlikely vector

Conclusion Based on the preceding factors, it was concluded that risk management for Chagas' disease is not warranted for dogs or cats or their semen.

Chagas' disease, or American trypanosomiasis, is a zoonotic, vector-borne disease transmitted by triatomine bugs and caused by *T cruzi*. All mammals are considered susceptible to infection, with infection demonstrated in >100 mammalian species. Avian species are not susceptible. The disease is best recognized in dogs and people, with dogs serving as a major domestic reservoir. Domestic pigs and cats can also be infected, but their role as reservoirs for human infection is limited. Wildlife reservoirs include opossums, armadillos, raccoons, woodrats, and nonhuman primates.

Transmission and Epidemiology:

Chagas' disease is endemic in 21 countries of South America, Central America, and Mexico, and is increasingly reported in the southern USA. Chagas' was once confined to the Americas, but human and animal migration has resulted in distribution to Europe, where it is an emerging disease of Spain, Switzerland, France, Italy, Germany, and England. Seropositivity in dogs in endemic regions can vary from 5%–92%. In some areas such as Venezuela, the seropositivity of dogs is similar to that of people, whereas in other areas, such as Campeche, Mexico, seropositivity can be higher in dogs than in people.

The nocturnal and hematophagous triatomine insects of the *Triatoma*, *Rhodnius*, and *Panstrongylus* genera serve as vectors for *T cruzi*. Common names include the "kissing bug.

In insectivorous animals, including dogs, consumption of infected bugs or materials contaminated with infected triatomine feces is a major mode of transmission. The opossum (*Didelphis* species) is a unique

host for *T cruzi*, because the parasite can complete its entire life cycle without the need for a vector. *T cruzi* maturation occurs in the anal odoriferous glands, and infective trypomastigotes can be shed in feces or urine and ingested. Additional methods of transmission include transplacentally or via blood transfusions and organ transplant.

Clinical Findings and Lesions:

Chagas' disease is divided into acute and chronic phases, with the chronic phase further subdivided into latent and symptomatic chronic disease. Incubation ranges from 5–42 days before acute disease. Acute infections may be asymptomatic or consist of nonspecific febrile illness with a chancre at the site of parasitic entry. Dogs may also present with regional or generalized lymphadenopathy, anorexia, lethargy, vomiting, diarrhea, and hepatomegaly or splenomegaly. Rarely, acute clinical myocarditis is seen. Parasitemia peaks 2–3 wks. after infection and dissipates after the first month as the organism moves to predominantly tissue-borne disease.

The latent phase can last for months to years. Chronic disease symptoms can include generalized weakness or sudden death. Symptomatic dogs commonly present with right-side congestive heart failure. This can progress to myocarditis, with arrhythmias and bilateral cardiac dilation. Histologic examination of cardiac muscle may contain unruptured pseudocysts without inflammation or contain ruptured pseudocysts with lymphocytic, monocytic, and/or neutrophilic inflammation. Death secondary to heart failure is common.

Treatment and Control:

Benznidazole is the drug of choice for treatment, but nifurtimox can also be used. In dogs, benznidazole is administered at 5–10 mg/kg/day, PO, for 2 mo. In the USA, both of these drugs lack FDA approval, and their use requires permission from the CDC as investigational protocols. Symptomatic treatment for heart failure and arrhythmias is also recommended.

No vaccine is available; thus, control focuses on preventing disease transmission. Vector control methods include pesticide application to eliminate triatomine vectors and decreasing vector attraction to dwellings at night by turning off outdoor lighting. Breeding of positive bitches is discouraged.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Giardia	**5.4.5**	Eydal et al. 2001, Lind 2012, Rijks et al. 2016 Fjøludottir 2013 Skirnisson et al. 2017	Indigenous*	Present*	Low/ moderate	Low/ moderate	n.a.*

Lind 2014	Zoonotisk risiko (Hund-> Homo)	Impact hos hund	Impact hos homo	Forekomst (endemicitet)
Giardia lamblia	Lav	Lav	Lav	Pan-europæisk

*Giardia has been identified in Iceland in native cats (Eydal et al. 2001) and dogs (Fjøludottir 2013), and has been declared indigenous in both dogs and cats (Skirnisson et al. 2017).

Giardiasis is a chronic, intestinal protozoal infection seen worldwide in most domestic and wild mammals, many birds, and people. Infection is common in dogs, cats, ruminants, and pigs. *Giardia* spp have been reported in 0.44%–39% of fecal samples from pet and shelter dogs and cats, 1%–53% in small ruminants, 9%–73% in cattle, 1%–38% in pigs, and 0.5%–20% in horses, with higher rates of infection in younger animals. Farm prevalences in production animals vary between 0% and 100%, with the highest prevalence in younger animals. The cumulative incidence on a farm where *Giardia* has been diagnosed is 100% in cattle and goats and nearly 100% in sheep.

Three major morphologic groups have been described: *G muris* from mice, *G agilis* from amphibians, and a third group from various warm-blooded animals. There are at least four species in this third group, including *G ardeae* and *G psittaci* from birds, *G microti* from muskrats and voles, and *G duodenalis* (also known as *G intestinalis* and *G lamblia*), a species complex with a wide mammalian host range infecting people and domestic animals. Molecular characterization has shown that *G duodenalis* is in fact a species complex, comprising seven assemblages (A to G), some of which have distinct host preferences (e.g., assemblage C/D in dogs, assemblage F in cats) or a limited host range (e.g., assemblage E in hoofed livestock), whereas others infect a wide range of animals, including people (assemblage A and B). There is increasing evidence that some assemblages (A and B) that infect domestic animals can also infect people, although transmission patterns are not totally understood. Dogs have mainly assemblages C and D, cats have assemblages A1 and F, and people are infected with assemblages A2 and B; however, some studies have identified human assemblages of *Giardia* in canine fecal samples.

Cycle and Transmission:

Flagellate protozoa (trophozoites) of the genus *Giardia* inhabit the mucosal surfaces of the small intestine, where they attach to the brush border, absorb nutrients, and multiply by binary fission. They usually live in the proximal portion of the small intestine. Trophozoites encyst in the small or large intestine, and the newly formed cysts pass in the feces. There are no intracellular stages. The prepatent period is generally 3–10 days. Cyst shedding may be continual over several days and weeks but is often intermittent,

especially in the chronic phase of infection. The cyst is the infective stage and can survive for several weeks in the environment, whereas trophozoites cannot.

Transmission occurs by the fecal-oral route, either by direct contact with an infected host or through a contaminated environment. Characteristics that facilitate infection include the high excretion of cysts by infected animals and the low dose needed for infection. *Giardia* cysts are infectious immediately after excretion and are very resistant, resulting in a gradual increase in environmental infection pressure. High humidity facilitates survival of cysts in the environment, and overcrowding favors transmission.

Clinical Findings and Lesions:

Giardia infections in dogs and cats may be inapparent or may produce weight loss and chronic diarrhea or steatorrhea, which can be continual or intermittent, particularly in puppies and kittens. Feces usually are soft, poorly formed, pale, malodorous, contain mucus, and appear fatty. Watery diarrhea is unusual in uncomplicated cases, and blood is usually not present in feces. Occasionally, vomiting occurs. Giardiasis must be differentiated from other causes of nutrient malassimilation (e.g., exocrine pancreatic insufficiency and intestinal malabsorption. Clinical laboratory findings usually are normal.

Gross intestinal lesions are seldom evident, although microscopic lesions, consisting of villous atrophy and cuboidal enterocytes, may be present.

Diagnosis:

Because *Giardia* cysts are excreted intermittently, several fecal examinations should be performed if giardiasis is suspected (e.g., three samples collected throughout 3–5 days). *Giardia* may be underdiagnosed, because the cysts are intermittently shed.

For the detection of parasite antigen, immunofluorescence assays and ELISA are commercially available. An in-house ELISA available for use in dogs and cats is a useful tool for clinical diagnosis, particularly when coupled with a centrifugal flotation examination of feces. It is best to test symptomatic animals with a combination of a direct saline smear of feces, fecal flotation with centrifugation, and a sensitive, specific ELISA optimized for use in the animal being tested (e.g., ELISA for dogs and cats).

Treatment:

Fenbendazole (50 mg/kg/day for 5–10 days) effectively removes *Giardia* cysts from the feces of dogs; no adverse effects are reported, and it is safe for pregnant and lactating animals. This dosage is approved to treat *Giardia* infections in dogs in Europe.

Control:

Giardia cysts are immediately infective when passed in the feces and survive in the environment. Cysts are a source of infection and reinfection for animals, particularly those in crowded conditions (e.g., kennels, catteries, or intensive rearing systems for production animals). Feces should be removed as soon as possible (at least daily) and disposed of with municipal waste. Infected dogs and cats should be bathed to remove cysts from the hair coat. Prompt and frequent removal of feces limits environmental contamination, as does subsequent disinfection. Cysts are inactivated by most quaternary ammonium compounds, steam, and boiling water.

To increase the efficacy of disinfectants, solutions should be left for 5–20 min before being rinsed off contaminated surfaces. Disinfection of grass yards or runs is impossible, and these areas should be considered contaminated for at least a month after infected dogs last had access. Cysts are susceptible to desiccation, and areas should be allowed to dry thoroughly after cleaning.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Neosporosis	**5.4.6**	Merck Veterinary Manual Donahoe 2015, Bartels et al. 2007, Lind 2014	Low	Moderate	Low/ Moderate	Negligible	Low

Lind 2014	**Zoonotisk risiko (Hund-> Homo)**	**Impact hos hund**	**Impact hos homo**	**Forekomst (endemicitet)**
Neospora caninum	Ingen	Lav	Ingen	Pan-europæisk

Neosporosis is caused by the protozoan parasite *Neospora caninum*. Neosporosis has been recognized in dogs, cattle, horses, and other animals, but the dog is the definitive host. Infection is uncommon, but can be acquired by ingesting contaminated food and water, or ingesting infected tissues. It may also be transferred from a mother to a fetus still in the womb (transplacentally).

Neospora caninum is a microscopic protozoan parasite with worldwide distribution. Many domestic (e.g., dogs, cattle, sheep, goats, water buffalo, horses, chickens) and wild and captive animals (e.g., deer, rhinoceros, rodents, rabbits, coyotes, wolves, foxes) can be infected. Neosporosis is one of the most common causes of bovine abortion, especially in intensively farmed cows. Neosporosis abortion also occurs in sheep, goats, water buffalo, and camelids, although they may be less susceptible than cattle.

Both puppies and older dogs may be affected. Most severe infections occur in young puppies, which typically develop paralysis of the legs, particularly the hind legs. The paralysis is often progressive and results in rigid contracture of the muscles. In some dogs, only neurologic signs (such as inflammation of the brain and spinal cord) are seen. Disease of the peripheral nerves and spinal nerve roots appears typical of neosporosis. Skin inflammation with sores, inflammation of the liver, pneumonia, and inflammation of the brain may also occur. If not treated promptly, death is likely. There is currently no vaccine.

Epidemiology:

Neosporosis in cattle herds manifests in both endemic and epidemic abortion patterns, but it is also possible for a herd to have a high infection prevalence without a noticeable abortion problem. Both endemic and epidemic transmission patterns in cattle are positively associated with the presence and number of dogs in and around farms. Endemic abortion is mainly associated with recrudescence of latent organisms during pregnancy followed by transplacental transmission to the fetus, although occasional transmission from dogs or other canids may compound the problem. Epidemic abortion is a possible consequence of sudden large-scale transmission to pregnant cattle, presumably by ingestion of a mixed ration or water that has been contaminated with infected canine feces. The use of mixed rations in dairy herds probably accounts for the greater prevalence of neosporosis in dairy cattle than in extensively grazed beef cattle.

Transmission:

Dogs are definitive hosts of *N caninum* and are capable of shedding oocysts in feces after eating tissues of infected animals. Gray wolves, coyotes, and dingoes are also definitive hosts, and many other wild canids are suspected. *Neospora* oocysts have an impervious shell that enables survival in soil and water for prolonged periods after canine feces have decomposed. Intermediate hosts such as cattle become infected by ingesting oocysts. Cattle do not produce oocysts and thus do not transmit infections horizontally to other cattle, but latent infection may endure permanently in their tissues and is transmitted to canids by carnivorism.

Dogs have been shown to become infected by eating infected cattle (including placentas) and deer and are presumed to become infected by consuming raw meat diets, barnyard chickens, and a variety of wild animals.

Control:

It is common for dairy and beef herds to have at least a small percentage of *Neospora*-infected cattle. Although reducing the risk of *Neospora* transmission is a useful goal, complete eradication from a herd is usually impractical. Contamination of feedstuffs used in mixed rations by canine feces should be avoided. Large dairies can consider erecting dog-proof fences around the area in which feedstuffs are stored outdoors, and automatic gates can be installed to facilitate the daily traffic of heavy machinery. Smaller dairy farms may be able to protect feedstuffs within traditional buildings such as barns, grain bins, and silos.

Dead stock, offal from home slaughter, and placentas should be discarded in a manner that prevents ingestion by dogs to reduce the risk that dogs will become infected and shed *Neospora* oocysts on the farm. Dogs seropositive for *Neospora* have reduced likelihood for future shedding of oocysts than do seronegative dogs; therefore, serologic testing of farm dogs is seldom useful.

Zoonotic Risk:

Despite its similarity to *Toxoplasma*, *Neospora* infection has not been clearly associated with any human disease. Laboratory workers should guard against inoculation, which caused fetal lesions in parenterally inoculated primates.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
***Trypanosoma* (Surra)**	**5.4.7**	AUS 2012 OIE 2013 Technical Disease Card	Very Low/Negl.	Negligible	Very Low	Negligible	Negligible

AUS 2012 Risk review

Regarding the importation of dogs and cats the following factors were considered relevant to the biosecurity risk presented by *T. evansi*:

- Surra is widespread in Asia, Africa (north of the tsetse belt), Central and South America, and the Middle East.
- Subclinical infection with *T. evansi* is known to occur in dogs and cats.
- Treatment cannot be relied upon to eliminate infection.
- Although surra is widespread, it affects dogs and cats uncommonly.
- The role that vectors play in the spread of *T. evansi* in association with dogs and cats is poorly defined and other modes of transmission (e.g. feeding on infected tissues) may be more important.
- Although the international movement of pets has resulted in the introduction of a number of exotic diseases to previously surra-free countries, there is no evidence that implicates dogs and cats in the epidemiology of *T. evansi*.

Conclusion Based on the preceding factors, it was concluded that risk management for *T. evansi* was not warranted for dogs or cats or their semen.

AUS 2012 Surra (*Trypanosoma evansi* Infection)

Background Surra is a disease caused by the flagellate protozoan *Trypanosoma evansi*, which can affect most domesticated mammals and some wild species. Infection may be subclinical or result in signs ranging from chronic weight loss to acute death. The disease is most severe in donkeys, horses, mules, deer, camels, llamas, dogs and cats (Geering et al. 1995). Surra is present in Asia, Africa (north of the tsetse belt), Central and South America, and the Middle East (OIE 2010; Radostits et al. 2007). Surra is a multiple-species OIE-listed disease (OIE 2012) and is a nationally notifiable disease in Australia (DAFF 2011). The *Manual of diagnostic tests and vaccines for terrestrial animals* (OIE 2010) includes a chapter on surra, but there are no *Terrestrial animal health code* recommendations for surra relevant to the importation of live animals.

Epidemiology *T. evansi* is transmitted mechanically by biting flies, particularly of the genera *Tabanus*, *Stomoxys*, *Atylous* and *Lyperosia*. Vampire bats have been reported to transmit the parasite in South and Central America (Geering et al. 1995). Mechanical vectors, including horseflies or march flies (family Tabanidae) and stable flies (family Muscidae), are widespread throughout Australia (Seddon and Albiston 1967a; Seddon and Albiston 1967b). Other modes of infection include feeding on infected tissues (Singh et al. 1993), transmission in milk and possibly via the venereal route (CFSPH 2009).

Surra occurs widely throughout Asia although companion animals are uncommonly affected (Irwin and Jefferies 2004). The reason for companion animals being less frequently affected is unclear. Dogs may be less susceptible to bites from tabanids due to their thick fur coat (Hoare 1972), although this is likely to be breed-dependent. Little information is available regarding the importance and epidemiology of many arthropod vectors of companion animals in Asia. Published surveys tend to record resident ectoparasites but tabanid flies are not described; their importance as disease vectors in dogs and cats is poorly understood (Irwin and Jefferies 2004). Non-vector routes of transmission (e.g. ingestion of infected tissues) may be a more important source of infection in dogs (Hoare 1972; Singh et al. 1993) and possibly cats.

Several studies have looked at the introduction of foreign pathogens to countries as a result of international travel of pets (Duscher et al. 2010; Hendrix et al. 1998b). Although the international movement of pets has resulted in the introduction of a number of exotic diseases to previously free countries, there is no evidence that surra has been introduced via companion animals (Hendrix et al. 1998a; Hendrix et al. 1998b). A single case of surra was diagnosed in the Netherlands from a young dog imported directly from Nepal. Despite successful treatment of the infection, the dog subsequently died of complications (Hellebrekers and Slappendel 1982). No additional cases have been reported since.

Clinical signs During the acute phase of the disease, there is intermittent pyrexia, subcutaneous oedema, progressive anaemia, blindness, lethargy and haemostatic abnormalities. During the chronic phase, there is a worsening of clinical signs, and other signs such as cachexia, widespread oedema, corneal opacity, incoordination and posterior paralysis are observed (Da Silva et al. 2010; Singh et al. 1993). Dogs may show severe neurological signs and the disease is often fatal in dogs and cats (Da Silva et al. 2010; Geering et al. 1995; Singh et al. 1993). Dogs may have nervous signs that resemble rabies (OIE 2013), however, dogs may also appear clinically normal despite parasitemia (Irwin and Jefferies 2004).

T. evansi is not known to have a zoonotic potential (OIE 2013).

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Feline spongiform encephalopathy (FSE)	**5.5.1**	CFSPH 2016* NZ Import Risk Analysis 2009**	Negligible	Negligible	High	Negligible	Negligible

*CFSPH 2016: *http://www.cfsph.iastate.edu/Factsheets/pdfs/feline_spongiform_encephalopathy.pdf*
**NZ Import Risk Analysis 2009: *https://www.mpi.govt.nz/dmsdocument/2796/loggedIn*

FSE is a member of the transmissible spongiform encephalopathies (TSEs), a group of neurodegenerative disorders caused by unconventional disease agents. These agents are resistant to the treatments that ordinarily destroy bacteria, spores, viruses and fungi. They are generally thought to be prions, although a minority opinion suggests that TSEs may be caused by virions or retroviruses. Prions are infectious proteins that appear to replicate by converting a normal cellular protein into copies of the prion. The cellular protein, which is called PrPc, is found on the surface of neurons. Pathogenic isoforms of PrPc are designated PrPres; PrPSc or PrPTSE are other names for this protein. Prions that cause different diseases (e.g. FSE or scrapie) are considered to be different strains of PrPres. FSE is caused by the same agent that is responsible for BSE in cattle.

Transmission

The BSE prion is thought to be transmitted to cats when they ingest contaminated bovine tissues. Cooking or rendering does not destroy this agent. Horizontal transmission has not been reported between cats. One TSE in a housecat, reported in 1998, was caused by a prion that was distinct from BSE. The authors suggested that this may have been a new type of FSE. No other infections with this prion have been reported in cats.

Feline spongiform encephalopathy has been described in a captive cheetah, puma, an ocelot, and a tiger from zoological collections in Great Britain.

In addition to the non-domestic felids, 87 domestic cats in Great Britain and sporadic cases in Norway, Northern Ireland and Liechtenstein have been diagnosed with FSE. All cats were > 2 years old. Clinically, affected cats initially demonstrated behavior changes (more timid or aggressive), with subsequent ataxia, hypermetria, and hyperesthesia to sound and touch. Histopathology revealed spongiform degeneration in the neuropil of the brain and spinal cord with the most severe lesions localized to the medial geniculate nucleus of the thalamus and the basal nuclei.

A ban on bovine spleen and CNS tissue in pet foods was initiated in 1990, and all but one of the FSE cases to date occurred in cats born prior to the ban.

Feline spongiform encephalopathy (FSE) belongs to the transmissible spongiform

encephalopathies (TSEs) and their aetiological agents are generally considered to be prions.
These are infectious protein agents that affect the central nervous system causing neurodegenerative disease in humans and animals. FSE was first recognised during the bovine spongiform encephalopathy (BSE) epidemic in Britain. The first case in a felid was diagnosed in 1990. There is no evidence that FSE occurs in any manner other than through ingestion of contaminated food containing the BSE agent. TSEs have long incubation periods and development of clinical signs in the cat takes about five years. There is no evidence of vertical transmission of TSEs in the cat.
Approximately 90 cases of FSE were reported worldwide to 2004, predominantly from the UK.
FSE has probably now disappeared, since world-wide strict measures are in place to exclude BSE infected cattle from entering the food chain. There have been no reports from the UK since 2001. TSE has not been reported in the dog. The likelihood of importing an infected cat is remote. In addition FSE is not contagious and cats are extremely unlikely to end up in the food chain. Therefore the likelihood that the agent could be imported and transmitted to other animals is negligible.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Paragonimus westermani	**5.6.1**	Merck Veterinary Manual Liu et al.2008 Parasitipedia.net	Very low	Very low	Moderate	Moderate	Very low

Paragonimus kellicotti and *P westermani* usually are found in cysts, primarily in the lungs of dogs, cats, and several other domestic and wild animals. They also have been found rarely in other viscera or the brain. Infection is most common in China, southeast Asia, and North America.
Human infection with *P. westermani* occurs by eating inadequately cooked or pickled crab or crayfish that harbor metacercariae of the parasite. The metacercariae excyst in the duodenum, penetrate through the intestinal wall into the peritoneal cavity, then through the abdominal wall and diaphragm into the lungs, where they become encapsulated and develop into adults. The worms can also reach other organs and tissues, such as the brain and striated muscles, respectively. However, when this takes place completion of the life cycles is not achieved, because the eggs laid cannot exit these sites. Time from infection to oviposition is 65 to 90 days. Infections may persist for 20 years in humans.
Cats and dogs are considered the most important animal hosts in the endemic regions in China. Unlike other countries, most cats and dogs are not kept as pets in China but usually roam freely in villages and cities and, thus, have ready access to crabs or crayfish infected with lung flukes. In endemic regions studied to date, most cats and dogs examined have proven to be infected with the parasite. The intensity of infection has been as high as 34 adult worms per dog.

The adult flukes are fleshy, reddish brown, oval, and ~14 × 7 mm. The eggs are golden brown, oval, distinctly operculated, and ~100 × 60 µm. The eggs pass through the cyst wall, are coughed up, swallowed, and passed in the feces. The life cycle includes several snails as the first intermediate host, and crayfish or crabs as the second. Dogs and cats become infected by eating raw crayfish or crabs that contain the encysted cercariae. After penetrating the intestinal wall and wandering in the peritoneal cavity, the young flukes pass through the diaphragm to the lungs, where they become established.
Infected animals may have a chronic, deep, intermittent cough and eventually become weak and lethargic, although many infections pass unnoticed. A diagnosis is based on finding the characteristic eggs in feces or coughed-up material. The location of the flukes in the lungs is determined by x-ray. Several drugs provide effective treatment for lung fluke infections.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Schistosoma japonicum	**5.6.2**	Merck Veterinary Manual Carabin et al. 2015 CDC*	Very low	Uncertain	Moderate	High	Uncertain

* *https://www.cdc.gov/dpdx/schistosomiasis/index.html*

Schistosomiasis

Schistosomes are members of the genus *Schistosoma*, family Schistosomatidae. Adult worms are obligate parasites of the vascular system of vertebrates. Schistosomes are dioecious.

S japonicum is endemic in several countries of the Far East. Dogs and cats acquire schistosomiasis when the initial parasitic infection penetrates the skin. The first and beginning stage of infection is known as cercaria. Dogs and cats usually contract schistosomiasis from contaminated fresh water which contains the snail that hosts the infection. In areas of high risk of infection, standing water should be avoided by dogs and cats. Human contact with water is thus necessary for infection by schistosomes. Various animals, such as dogs, cats, rodents, pigs, horse and goats, serve as reservoirs for *S. japonicum*, and dogs for *S. mekongi*.

In the first stage on infection, the parasite burrows into the skin, maturing into the second stage, known as schistosomula. It then migrates to the lungs and liver, where it matures into the adult form. The adult worm then migrates to its final and preferred area of the body, which typically includes the bladder, rectum, intestines, liver, and/or veins that carry blood from the intestines to liver, spleen, and lungs. Eggs are then shed via feces.

If the shed eggs are exposed to fresh water, the parasite hatches and infects a snail. Once inside the snail, the eggs mature and look for a feline or canine host to complete the life cycle. Once a dog or cat is infected, an inflammatory response is triggered throughout the body and the infection spreads, known as disseminated visceral granulomata.

Epidemiology:

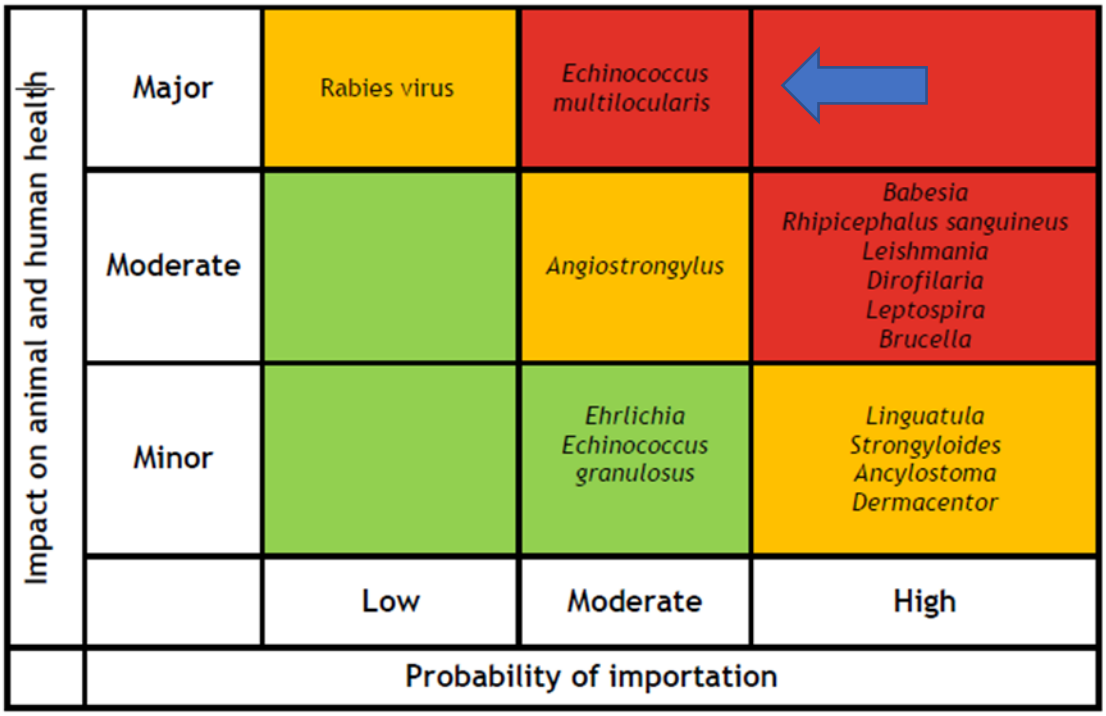
A relatively high infection prevalence was discovered in dogs (and cats) in two geographical regions of China, together with a relatively high transmission index (eggs per day) estimate, raising potentially important implications for current control criteria of S. japonicum in China, which is essentially based on infection in humans and bovines, rather than any current concern over other less agriculturally important domestic animals. However, from parasitological data alone here it is unclear whether transmission could be maintained within dogs in the absence of bovines in the marshland or of rodents in the hilly regions, or whether they are mainly just 'spill-over' hosts.

The role that animals play in the transmission of Schistosoma japonicum to humans in the Philippines remains uncertain and prior studies have not included several species, adjustment for misclassification error and clustering, or used a cohort design. Dogs, cats and rats were found to play a role in schistosomiasis transmission, with dogs and rats showing more consistent effects. Therefore, schistosomiasis elimination efforts should not exclude the possibility of targeting infection in dogs, and possibly cats, as well as in humans, as this approach could improve the effectiveness of MDA programs in this area of the Philippines. Rats could be considered as a good sentinel to monitor infection levels in the environment.

Further genetic and etiological research is needed to understand the role of other animals in transmission to humans, as well as the environmental factors associated with the effect of the region of residence. Employing a One Health approach which would involve experts in environmental sciences, veterinary and human medicine may contribute to the elimination of schistosomiasis in human communities.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Echinococcus multilocularis	**5.7.1**	Merck Veterinary Manual, EFSA 2015, Lind 2014, Høgåsen 2012, Rijks et al. 2016, OIE TAHC, chapt. 8.6, 2017, EUC 2017	Moderate	Moderate	Negligible	High	Moderate

Lind 2014	**Zoonotisk risiko (Hund-> Homo)**	**Impact hos hund**	**Impact hos homo**	**Forekomst (endemicitet)**
Echinococcus multilocularis	Moderat	Ingen	Alvorlig	Central-Europa (inkl. DK)



From Høgåsen et al. 2012

OIE TAHC Article 8.6.5. Recommendations for the importation of dogs and wild canids from an infected country:

Veterinary Authorities of *importing countries* should require the presentation of an *international veterinary certificate* attesting that:

1) the animal has been treated between 24 and 72 hours prior to embarkation with praziquantel (5 mg/kg), or another cestocidal product with a comparable efficacy against intestinal forms of *E. multilocularis*;

2) adequate precautions have been taken to avoid reinfection of the animal between treatment and embarkation.

EU Commission delegated regulation of 21.11.2017 with regard to preventive health measures for the control of *E. multilocularis infection in dogs:*

The regulation lays down rules for the application of preventive health measures for the control of E.m. infection in dogs intended for non-commercial movement into the territory or part of the territory of certain Member States (Malta, Finland, Ireland, the United Kingdom). Yearly surveys to document freedom from E.m. primarily in foxes are mandatory for these Member States.

Foxes are the definitive host for *Echinococcus multilocularis*. Microtine rodents (such as voles) are the intermediate hosts. This parasite has been rarely found in the brain of people, in which the invasive, thin-walled multilocular hydatid cysts produce innumerable exogenous daughter cysts that bud protoscolices. Surgical intervention is more successful in removing unilocular hydatid cysts of *E granulosus*.

Most urban dogs and cats eat prepared foods and have restricted access to natural prey. Suburban, rural, and hunting dogs have more access to various small mammals, in addition to raw meat and offal from domestic and wild ungulates. A number of cestodes can be expected in such dogs. On sheep ranges and wherever wild ungulates and wild canids are common, dogs may acquire *Echinococcus granulosus* (the hydatid tapeworm). Sylvatic *Echinococcus multilocularis* (the alveolar hydatid tapeworm), previously known only from arctic North America, has been found in wildlife in midwestern and western USA and Canada. The parasite is also endemic in many parts of central and eastern Europe, particularly France, Germany, and Switzerland. Thus far, infections in cats or dogs are generally rare. However, in addition to multiple reports from dogs and cats in central Europe, the parasite has recently been identified in a few dogs across Canada.

Association with infected dogs may result in human infection with metacestodes of *E granulosus*, *E multilocularis*, *T multiceps*, *T serialis*, or *T crassiceps* in various tissues (by ingestion of eggs passed in dog feces), or adult *D caninum* in the intestine (by ingestion of infected fleas). The presence of meta-cestodes in livestock may limit commercial use of such carcasses or offal meats. Thus, cestodes of dogs and cats may be of both economic and public health importance:

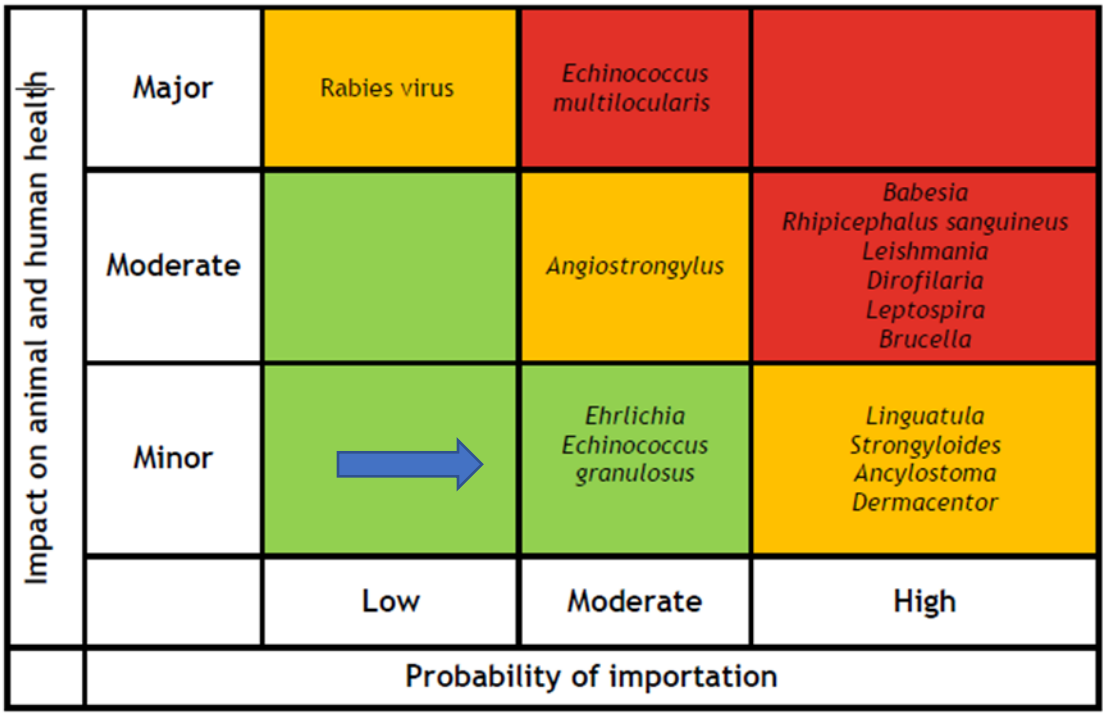
Cestode[a]	Host of Adult Worm	Name of Metacestode (Intermediate) Stage	Measurements of Metacestode	Principal Intermediate Hosts	Site of Metacestode
Echinococcus multilocularis	Canids and domestic cats	Alveolar hydatid cyst	Variable, penetrates like neoplastic tissue	Field mice, voles, lemmings, sometimes domestic mammals and people	Usually liver, various other organs and tissues

Adult cestodes in the intestine of dogs and cats rarely cause serious disease, and clinical signs, if present, may depend on the degree of infection, age, condition, and breed of host. Clinical signs vary from unthriftiness, malaise, irritability, capricious appetite, and shaggy coat to colic and mild diarrhea; rarely, intussusception or blockage of the intestine, emaciation, and seizures are seen.

Control of tapeworms of dogs and cats requires therapy and prevention. Animals that roam freely often become reinfected by ingestion of metacestodes in carrion or prey animals. Effective treatment should remove the attached scolices from the small intestine of infected animals. Praziquantel is approved for treatment of *Echinococcus* spp. For cats praziquantel are approved for treatment of *D caninum*, and praziquantel is approved for treatment of *E multilocularis*. Outside the USA and UK, praziquantel is approved for use in multiple countries at 5 mg/kg for treatment of *J pasqualei* in dogs (as praziquantel/pyrantel/febantel) and cats (as praziquantel/pyrantel).

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Echinococcus granulosus	**5.7.2**	Merck Veterinary Manual, EFSA 2015, Lind 2014, Høgåsen et al. 2012, Rijks et al. 2016, Sigurdarson, 2010, OIE TAHC, chapt. 8.5, 2017, EUC 2017 Davidson et al. 2016, Deplaze et al. 2011	Moderate	Moderate	Negligible	High	Moderate

Lind 2014	**Zoonotisk risiko (Hund-> Homo)**	**Impact hos hund**	**Impact hos homo**	**Forekomst (endemicitet)**
Echinococcus granulosus	Moderat	Ingen	Alvorlig	Syd-Europa



From Høgåsen et al. 2012

OIE TAHC Article 8.5.5.

Recommendations for the importation of dogs and wild canids from an infected country

Veterinary Authorities of *importing countries* should require the presentation of an *international veterinary certificate* attesting that:

1) the animal has been treated between 24 and 72 hours prior to embarkation with praziquantel (5 mg/kg), or another cestocidal product with comparable efficacy against intestinal forms of *E. granulosus*;

2) adequate precautions have been taken to avoid reinfection of the animal between treatment and embarkation.

Echinococcus granulosus is a tapeworm found in the small intestine of the canid definitive host. Its eggs are ingested by the intermediate hosts, wild and domestic herbivores, e.g., sheep, cattle, and moose. People can also serve as intermediate hosts. After hatching in the intestine of the intermediate host, the oncospheres invade the circulatory system and lodge in various organs (the liver and lungs), where they develop into large, thick-walled, unilocular hydatid cysts that bud protoscolices endogenously. Hydatids have been rarely reported in the CNS of domestic animals and are rare in people, in which they produce symptoms similar to those of a brain tumor.

Most urban dogs and cats eat prepared foods and have restricted access to natural prey. Suburban, rural, and hunting dogs have more access to various small mammals, in addition to raw meat and offal from domestic and wild ungulates. A number of cestodes can be expected in such dogs. On sheep ranges and wherever wild ungulates and wild canids are common, dogs may acquire *Echinococcus granulosus* (the hydatid tapeworm).

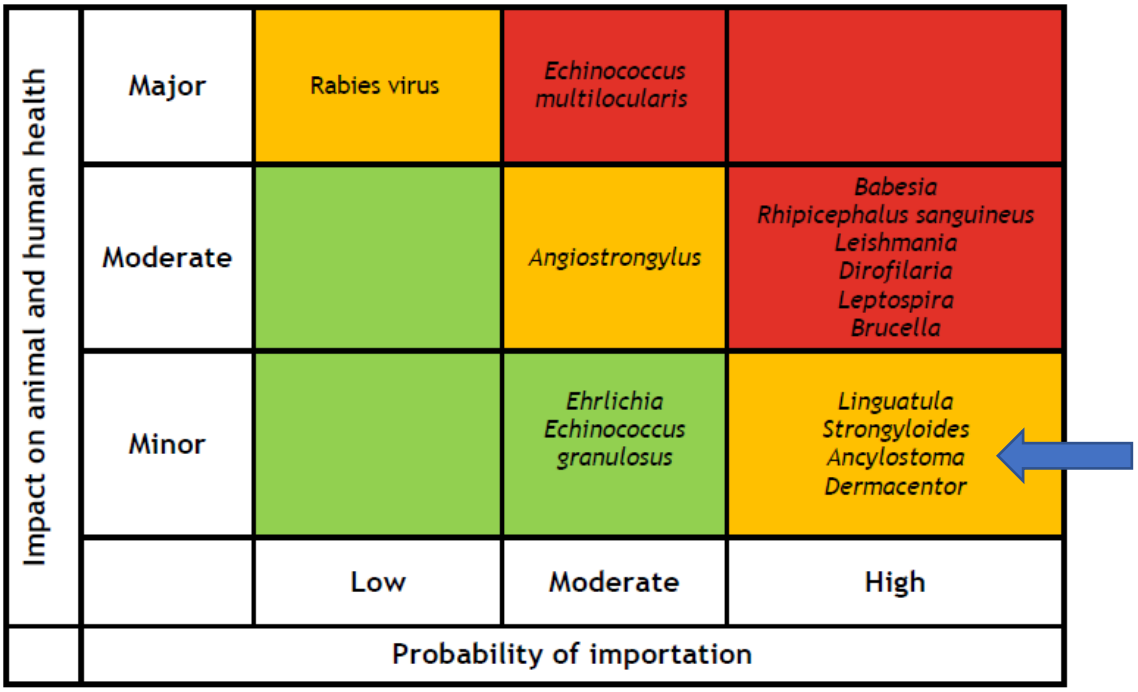
Association with infected dogs may result in human infection with metacestodes of *E granulosus*, *E multilocularis*, *T multiceps*, *T serialis*, or *T crassiceps* in various tissues (by ingestion of eggs passed in dog feces), or adult *D caninum* in the intestine (by ingestion of infected fleas). The presence of metacestodes in livestock may limit commercial use of such carcasses or offal meats. Thus, cestodes of dogs and cats may be of both economic and public health importance.

However, in the period 1953-1979, cysts of echinococcus were recorded in a total of 21 old ewes, all of which came from few farms on 2 small areas in East-Iceland. There was an indication that the parasite had been introduced to Iceland by an imported dog. After 1979 no hydatid cysts have been found in any animal in Iceland (Sigurdsson, 2010).

Cestode[a]	Host of Adult Worm	Name of Metacestode (Intermediate) Stage	Measurements of Metacestode	Principal Intermediate Hosts	Site of Metacestode
Echinococcus granulosus	Dogs, wolves, foxes, and several other wild carnivores	Hydatid cyst	Diameter 50–100 mm, sometimes ≥150 mm	Sheep, cattle, pigs, horses, moose, deer; occasionally people	Commonly in liver and lungs, occasionally in other organs and tissues

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Ancylostoma caninum	**5.8.1**	Merck Veterinary Manual, Høgåsen et al. 2012, Lind 2014 Fjøludottir 2013	High	Moderate	Moderate	Low/ Moderate	Low

Lind 2014	Zoonotisk risiko (Hund-> Homo)	Impact hos hund	Impact hos homo	Forekomst (endemicitet)
Ancylostoma caninum	Moderat	Moderat- Alvorlig	Lav-Moderate	Syd-og Central-Europa



From: Høgåsen et al. (2012)

Hookworms in Small Animals

Ancylostoma caninum is the principal cause of canine hookworm disease in most tropical and subtropical areas of the world. *A tubaeforme* of cats has a similar but more sparse distribution. *A braziliense* of dogs and cats is sparsely distributed from Florida to North Carolina in the USA. It is also found throughout

Central and South America and Africa. *A ceylanicum* of dogs, cats, and people is widely distributed throughout Asia, the Middle East, and parts of South America. *Uncinaria stenocephala* is the principal canine hookworm in cooler regions; it appears to be the predominant canine hookworm in Canada and the northern fringe of the USA, where it is primarily a fox parasite. *U stenocephala* also is seen in cats. It has also been found in arctic foxes in IS. *A caninum* males are ~12 mm long, females, ~15 mm; the other species are somewhat smaller. The infective larvae of canine hookworms, particularly those of *A braziliense*, may penetrate and wander under the skin of people and cause **cutaneous larva migrans**.

The elongate (>65 µm), thin-walled, hookworm eggs in the early cleavage stages (2–8 cells) are first passed in the feces 15–20 days after infection; they complete embryonation and hatch in 24–72 hrs. on warm, moist soil. Transmission may result from ingestion of infective larvae from the environment and additionally, in the case of *A caninum*, via the colostrum or milk of infected bitches. Infections with *A caninum*, *A braziliense*, *A tubaeforme*, or *A ceylanicum* can also result from larval invasion through the skin, but this route is of little significance for *U stenocephala*. Skin penetration in young pups is followed by migration of the larvae through the blood to the lungs, where they are coughed up and swallowed to mature in the small intestine. However, in animals >3 mo. old, *A caninum* larvae, after migration through the lungs, are arrested in the somatic tissues. These arrested larvae are activated during pregnancy, then accumulate in the mammary glands. Arrested development may also occur in the mucosa of the small intestine; activation may occur after removal of adult worms from the intestine.

Clinical Findings:

An acute normocytic, normochromic anemia followed by hypochromic, microcytic anemia in young puppies is the characteristic, and often fatal, clinical manifestation of *A caninum* infection. Surviving puppies develop some immunity and show less severe clinical signs. Nevertheless, debilitated and malnourished animals may continue to be unthrifty and suffer from chronic anemia. Mature, well-nourished dogs may harbor a few worms without showing signs; they are of primary concern as the direct or indirect source of infection for pups. Diarrhea with dark, tarry feces accompanies severe infections. Anemia, anorexia, emaciation, and weakness develop in chronic disease.

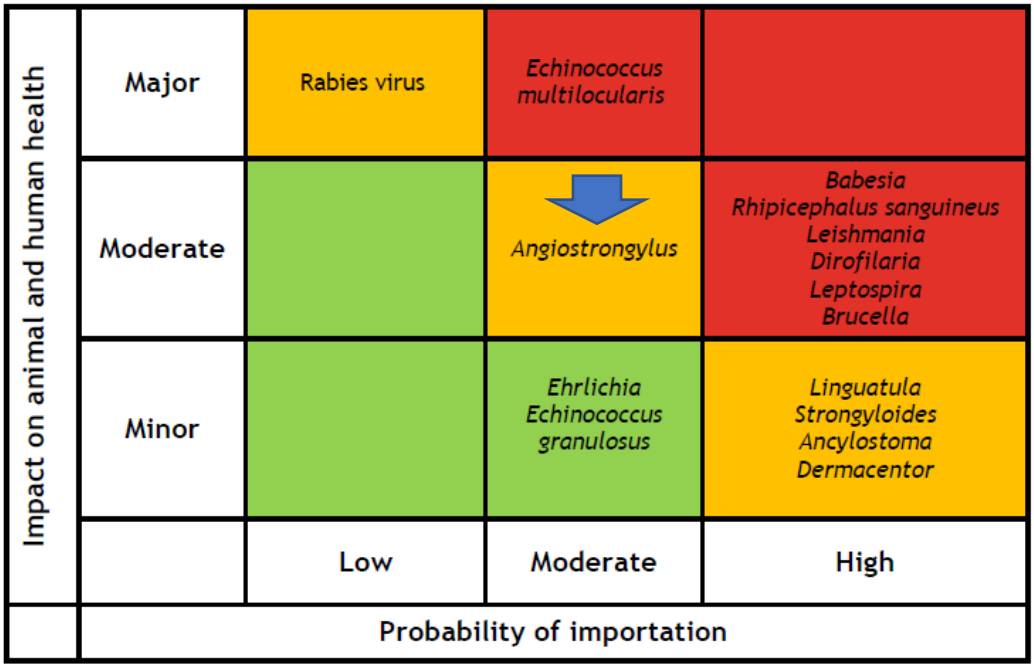
Lesions:

Anemia results directly from the bloodsucking and the bleeding ulcerations that result when *A caninum* shift feeding sites. The amount of blood loss due to a single worm in 24 hrs. has been estimated to be up to 0.1 ml. There is no interference with erythropoiesis in uncomplicated hookworm disease. The liver and other organs may appear ischemic, and some fatty infiltration of the liver may occur. Hemorrhagic enteritis with a swollen intestinal mucosa that shows red, small ulcers and attached worms is usually seen in acute, fatal cases. *A braziliense*, *A tubaeforme*, *A ceylanicum*, and *U stenocephala* are not avid blood feeders, and anemia rarely develops. However, hypoproteinemia is characteristic, and serum seepage around the site of attachment in the intestine may reduce blood protein by >10%.

In dogs, dermatitis due to larval invasion of the skin may be seen with any of the hookworms but has been seen most frequently in the interdigital spaces with *U stenocephala*; skin infections with *U stenocephala* rarely mature. Pneumonia and lung consolidation may result from overwhelming infections in pups.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Angiostrongylus vasorum	**5.8.2**	Merck Veterinary Manual AUS 2012, Høgåsen et al. 2012, Willesen et al. 2014, Lind 2014, Thordarson 2018	Moderate	Moderate	Mod./High	Negligible	Moderate

Lind 2014	**Zoonotisk risiko (Hund-> Homo)**	**Impact hos hund**	**Impact hos homo**	**Forekomst (endemicitet)**
Angyostrongylus vasorum	Ingen	Alvorlig	-	Pan-europæisk (inkl. DK)



From: Høgåsen et al. (2012)

AUS 2012 Current biosecurity measures

There are no specific biosecurity measures for Canine Pulmonary Angiostrongylosis (CPA). Current import conditions for internal parasites have the following requirements:

• Within four days before export, dogs and cats must be treated with an approved anthelmintic that is effective against nematodes and cestodes. The active ingredients and dose rate must be recorded on the veterinary certificate.

Risk review

Regarding the importation of dogs and cats, the following key factors were considered relevant to the biosecurity risk presented by CPA:

• CPA is not an OIE-listed disease and it is not a nationally notifiable disease in Australia.

• *A. vasorum* has a wide geographic distribution and is known to occur in dog populations in Europe, North and South America, and Africa.

• There is a history of importation of dogs from infected countries, with one case of CPA reported in Australia in 2007.

• Prevalence estimates in endemically infected countries are not readily available, but are likely to vary considerably between regions and within a population based on exposure to infected intermediate or paratenic hosts.

• The anthelmintic treatment specified in the existing import requirements is aimed at parasites of the gastrointestinal tract (roundworm, whipworm, hookworm, tapeworm) and is unlikely to eliminate infection with *A. vasorum*.

• Recommended diagnostic testing (Baermann technique) has a relatively low sensitivity of detection (i.e. a high number of false negative results) making it unsuitable as a quarantine screening test.

• There is limited information on the efficacy of treatment options in dogs in the general population. Macrocyclic lactones appear to be reasonably effective in reducing parasitic burdens and clinical manifestations.

Conclusion

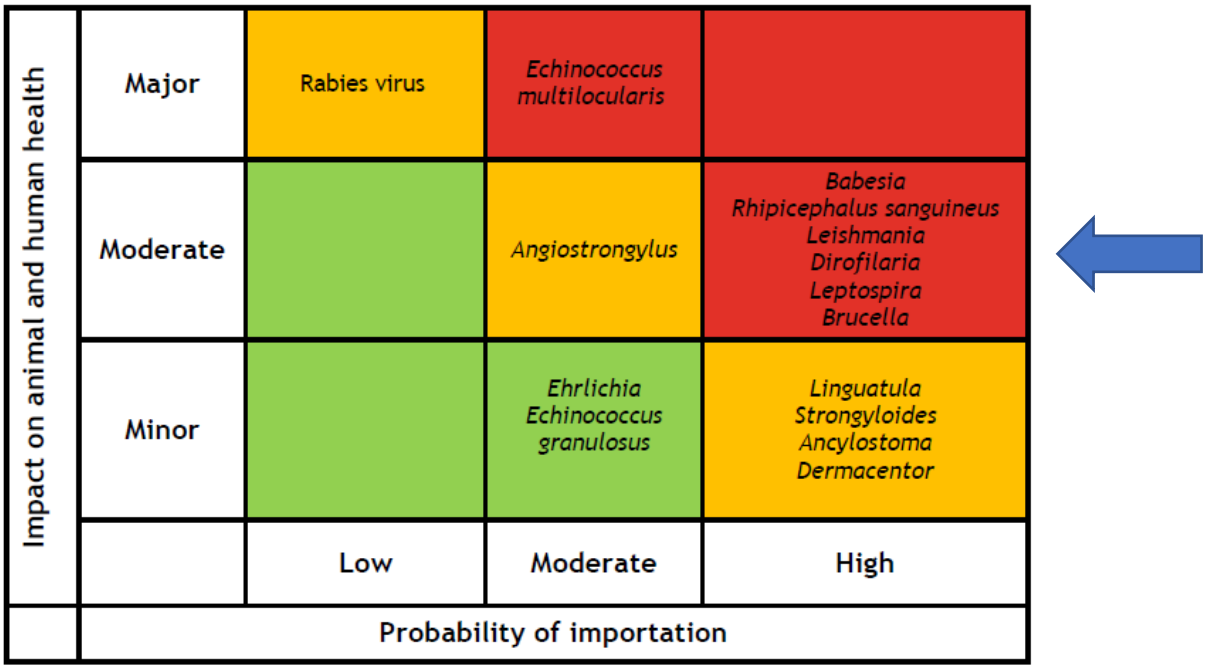
Based on the preceding factors, it was concluded that risk management for CPA is not warranted for either dogs or dog semen.

Angiostrongylus vasorum, the French heartworm, is a parasitic nematode that has dogs and other carnivores (e.g. foxes, wolves, coyotes, badgers) as final hosts. *Angiostrongylus vasorum* has an indirect life cycle, with dogs and other carnivores as final hosts, and snails (*Achatina, Bradybaena, Helix*, etc.), slugs (*Limax, Deroceras*, etc.) or other freshwater mollusks (*Biomphalaria, Physa*, etc.) as intermediate hosts. Several small vertebrates can act as transport hosts (= paratenic hosts), e.g. frogs, lizards, rats, etc. The worms do not complete development in these transport hosts, but remain encysted until a suitable final host eats them or their carcasses. *Angiostrongylus vasorum* is native to Western Europe (France, Spain, Germany, Great Britain, etc.) but it is clearly spreading to other European countries. It has been also found in North America, and some Asiatic and African regions. There are even some reports from South America. Studies in some European countries showed that up to 7.5 % of dogs with undefined pulmonary symptoms were infected with this parasite. Other studies showed that up to 25% of the foxes in a population may be infected. It seems that there is a correlation between incidence in foxes and in dogs, suggesting that foxes are the main reservoir in many regions. *Angiostrongylus vasorum* is quite harmful for dogs.

Angiostrongylus vasorum does not affect cats. The first canine case in Denmark was reported in 1983 in a dog returning from a stay in France. The disease has since spread gradually to the whole of the country, the main focus still being around Copenhagen and on the island of Zealand. In a survey among dogs with relevant clinical signs in 2012 - 2013, 24 out of 171 dogs (14%) had heartworms. In December 2017, a case was discovered in the Icelandic quarantine facility in two dogs imported from Switzerland.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Dirofilaria immitis	**5.8.3**	Merck Veterinary Manual Høgåsen et al. 2012, Lind 2014	High	Moderate	Moderate/ High	Moderate	Moderate

Lind 2014	Zoonotisk risiko (Hund-> Homo)	Impact hos hund	Impact hos homo	Forekomst (endemicitet)
Dirofilaria immitis	Moderat	Alvorlig	Moderat	Syd- og øst Europa



From Høgåsen et al. 2012

Heartworm (HW) disease is caused by the filarial organism, *Dirofilaria immitis*. At least 70 species of mosquitoes can serve as intermediate hosts; *Aedes*, *Anopheles*, and *Culex* are the most common genera acting as vectors. Patent infections are possible in numerous wild and companion animal species. Wild animal reservoirs include wolves, coyotes, foxes, California gray seals, sea lions, and raccoons. In companion animals, HW infection is diagnosed primarily in dogs and less commonly in cats and ferrets. HW disease has been reported in most countries with temperate, semitropical, or tropical climates, including the USA, Canada, Australia, Latin America, and southern Europe. In companion animals, infection risk is greatest in dogs and cats housed outdoors. Although any dog or cat, indoor or outdoor, is

capable of being infected, most infections are diagnosed in medium- to large-sized, 3- to 8-yr-old dogs living outside.

HW infection rates in other companion animals such as ferrets and cats tend to parallel those in dogs in the same geographic region. No age predilection has been reported in ferrets or cats, but male cats have been reported to be more susceptible than females. Indoor and outdoor ferrets and cats can be infected. Other infections in cats, such as those caused by the feline leukemia virus or feline immunodeficiency virus, are not predisposing factors.

Life Cycle:

Mosquito vector species acquire microfilariae (a neonatal larval stage) while feeding on an infected host. Once ingested by the mosquito, microfilariae develop into the first larval stage (L_1). They then actively molt into the second larval stage (L_2) and again to the infective third stage (L_3) within the mosquito in ~1–4 wks., depending on environmental temperatures. This development phase requires the shortest time (10–14 days) when the average ambient temperature is >81°F (27°C) and the relative humidity is 80%. When mature, the infective larvae migrate to the labium of the mosquito. As the mosquito feeds, the infective larvae erupt through the tip of the labium with a small amount of hemolymph onto the host's skin. The larvae migrate into the bite wound, beginning the intra-mammalian phase of the life cycle. A typical *Aedes* mosquito is capable of surviving the complete development of only small numbers of HW larvae, usually <10 larvae per mosquito.

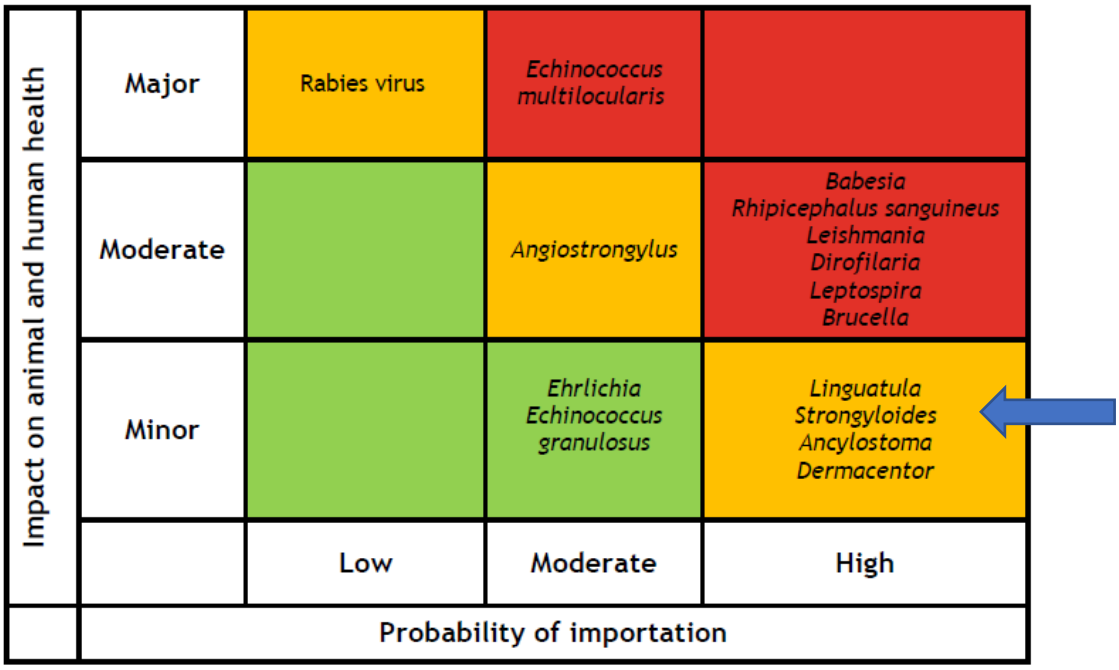
In canids and other susceptible hosts, infective larvae (L_3) molt into a fourth stage (L_4) in 3–12 days. After remaining in the subcutaneous tissue, abdomen, and thorax for ~2 mo., L_4 undergo their final molt at day 50–70 into young adults, arriving in the heart and pulmonary arteries ~70–120 days after initial infection. Only 2.5–4 cm in length on arrival, worms rapidly grow in the pulmonary vasculature to adult worms (males ~15 cm long, females ~25 cm). When juvenile heartworms first reach the lungs, blood flow forces them into the more distal small pulmonary arteries of the caudal lung lobes; as the parasites grow, they occupy larger and larger pulmonary arteries, moving into the right ventricle and atrium when the worm burden is high. Gravid females produce microfilariae as early as 6 mo. after infection but more typically at 7–9 mo. after infection.

Microfilariae are detectable in most infected canids (~80%) not receiving macrolide prophylaxis and occasionally in those dogs placed on macrolide preventives when a HW infection was already present. The number of circulating microfilariae does not correlate well to the adult female HW burden. Adult worms typically live 3–5 yrs., whereas microfilariae may survive for up to 2 yrs. in the dog, while awaiting arrival of a mosquito intermediate host.

Most dogs are highly susceptible to HW infection, and most (an average of 56%) experimentally administered infective larvae (L_3) develop into adults. Ferrets and cats are susceptible hosts, but the rate of infective larvae developing into adults is low (an average of 6% in cats and 40% in ferrets). In cats, the adult burden is often only one to three worms. It appears that early death of juvenile worms on arrival at the pulmonary vasculature is largely responsible for the heartworm-associated respiratory disease (HARD) syndrome in cats. HARD does not require maturation of heartworms but is due to the body's response to the dying/dead heartworms. When maturation does occur, adult worm survival in cats is typically not longer than 2–3 yr. In all animals capable of being infected, aberrant larval migration may occur, resulting in parasitic lesions in the CNS, systemic arterial system, and in visceral and subcutaneous sites.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Strongyloides stercoralis	**5.8.4**	Merck Veterinary Manual Høgåsen et al. 2012, Lind 2014, Eydal & Skirnisson 2016	High	Low/ Moderate	Low/ Moderate	Low/ Moderate	Low/ Moderate

Lind 2014	Zoonotisk risiko (Hund-> Homo)	Impact hos hund	Impact hos homo	Forekomst (endemicitet)
Strongyloides stercoralis	Lav	Moderat	Moderat	Pan-europæisk (sydlig)



From Høgåsen et al. 2012

Strongyloides worms, called also threadworms (in the US) or pinworms (in the UK) represent a genus of parasitic roundworms that affects many domestic and wild vertebrate species, including dogs, cats and humans. They are found worldwide in tropical and subtropical regions of Asia, Africa and America, mainly

in rural areas with poor sanitation standards. *Strongyloides stercoralis* is the best investigated *Strongyloides* species because of its importance as a human parasite. Other *Strongyloides* species of veterinary importance behave similarly, but their life cycles are not completely elucidated. *Strongyloides stercoralis* has a special and complex life cycle. It can complete its development both asexually and bisexually. *Strongyloides* infections in adult dogs are mostly benign and almost asymptomatic, perhaps a light diarrhea. Unusual severe infections can cause loss of appetite, weakness, weight loss, dehydration, fever and shallow breathing. However, infection of puppies, especially through the mother milk may be serious and even fatal. Lung damage due to worm-induced pneumonia, gut inflammation (enteritis) with bleeding and damage to the gut's wall, and mucous diarrhea have been reported. The risk is especially high if animals are crowded under hot and humid conditions with poor sanitation. *Strongyloides stercoralis* is also a human parasite that can be transmitted from dogs to humans (mainly through the skin). Human infections are also benign for people with a healthy immune system. However, for people with a weak immune system (e.g. HIV patients) *Strongyloides stercoralis* infections can be very serious and often fatal. In cats, *Strongyloides tumefaciens* is usually also benign and asymptomatic. Occasionally small white nodules may develop in the thick intestine, associated with diarrhea. Diagnosis is based on detection of eggs and/or larvae (~600 micrometers long) in the feces.

Strongyloides stercoralis is a small, slender nematode that when fully mature is ~2 mm long, located at the base of the villi in the anterior half of the small intestine of dogs and cats. The worms are almost transparent and all but impossible to see grossly at necropsy. Usually, infections are associated with warm, wet, crowded, unsanitary housing. The species found most often in dogs is identical to that found in people.

The parasitic worms are all females. The eggs embryonate rapidly, and most larvae hatch before being passed in the feces. Under appropriate conditions of warmth and moisture, development in the environment is rapid; the third larval stage may be reached in little more than a day. Some of these larvae develop into infective filariform larvae; others develop into free-living worms that mate and produce progeny similar to that of the parasitic female. The filariform larvae penetrate the skin but also may infect a host via ingestion. Trans-mammary transmission is possible. Progeny may be shed in the feces 7–10 days after infection. Autoinfection caused by larvae that developed to the infective stage within the GI tract can result in infections in which dogs shed larvae for lengthy periods.

Control:

Poor sanitation and mixing of susceptible with infected dogs can lead to a rapid buildup of the infection in all dogs in a kennel or pen. Dogs with diarrhea should be promptly isolated from dogs that appear healthy. Direct sunlight, increased soil or surface temperatures, and desiccation are deleterious to all free larval stages. Thorough washing of wooden and impervious surfaces with steam or concentrated salt or lime solutions, followed by rinsing with hot water, effectively destroys the parasite. Because the disease in people can be serious, caution should be exercised when handling infected dogs. The disease in people (as in dogs) is much more likely to be severe if the person is immunosuppressed.

Case Report (Eydal & Skirnisson, 2016)

The first case of *S. stercoralis* infection diagnosed outside quarantine in Iceland was in an unhealthy household puppy purchased from an Icelandic breeding kennel (Kennel A) in 2012. A total of nine puppies

purchased from Kennel A, and two dogs which had contact with dogs from the kennel, were diagnosed with *S*. *stercoralis*. Kennel dogs: In 2012 *S*. *stercoralis* was confirmed in dozens of dogs in Kennel A. Follow-up examinations after anthelmintic treatments indicated a successful removal of worms in imported and household dogs. In spite of more than a dozen anthelmintic treatment actions and other arrangements in Kennel A since 2012, recurrent infections have repeatedly been confirmed, the last one in 2015. The nematode is believed to have been introduced to the breeding kennel with an imported dog, in spite of anthelmintic treatments in quarantine.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Cheyletiellosis	**5.9.1**	Merck Veterinary Manual Skirnisson et al. 1997, 2017	Indigenous*	Present*	Moderate	High	n.a.*

**Cheyletiella spp*. have been identified in Iceland with sporadic occurrence in dogs (Skirnisson 1997, Skirnisson et al. 2017).

Mange is caused by microscopic mites that invade the skin of otherwise healthy animals. The mites cause irritation of the skin, resulting in itching, hair loss, and inflammation. All forms of mange are highly contagious. Both dogs and cats are very susceptible. Horses and other domestic animals can also be infected. There are several types of mange that affect dogs, including canine scabies (sarcoptic mange), ear mites (otodectic mange), walking dandruff (cheyletiellosis), and trombiculosis. Demodicosis is not considered mange, but it is also caused by mites.

Cheyletiella yasguri mites cause walking dandruff in dogs. (The dandruff that is seen "walking" is actually the mites moving about on the skin of the dog.) Although these mites often stay on their preferred hosts, infections across species are possible. Walking dandruff is very contagious, especially in kennels, catteries, or multi-pet households. Regular use of certain insecticides to control flea infestations has a side benefit of often controlling the mites that cause walking dandruff. Humans can also be infested with these species of mites. Mites that cause walking dandruff spend their entire 3-week life cycle on their host.

Scaling of the skin and infestation along the back are common signs of walking dandruff. Intense itching is frequent, though some animals do not itch at all. Pets that show no signs can carry the mites and transmit them to other pets and humans.

Although a definitive diagnosis is usually made by examining the mites with a microscope, a tentative diagnosis is often made based on the presence of mites and an examination of the animal's skin. The mites and eggs are hard to find, especially on animals that are bathed often.

In many cases, veterinarians will prescribe weekly dipping in an insecticide to eliminate the mites. In addition, treating the pet's environment is necessary to kill mites in bedding, carpets, and other areas. Insecticidal treatment of kennels and other multi-pet communities is required to halt mite infestations.

Owners of pets infested with these mites may want to check with their physicians regarding medication and other steps to control mite infestations in themselves, their family members, and the home environment. It is very easy these mites to spread from pets to owners.

Cheyletiella blakei mites are the most common cause of walking dandruff in cats. (The dandruff that is seen "walking" is actually the mites moving about on the skin of the cat.) *Cheyletiella* mites are very contagious, especially in catteries or multi-pet households. Regular use of certain insecticides to control flea infestations has a side benefit of often controlling the mites that cause walking dandruff. Humans are frequently infested with this mite. Mites that cause walking dandruff have 4 pairs of legs and large hook-like mouthparts. They live on the skin's surface, and they spend their entire 3-week life cycle on their host.

Scaling of the skin and infestation along the back are common signs of walking dandruff. Intense itching is frequent among infested cats, though there may be no itching at all. Cats may develop skin crusts and many small bumps along their back. Some cats may show no signs of infestation but carry the mites and transmit them to other pets and humans.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Fleas	**5.9.2.**	Merck Veterinary Manual	High	High	Low	High	High

There are >2,200 species of fleas recognized worldwide. In North America, only a few species commonly infest dogs and cats: *Ctenocephalides felis* (the cat flea), *Ctenocephalides canis* (the dog flea), *Pulex simulans* (a flea of small mammals), and *Echidnophaga gallinacea* (the poultry sticktight flea). However, by far the most prevalent flea on dogs and cats is *C felis*. Cat fleas cause severe irritation in animals and people and are responsible for flea allergy dermatitis. They also serve as the vector of typhus-like rickettsiae and *Bartonella* spp. and are the intermediate host for filarid and cestode parasites. Cat fleas have been found to infest >50 different mammalian and avian hosts throughout the world. In North America, the most commonly infested hosts are domestic and wild canids, domestic and wild felids, raccoons, opossums, ferrets, and domestic rabbits.

Larvae are susceptible to desiccation, with prolonged exposures to relative humidity <50% being lethal. The areas within a home with the necessary humidity are limited, and suitable outdoor sites are even rarer. Flea development occurs outdoors only where the ground is shaded and moist (1%–20% soil moisture content) and where the flea-infested animal spends a significant amount of time so that adult flea feces will be deposited into the larval environment. In the indoor environment, flea larvae probably survive only in the protected microenvironment deep within carpet fibers, in cracks between hardwood floors in humid climates, and on unfinished concrete floors in damp basements. The larval stage usually lasts 5–11 days but may be prolonged for 2–3 wks., depending on availability of food and climatic conditions.

The pre-emerged adult (which is a fully formed adult flea) residing in the cocoon is the stage that can extend the longevity of the flea. If the pre-emerged adult does not receive the proper stimulus to emerge, it can remain quiescent in the cocoon for several weeks until a suitable host arrives. Emergence can be delayed up to 350 days if pre-emerged adults are protected from desiccation. Newly emerging fleas move to the top of the carpet pile or vegetation, where they are more likely to encounter a passing host. Under ideal conditions of temperature (27°C [80.6°F]) and relative humidity (90%), a newly emerged cat flea can survive ~12 days before requiring a blood meal; at 50% relative humidity, this interval drops to ~3 days. It is these newly emerged unfed fleas that infest animals and bite people. There is generally minimal inter-host movement of cat fleas. However, it has been documented that before *C felis* reaches reproductive status, there can be some limited movement on and off hosts. Cat fleas that have found a preferred host (e.g. dog, cat, opossum, etc.) and have initiated reproduction generally do not leave their host unless forced off by grooming or insecticides.

Depending on temperature and humidity, the entire life cycle of the cat flea can be completed in as little as 12–14 days or can be prolonged for up to 350 days. However, under typical household conditions with normal pet and human activity, cat fleas complete their life cycle in 3–8 wk.

Cat fleas are susceptible to cold. No stage of the life cycle (egg, larva, pupa, or adult) can survive exposure to <3°C (37.4°F) for several days. Therefore, cat fleas survive winters in north temperate climates as adults on untreated dogs and cats or on small wild mammals (e.g., raccoons or opossums) in the urban environment. As these animals pass through yards in the spring or set up nesting sites in crawl spaces or attics, the eggs laid by surviving female fleas drop off and subsequently develop to adults. Cat fleas may also survive the winter as pre-emerged adults in microenvironments protected from the cold.

Fleas can cause iron-deficiency anemia in heavily infested hosts, particularly in young animals. Fleas in the genus *Ctenocephalides* have been reported to produce anemia in poultry, dogs, cats, goats, calves, and sheep.

Cat fleas are also involved in disease transmission. Murine typhus, caused by *Rickettsia typhi* and *Rickettsia felis*, is a mild to severe febrile disease of people characterized by headaches, chills, and skin rashes, with infrequent involvement of the kidneys and CNS. The disease is seen in people and many small mammals along the southeastern, southwestern, and Gulf coasts. In the USA, the principal transmission cycle involves opossums and cat fleas. Cat fleas also serve as the intermediate host of the nonpathogenic subcutaneous filarid nematode of dogs, *Dipetalonema reconditum*. *Dipylidium caninum*, the common intestinal cestode of dogs and cats (and rarely children), develops as a cysticercoid in *C felis*, *C canis*, and *Trichodectes canis*. Flea larvae ingest the eggs of the tapeworm, which develop into cysticercoids in the body of the flea. When grooming themselves, dogs and cats may ingest infected fleas, and the cysticercoids are released.

A flea-infested dog or cat can easily introduce fleas into a home where they deposit eggs that then develop into newly emerging fleas. These then infest other pets and bite people.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Lice	**5.9.3.**	Merck Veterinary Manual Eydal, 1992 parasitipedia.net	Indigenous*	Present*	Low	Very low	n.a.*

*Dog lice (*Linognathus setosus*) were found in 1989 in Iceland (Eydal, 1992).

Numerous species of lice parasitize domestic animals. Lice are largely host specific, living on one species or several closely related species. Lice are obligate ectoparasites and depend on the host to complete their life cycle. Usually not a serious threat neither for pets, nor for humans. Recent taxonomic changes have complicated the orders and suborders of lice. In general, lice are divided into two categories: bloodsucking (or sucking) lice (order *Anoplura*) and chewing (or biting) lice (formerly the order *Mallophaga*, now composed of three suborders). Bloodsucking lice are parasites of mammals, whereas chewing lice infest both mammals and birds. Lice live within the microenvironment provided by the skin and its hair or feathers, and are transmitted primarily by contact between hosts. All life stages occur on the host, although lice may survive off the host for a period of time. In temperate regions, lice are most abundant during the colder months and often are difficult to find in the summer. Infestations are most often seen on stressed animals, and husbandry and individual health are important in treatment and management of these parasites.

Lice are small, flightless insects that live in the hair or feathers of animals and people. Most lice are of the biting or chewing type (order *Mallophaga*), including the cat louse (*Felicola subrostrata*). Lice are most often seen on older, longhaired cats that are no longer able to groom themselves.

Lice live within the environment provided by the skin and hair. They move from host to host by direct contact. In temperate regions, lice are most common during the colder months and hard to find in the summer. Most *Mallophaga* lice have definite preferences as to their hosts: they will often live on only one species or several closely related species.

Lice have claws on their legs that are adapted for clinging to hair. Females glue their eggs, known as nits, to the hairs of the host near the skin. The nits are tightly attached and ordinary shampooing will not dislodge them. It takes about 3 to 4 weeks for most lice to go from nit to adult.

The first signs that your cat may have lice are scratching, biting, and rubbing of infested areas. If the lice are abundant, the hair might also be matted. Usually, diagnosis is made by seeing lice on the infested cat. Parting the hair often reveals the lice. Lice are active and can be seen moving through the hair.

Female lice glue their eggs, called nits, to the hairs of the host near the skin. Ordinary shampooing and washing will not dislodge the nits. Nits are pale, translucent, and almost oval in shape. Once the nits hatch, the lice undergo a nymphic stage before reaching adulthood. The immature nymphs look very much like adult lice, only smaller. It takes about 3 to 4 weeks for most lice to go from nit to reproductively capable adult, although this period varies with the species.

Dogs can be infested with one species of bloodsucking lice, *Linognathus setosus*, and two species of chewing lice, *Trichodectes canis* and *Heterodoxus spiniger*. *H spiniger* is considered rare in North America.

It is distributed worldwide but appears to be more common in warmer environments; infestations are heavier on animals in poor physical condition. *H spiniger* exhibits atypical behavior for a chewing louse—it is a blood-feeder. Dogs neglected or in poor health may become heavily infested with *L setosus*, which tends to prefer longhaired breeds. *T canis* prefers the head, neck, and tail of the host, and it may be found around wounds and body openings. Infestations may be heavy on very young and very old animals. Infested dogs rub, bite, and scratch the affected area and have a rough, matted coat.

Cats can be infested with one species of chewing lice, *Felicola subrostratus*, although there are rare reports of *H spiniger* on feral cats in other regions of the world. The louse may be seen more frequently on older, longhaired cats that are unable to groom themselves.

With widespread use of monthly flea and tick preventives, pediculosis in dogs and cats has become rare in the USA. Infestation is usually seen on debilitated, feral, stray, or shelter animals.

Dogs can be infested with 2 species of lice, *Linognathus setosus* (the dog sucking louse) and *Heterodoxus spiniger* (the biting louse). Dogs in poor health can become heavily infested. The biting louse is rare in North America. It can serve as an intermediate host for intestinal tapeworms.

The first signs that a dog may have lice include scratching, biting, and rubbing of infested areas. A dog with lice will have a rough, dry coat. If the lice are abundant, the hair might also be matted. Sucking lice cause small wounds that can become infected. Usually, the diagnosis is made by seeing lice on the infested pet. Parting the hair often reveals the lice. Chewing lice are active and can be seen moving through the hair. Sucking lice usually move more slowly. They are often found with their mouth-parts embedded in the skin.

Using a fine-toothed comb to dislodge nits is a tedious process that will not kill lice that have hatched. Dogs, cats, and other pets are usually treated with dips, washes, sprays, or dusts that kill lice.

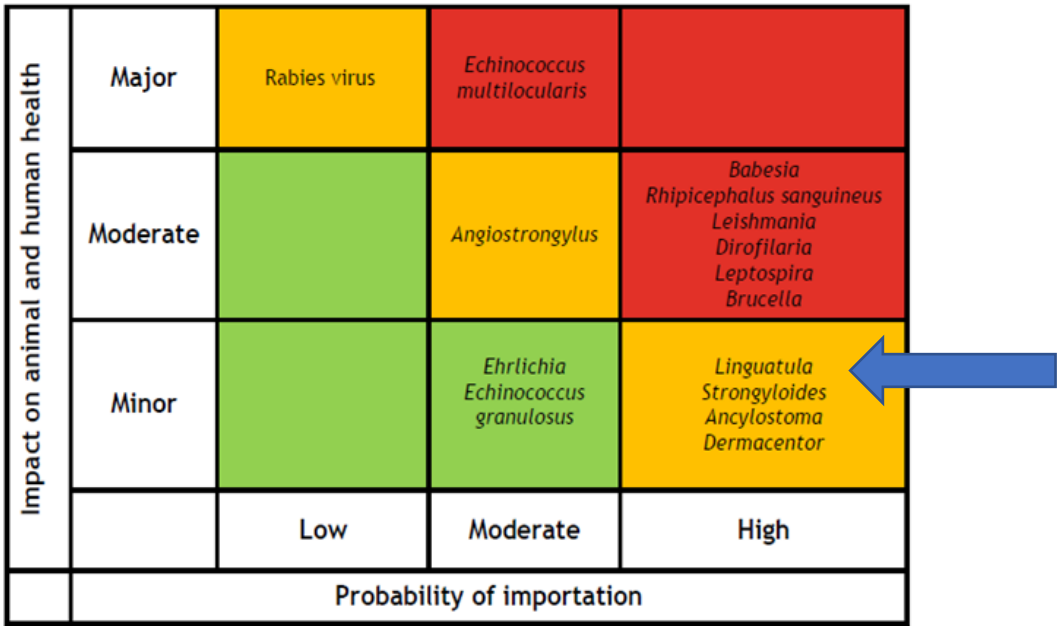
Lice dropped or pulled from the host die in a few days, but eggs may continue to hatch over 2 to 3 weeks. Thus, lice control treatments should be repeated 7 to 10 days after the first treatment. Careful inspection of your pet's coat should be continued daily for at least 2 weeks after you see the last louse. Be sure to carefully collect any lice (dead or alive) removed from your pet and dispose of them promptly in a sealed container (such as a zip-closure plastic bag).

In severe louse infestations, the dog may damage its skin by scratching. Bacterial infections and scratch wounds are common. If these conditions are present, your veterinarian may prescribe an antibiotic or other medication.

In addition to killing the lice on your pet, you will want to be sure that lice are not infesting your dog's bedding, collar, grooming tools (including bushes or combs), and other similar objects in your dog's environment. Careful cleaning and inspection of these objects can help provide your pet with continued relief from the irritation caused by lice.

The lice that infest dogs, cats, and other pets are not normally attracted to humans. Therefore, while care in dealing with the lice infecting your pet is recommended, owners should understand that people rarely get lice from their pets.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Linguatula serrata	**5.9.4.**	parasitipedia.net Høgåsen et al. 2012	High	Moderate	Low	Low	Low



From Høgåsen et al. 2012

Linguatulosis is a condition associated with the organism *Linguatula serrata*. More generally, linguatulosis can be considered a form of "pentastomiasis", which refers to all diseases caused by pentastomids, including *porocephaliasis*.

Usually a minor problem. The disease is often accidentally identified during autopsy because of its asymptomatic effect on the body.

Geographic range

L.serrata can be found worldwide but especially in warm subtropical and temperate regions, rather occasional.

Epidemiology

Infection of Cairo dogs varies from male to female, with a ratio of 1.9:1 (58.97% and 30.77%, respectively. Prevalence of infection rate is higher during spring and summer (66.67%), compared during fall and winter, which constitutes 33.33% of the infection rate. The probable source of canine infections is infected

lymph nodes of cattle, sheep, goats, and/or camels, which produce the symptoms of halzoun and the marrara syndrome in man when consumed raw .

Life cycle

Adult *L. serrata* embed their forebody into the nasopharyngeal mucosa, feeding on blood and fluids. Females live at least two years and produce millions of eggs. Eggs exit the host in nasal secretion or, if swallowed, with feces. When swallowed by an intermediate host, the four-legged larvae (resembling a mite) hatch in the small intestine, penetrate the intestinal wall, and lodge in tissues, particularly in lungs, liver, and lymph nodes. The nymphal stage develops. When eaten by a definitive host, infective nymphs either attach in the upper digestive tract or quickly travel there from the stomach, reaching the *nasopharynx*. Females begin egg production in about six months.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Scabies	**5.9.5.**	Merck Veterinary Manual Parasitipedia.net	High	High	Moderate	Low	Moderate

Canine Scabies (Sarcoptic Mange)

This form of mange is caused by the mite *Sarcoptes scabiei canis*. This highly contagious parasite is found on dogs worldwide. It is often called canine scabies. Although the mites that cause mange are fairly picky in selecting their host, humans and other animals that come in contact with an infected dog may also become infected. The entire life cycle (17 to 21 days) of these mites is spent on the infested dog. Females burrow tunnels in the skin to lay eggs. Mange is easily spread between animals by contact. Indirect transmission, such as through infested bedding, is less common, but it can occur. The incubation period varies from 10 days to 8 weeks, depending on how severely the dog is infested, part of the body affected, number of mites transmitted, and the individual dog's health and hygiene.

Not all dogs have signs when they are infested with sarcoptic mange mites. Usually, though, the animal will have intense itching that comes on suddenly. The itching is probably caused by sensitivity to the mites' droppings. Initially, infested skin will erupt with small, solid bumps. Because the dog scratches or bites itself to relieve the itch, these bumps and the surrounding skin are often damaged, causing thick, crusted sores. Secondary yeast or bacterial infections can develop in the damaged skin. Usually, the sores appear first on the abdomen, chest, ears, elbows, and legs. If the mange is not diagnosed and treated, the sores can spread over the entire body. Dogs with long-term, recurring mange develop oily dandruff (seborrhea), severe thickening of the skin with wrinkling and crust build-up, and oozing, weeping sores. Dogs affected this severely can become emaciated and may even die.

"Scabies incognito" is a term used to describe hard-to-diagnose mange. If a dog is regularly bathed and has a well-groomed coat, the mites might be hard to find, even if the dog shows signs of infestation such as itching. The other typical signs of mange—crusts and scales on the skin—are removed by regular bathing.

If mange is suspected, do a physical examination, including collecting skin scrapings and possibly a stool sample. Some clinics might also use a blood test to diagnose mange. If mites are not found, but the signs are highly suggestive of mange, trial treatment is warranted. Mange is very highly contagious and can spread easily between animals of different species and even to humans. Thus, you should ask your veterinarian for advice on how to avoid contracting mange from your pet.

Treatment should include all dogs and other animals that have been in contact with one another. It may be necessary to clip the hair. The crusts and dirt should be removed by soaking with a medicated (anti-seborrheic) shampoo, and an anti-mite dip applied. Lime-sulfur is highly effective and safe for use in young animals. This is frequently prescribed by veterinarians. Several dips may be required. Internal medicines may also be prescribed. Some internal mange medications are also used for heartworm prevention, so

your veterinarian may want to test your dog for heartworms before treatment. Treatment for secondary infections may also be necessary.

Disease/ Infection	IS #	References	Entry	Establ.	Impact		Risk
					Dog/Cat	Man	
Otodectes cynotis	**5.9.6.**	Merck Veterinary Manual Parasitipedia.net Skirnisson et al. 2018	Indigenou***s****	Present*	Moderate	Low	n.a.*

** Otodectes cynotis* has been reported to be indigenous in Iceland among the artic fox, cats and dogs (Skirnisson et al., 2018).

Otodectes cynotis, the ear mite, affects mainly cats and occasionally dogs. Kittens are more at risk than adult cats. It also affects wild animals such as foxes, coyotes, ferrets, etc. The ear mite is a frequent and major cause of external otitis in cats, i.e. an inflammation of the external ear. Cat ear mites can occasionally infest humans as well.

Otodectes mites are 0.25 to 0.5 mm long. Females are larger than males. The life cycle lasts about 3 weeks. The mites live in the external ear. Sometimes they may affect other body parts, e.g. the tail. They do neither dig into the skin, nor suck blood, but feed on skin debris, exudates, which are produced as an allergic skin reaction to the mite saliva. Infections with secondary bacteria and appearance of pus are frequent, which complicate the disease.

Excess exudates and earwax build thick dark crusts that cover the external ear channel, which becomes eczematous. A dark secretion like coffee dregs accumulates in the ears. If examined under the microscope the living mites can be seen. Severe infestations can also lead to local bleeding. A possible complication is eardrum perforation with subsequent deafness and the risk that the infestation reaches the inner ear, the meninges (the brain membranes) and even the brain itself.

Affected animals suffer from intense itching and react shaking the head, licking the affected parts, vigorously scratching the ears, rubbing against objects (trees, furniture, etc.), sometimes up to self-mutilation.

Diagnosis can be made with an otoscope, which allows visualizing the mites.

Transmission among pets is by contact, typically from nursing queens to their kittens. Ear mites are highly contagious and a very brief contact is often enough for transmission.

SUMMARY TABLE 1: Dogs

	Dogs only		Risk Assessment Summary				
					Impact		
	Disease/infection	Number	Entry	Establishment	Dog/cat	Man	Risk
1	African Horse Sickness (AHS)	5.1.1.	very low	very low	very low	negligible	very low
2	Aujeszsky's disease (AD)	5.1.2.	negligible	negligible	high	negligible	negligible
4	Canine distemper (CD)	5.1.4.	high	high	high	negligible	high
9	Canine herpesvirus	5.1.9.	high	high	high	negligible	high
11	Kennel Cough (Infectious Tracheobronchitis)	5.1.11.	high	high	moderate	negligible	moderate
12	Hepatitis contagiosa canis (HCC)	5.1.12.	high	high	high	negligible	high
13	Rabies	5.1.13.	low	high	high	high	low
15	Canine parvovirus	5.1.15.	high	high	high	negligible	high
16	Foot-and-Mouth Disease (FMD)	5.1.16.	very low	very low	negligible	negligible	negligible
17	Canine leptospirosis	5.2.1.	high	moderate	moderate/high	moderate	moderate/high
18	Melioidosis	5.2.2.	very low	low	moderate	moderate	very low
19	Murine typhus	5.2.3.	low	very low	moderate	moderate	very low
20	Plague (Yersinia pestis)	5.2.4.	low	very low	moderate	moderate	very low
21	Rocky Mountain spotted fever	5.2.5.	high	very low	low/moderate	moderate/high	very low
22	Salmonellosis	5.2.6.	high	low	moderate	moderate	low
23	Tularemia	5.2.7.	low	low	moderate	high	low
24	Tuberculosis	5.2.8.	low	low	moderate	high	low
25	Anthrax	5.2.9.	very low	very low	high	high	very low
26	Borreliosis	5.2.10.	high	moderate	moderate	high	moderate
27	Boutoneneuse fever (Rickettsia)	5.2.11.	high	very low	low	high	very low
28	Campylobacter	5.2.12.	moderate	moderate	moderate	moderate	moderate
29	Canine brucellosis	5.2.13.	high	moderate	moderate/high	moderate/high	moderate
30	Canine ehrlichiosis	5.2.14.	moderate	low	moderate	low/moderate	low
31	Glanders	5.2.15.	very low	low	high	high	very low
32	Ringworm	5.3.1.	moderate	moderate	high	moderate	moderate
33	Babesiosis	5.4.1.	high	very low	moderate	low/moderate	very low

34	Hepatozoonosis	5.4.2.	low/moderate	very low/low	low/moderate	very low	very low/low
35	Leishmaniosis	5.4.3.	variable	very low	high	high	very low
36	Chaga's disease (Trypanosoma cruzi)	5.4.4.	low	negligible	moderate	moderate	negligible
37	Giardia	5.4.5.	indigenous	present	low/moderate	low/moderate	n.a.
38	Neosporosis	5.4.6.	low	moderate	low/moderate	negligible	low
39	Trypanosoma (Surra)	5.4.7.	very low/negl.	negligible	very low	negligible	negligible
41	Paragonimus westermani	5.6.1.	very low	very low	moderate	moderate	very low
42	Schistosoma japonicum	5.6.2.	very low	uncertain	moderate	high	uncertain
43	Echinococcus multilocularis	5.7.1.	moderate	moderate	negligible	high	moderate
44	Echinococcus granulosus	5.7.2.	moderate	moderate	negligible	high	moderate
45	Ancylostoma caninum	5.8.1.	high	moderate	moderate	low/moderate	moderate
46	Angiostrongylus vasorum	5.8.2.	moderate	moderate	mod./high	negligible	moderate
47	Dirofilaria immitis	5.8.3.	high	moderate	moderate/high	moderate	moderate
48	Strongyloides stercoralis	5.8.4.	high	low/moderate	low/moderate	low/moderate	low/moderate
49	Cheyletiellosis	5.9.1.	indigenous	present	moderate	high	n.a.
50	Fleas	5.9.2.	high	high	low	high	high
51	Lice	5.9.3.	indigenous	present	low	very low	n.a.
52	Linguatula serrata	5.9.4.	high	moderate	low	low	low
53	Scabies	5.9.5.	high	high	moderate	low	moderate
54	Otodectes cynotis	5.9.6.	indigenous	present	moderate	low	n.a.

Summary Table 2: Cats

	Cats only		Risk Assessment Summary				
					Impact		
	Disease/infection	Number	Entry	Establishment	Dog/cat	Man	Risk
3	Feline calicivirus (Feline Resp. Dis. Complex)	5.1.3.	high	high	moderate	negligible	moderate
5	Feline poxvirus (cowpox)	5.1.5.	moderate	moderate	low	low	low
6	Feline leukemia virus (FLV)	5.1.6.	sero-reactors	high	high	negligible	n.a.?
7	Feline infectious peritonitis (FIP)	5.1.7.	high	moderate	moderate	negligible	moderate
8	Feline immunodeficiency virus (FIV)	5.1.8.	sero-reactors	high	moderate	negligible	n.a.?
10	Feline parvovirus (panleukopenia)	5.1.10.	high	high	high	negligible	high
14	Feline rhinotracheitis (Cat flu)	5.1.14.	high	high	moderate	negligible	moderate
40	Feline spongiform encephalopathy (FSE)	5.5.1.	negligible	negligible	high	negligible	negligible

Summary Table 3: Dogs and cats

	Dogs and cats			Risk Assessment Summary				
						Impact		
	Disease/infection	Number	Cats	Entry	Establishment	Dog/cat	Man	Risk
1	African Horse Sickness (AHS)	5.1.1.		very low	very low	very low	negligible	very low
2	Aujeszsky's disease (AD)	5.1.2.		negligible	negligible	high	negligible	Negligible
3	Feline calicivirus (Feline Resp. Dis. Complex)	5.1.3.	Y	high	high	moderate	negligible	Moderate
4	Canine distemper (CD)	5.1.4.		high	high	high	negligible	High
5	Feline poxvirus (cowpox)	5.1.5.	Y	moderate	moderate	low	low	Low
6	Feline leukemia virus (FLV)	5.1.6.	Y	sero-reactors	high	high	negligible	n.a.?
7	Feline infectious peritonitis (FIP)	5.1.7.	Y	high	moderate	moderate	negligible	Moderate
8	Feline immunodeficiency virus (FIV)	5.1.8.	Y	sero-reactors	high	moderate	negligible	n.a.?
9	Canine herpesvirus	5.1.9.		high	high	high	negligible	High
10	Feline parvovirus (panleukopenia)	5.1.10.	Y	high	high	high	negligible	High
11	Kennel Cough (Infectious Tracheobronchitis)	5.1.11.		high	high	moderate	negligible	Moderate
12	Hepatitis contagiosa canis (HCC)	5.1.12.		high	high	high	negligible	High
13	Rabies	5.1.13.		low	high	high	high	Low
14	Feline rhinotracheitis (Cat flu)	5.1.14.	Y	high	high	moderate	negligible	Moderate
15	Canine parvovirus	5.1.15.		high	high	high	negligible	High
16	Foot-and-Mouth Disease (FMD)	5.1.16.		very low	very low	negligible	negligible	Negligible
17	Canine leptospirosis	5.2.1.		high	moderate	moderate/high	moderate	moderate/high
18	Melioidosis	5.2.2.		very low	low	moderate	moderate	very low
19	Murine typhus	5.2.3.		low	very low	moderate	moderate	very low
20	Plague (Yersinia pestis)	5.2.4.		low	very low	moderate	moderate	very low
21	Rocky Mountain spotted fever	5.2.5.		high	very low	low/moderate	moderate/high	very low
22	Salmonellosis	5.2.6.		high	low	moderate	moderate	low
23	Tularemia	5.2.7.		low	low	moderate	high	low
24	Tuberculosis	5.2.8.		low	low	moderate	high	low
25	Anthrax	5.2.9.		very low	very low	high	high	very low
26	Borreliosis	5.2.10.		high	moderate	moderate	high	moderate
27	Boutoneneuse fever (Rickettsia)	5.2.11.		high	very low	low	high	very low

28	Campylobacter	5.2.12.		moderate	moderate	moderate	moderate	moderate
29	Canine brucellosis	5.2.13.		high	moderate	moderate/high	moderate/high	moderate
30	Canine ehrlichiosis	5.2.14.		moderate	low	moderate	low/moderate	low
31	Glanders	5.2.15.		very low	low	high	high	very low
32	Ringworm	5.3.1.		moderate	moderate	high	moderate	moderate
33	Babesiosis	5.4.1.		high	very low	moderate	low/moderate	very low
34	Hepatozoonosis	5.4.2.		low/moderate	very low/low	low/moderate	very low	very low/low
35	Leishmaniosis	5.4.3.		variable	very low	high	high	very low
36	Chaga's disease (Trypanosoma cruzi)	5.4.4.		low	negligible	moderate	moderate	negligible
37	Giardia	5.4.5.		indigenous	present	low/moderate	low/moderate	n.a.
38	Neosporosis	5.4.6.		low	moderate	low/moderate	negligible	low
39	Trypanosoma (Surra)	5.4.7.		very low/negl.	negligible	very low	negligible	negligible
40	Feline spongiform encephalopathy (FSE)	5.5.1.	Y	negligible	negligible	high	negligible	negligible
41	Paragonimus westermani	5.6.1.		very low	very low	moderate	moderate	very low
42	Schistosoma japonicum	5.6.2.		very low	uncertain	moderate	high	uncertain
43	Echinococcus multilocularis	5.7.1.		moderate	moderate	negligible	high	moderate
44	Echinococcus granulosus	5.7.2.		moderate	moderate	negligible	high	moderate
45	Ancylostoma caninum	5.8.1.		high	moderate	moderate	low/moderate	moderate
46	Angiostrongylus vasorum	5.8.2.		moderate	moderate	mod./high	negligible	moderate
47	Dirofilaria immitis	5.8.3.		high	moderate	moderate/high	moderate	moderate
48	Strongyloides stercoralis	5.8.4.		high	low/moderate	low/moderate	low/moderate	low/moderate
49	Cheyletiellosis	5.9.1.		indigenous	present	moderate	high	n.a.
50	Fleas	5.9.2.		high	high	low	high	high
51	Lice	5.9.3.		indigenous	present	low	very low	n.a.
52	Linguatula serrata	5.9.4.		high	moderate	low	low	low
53	Scabies	5.9.5.		high	high	moderate	low	moderate
54	Otodectes cynotis	5.9.6.		indigenous	present	moderate	low	n.a.

Summary Table 4: Guide dogs							
	Modified as per risk-reduction assumption for Guide dogs	Risk Assessment Summary					
					Impact		
	Disease/infection	Number	Entry	Establishment	Dog/cat	Man	Risk
1	African Horse Sickness (AHS)	5.1.1.	negligible	negligible	very low	negligible	negligible
2	Aujeszsky's disease (AD)	5.1.2.	negligible	negligible	high	negligible	negligible
4	Canine distemper (CD)	5.1.4.	moderate	moderate	high	negligible	moderate
9	Canine herpesvirus	5.1.9.	moderate	moderate	high	negligible	moderate
11	Kennel Cough (Infectious Tracheobronchitis)	5.1.11.	moderate	moderate	moderate	negligible	moderate
12	Hepatitis contagiosa canis (HCC)	5.1.12.	moderate	moderate	high	negligible	moderate
13	Rabies	5.1.13.	very low	moderate	high	high	very low
15	Canine parvovirus	5.1.15.	moderate	moderate	high	negligible	moderate
16	Foot-and-Mouth Disease (FMD)	5.1.16.	negligible	negligible	negligible	negligible	negligible
17	Canine leptospirosis	5.2.1.	moderate	low	moderate/high	moderate	low
18	Melioidosis	5.2.2.	negligible	very low	moderate	moderate	negligible
19	Murine typhus	5.2.3.	very low	negligible	moderate	moderate	negligible
20	Plague (Yersinia pestis)	5.2.4.	very low	negligible	moderate	moderate	negligible
21	Rocky Mountain spotted fever	5.2.5.	moderate	negligible	low/moderate	moderate/high	negligible
22	Salmonellosis	5.2.6.	moderate	very low	moderate	moderate	very low
23	Tularemia	5.2.7.	very low	very low	moderate	high	very low
24	Tuberculosis	5.2.8.	very low	very low	moderate	high	very low
25	Anthrax	5.2.9.	negligible	negligible	high	high	negligible
26	Borreliosis	5.2.10.	moderate	low	moderate	high	low
27	Boutoneneuse fever (Rickettsia)	5.2.11.	moderate	negligible	low	high	negligible
28	Campylobacter	5.2.12.	low	low	moderate	moderate	low
29	Canine brucellosis	5.2.13.	moderate	low	moderate/high	moderate/high	low
30	Canine ehrlichiosis	5.2.14.	low	very low	moderate	low/moderate	very low
31	Glanders	5.2.15.	negligible	very low	high	high	negligible
32	Ringworm	5.3.1.	low	low	high	moderate	low
33	Babesiosis	5.4.1.	moderate	negligible	moderate	low/moderate	negligible
34	Hepatozoonosis	5.4.2.	very low/low	negl./very low	low/moderate	very low	negl./very low

35	Leishmaniosis	5.4.3.	variable	negligible	high	high	negligible
36	Chaga's disease (Trypanosoma cruzi)	5.4.4.	very low	negligible	moderate	moderate	negligible
37	Giardia	5.4.5.	indigenuos	present	low/moderate	low/moderate	n.a.
38	Neosporosis	5.4.6.	very low	low	low/moderate	negligible	very low
39	Trypanosoma (Surra)	5.4.7.	negligible	negligible	very low	negligible	negligible
41	Paragonimus westermani	5.6.1.	negligible	negligible	moderate	moderate	negligible
42	Schistosoma japonicum	5.6.2.	negligible	uncertain	moderate	high	uncertain
43	Echinococcus multilocularis	5.7.1.	low	low	negligible	high	low
44	Echinococcus granulosus	5.7.2.	low	low	negligible	high	low
45	Ancylostoma caninum	5.8.1.	moderate	low	moderate	low/moderate	low
46	Angiostrongylus vasorum	5.8.2.	low	low	mod./high	negligible	low
47	Dirofilaria immitis	5.8.3.	moderate	low	moderate/high	moderate	low
48	Strongyloides stercoralis	5.8.4.	moderate	very low/low	low/moderate	low/moderate	very low/low
49	Cheyletiellosis	5.9.1.	indigenuos	present	moderate	high	n.a.
50	Fleas	5.9.2.	moderate	moderate	low	high	moderate
51	Lice	5.9.3.	indigenous	present	low	very low	n.a.
52	Linguatula serrata	5.9.4.	moderate	low	low	low	low
53	Scabies	5.9.5.	moderate	moderate	moderate	low	low
54	Otodectes cynotis	5.9.6.	indigenous	present	moderate	low	n.a.

Summary Table 5: Comparisons of Risk assessments for All dogs vs. Guide dogs

Dog categories	Risk categories										
	High	High/ Mode -rate	Mode -rate	Mode -rate/ Low	Low	Low/ Very low	Very low	Very low/ Neglig- ible	Neglig- ible	Accept- able risk	Total
All dogs											
Diseases per risk category	5	1	11	1	7	1	11	0	4	15	41
% of all diseases	12%	2%	27%	2%	17%	2%	27%	0%	10%	37%	100%
Cumulative number of diseases	5	6	17	18	25	26	37	37	41		
Cumulative % of all diseases	12%	15%	41%	44%	61%	63%	90%	90%	100%		
Guide dogs											
Diseases per risk category	0	0	6	0	12	1	6	1	15	22	41
% of all diseases	0%	0%	15%	0%	29%	2%	15%	2%	37%	54%	100%
Cumulative number of diseases	0	0	6	6	18	19	25	26	41		
Cumulative % of all diseases	0%	0%	15%	15%	44%	46%	61%	63%	100%		

References

Alfredsson, M. et al. (2017) Surveillance of Ixodes ricinus ticks (Acari: Ixodidae) in Iceland. Parasites & Vectors 10: 466.

AHAC8: Animal Health Pack 8 – Infectious diseases of dogs. Land Based Colleges National Consortium http://www.lbcnc.org.uk.

Anon. (1976). :Guide Dogs "Less Hygiene Risk." Health Education Journal, 35(3), 207–207. https://doi.org/10.1177/001789697603500303

Australian Government Department of Agriculture, Fisheries and Forestry (AUS 2012) Importation of dogs and cats and their semen from approved countries. Draft policy review, July 2012. Available at http://www.daff.gov.au/ba.

Australian Government Department of Agriculture (2013) Importation of dogs and cats and their semen from approved countries. Final policy review, November 2013. Available at http://www.daff.gov.au/ba.

Bartels, C.J.M, et al. (2007). Quantification of vertical and horizontal transmission of *Neosporum caninum* infection in Dutch dairy herds. Veterinary Parasitology, 148, 83 – 92.

Biosecurity Australia (2001). Guidelines for Import Risk Analysis (Draft). 119 pp.

Bjarnadottir, L. (2007) A seroepidemiological survey for antibodies to Borrelia burgdorferi sensu lato in dogs in Iceland. Hovedopgave, Fagdyrlægeuddannelsen vedr. hund og kat, 21 pp.

Carabin, H. et al. (2015) Schistosoma japonicum in Samar, the Philippines: infection in dogs and rats as a possible risk factor for human infection. Epidemiol. Infect. 143, 1767 – 1776.

CFSPH (Center for Food Security & Public Health, Iowa State University) (2016) Feline Spongiform Encephalopathy. 7 pp. Available at: *http://www.cfsph.iastate.edu/Factsheets/pdfs/feline_spongiform_encephalopathy.pdf*

Corrin, K. & s. MacDiarmid (1997) The risks of introducing rabies through the importation of dogs. Surveillance 24(1), 22 – 25.

Department of Agriculture, Fisheries and Forestry, Australian Government (2004). Generic Import Risk Analysis (IRA) for Pig Meat - Final Import Risk Analysis Report. Available at: *http://www.agriculture.gov.au/SiteCollectionDocuments/ba/memos/2004/animal/2004-01a.pdf*

Deplazes, P. et al. (2011) Role of pet dogs and cats in the transmission of helminthic zoonoses in Europe,, with focus on echincoccosis and toxocarosis. Veterinary Parasitology, 182 41 – 53.

Donahue, S.I. et al.(2015) A review of neosporosis and pathological findings of Neospora caninum infection in wildlife..International Journal fo Parasitology: Parasites and Wildlife, 4, 216 – 238.

Davidson, R.K. et al. (2016) Echinococcus across the north: Current knowledge, furure challenges Food and Waterborne Parasitology 4, 39 – 53.

EFSA (2013). Scientific Opinion on the risk of entry of *Aethina tumida* and *Tropilaelaps spp*. in the EU. EFSA Panel on Animal Health and Welfare (AHAW), *https://efsa.onlinelibrary.wiley.com/doi/abs/10.2903/j.efsa.2013.3128*

EFSA VBD Story maps: African Horse Sickness, Hepatozoon canis, Leishmaniasis.

https://efsa.maps.arcgis.com/apps/MapJournal/index.html?appid=df1fa0739f144112957d2557f9e60a58#

EFSA AHAW Panel (EFSA Panel on Animal Health and Welfare) 2015. Scientific Opinion on canine leishmaniosis. EFSA Journal 2015;13(4):4075, 77 pp. doi:10.2903/j.efsa.2015.4075
Available online*: www.efsa.europa.eu/efsajournal*

EFSA AHAW Panel (EFSA Panel on Animal Health and Welfare) 2017. Characterisation of Hepatozoon canis (hepat) for scientific opinion on vector-borne diseases. doi:10.2903/j.efsa.2017.4810 Available online: http://arcg.is/2hfnUaT

European Commission (EUC) COMMISSION DELEGATED REGULATION (EU) 2018/772 of 21 November 2017 supplementing Regulation (EU) No 576/2013 of the European Parliament and of the Council with regard to preventive health measures for the control of *Echinococcus multilocularis* infection in dogs, and repealing Delegated Regulation (EU) No 1152/2011

Eydal, M. (1992) Hundalus finnst a Islandi. Dyrlæknaritid (Januar 1992) 30 – 31.

Eydal, M., S.H Richter & K. Skirnisson (2001): Poster presentation: Parasites of imported dogs and cats in Iceland 1989-2000. Bulletin of the Scandinavian Society for Parasitology, 11, No. 1-2, p. 31.

Eydal, M. & K. Skirnisson (2016) Strongyloides stercoralis found in imported dogs, household dogs and kennel dogs in Iceland. Icel. Agric. Sci. 29, 39 – 51.

Fjøludottir, E.H. (2013) The prevalence of intestinal helminths and protozoa in dogs in south-west Iceland. Master Thesis in Veterinary Medicine, Faculty of Health and Medical Sciences, University of Copenhagen. 72 pp.

FMD: Information available at: http://www.footandmouthdiseaseinfo.org/default.aspx

Hamel, D., et al. (2011) Canine vector-borne disease in travelled dogs in Germany – A retrospective evaluation of laboratory data from the years 2004 – 2008. Veterinary Parasitology, 181, 31 – 36.

Hamel, D. et al. (2012) Epidemiological aspects on vector-borne infections in stray and pet dogs from Romania and Hungary with focus on Babesia spp. Parasitol. Res. 110, 1537-1545.

Hamel, D., et al. ((2013) Artropod-borne infections in travelled dogs in Europe. Parasite, 20, 9.

Hamnes, I.S. et al. (2013) Parasittologisk og serologisk undersøkelse av prøver fra gatehunder importert til Norge fra land i Øst-Europa. Veterinærinstituttets rapportserie Rapport 15, 20 pp. Avaiable at: https://www.vetinst.no/rapporter-og-publikasjoner/rapporter/2012/importrisikovurdering-av-gatehunder-fra-st-europa

Hansen, A.P. & H. Holm (2001) Prevalens av felint leukemivirus og antistoffer mot felint immunsvigtvirus blant katter på Island. Fordypningsoppgave ved Norges veterinærhøgskole 2001. 20 pp.

Holst, B.S. et al. (2012) The first case of Brucella canis in Sweden: background, case report and recommendations from a northern European perspective. Acta Vet. Scand. 54: 18.

Høgåsen, H.R., Hamnes, I.S., Davidson, R. & Lund, A. (2012): Importrisikovurdering av gatehunder fra Øst-Europa. Veterinærinstituttets rapportserie, 11. Available at: https://www.vetinst.no/rapporter-og-publikasjoner/rapporter/2013/parasittologisk-og-serologisk-underskelse-av-prver-fra-gatehunder-importert-til-norge-fra-land-i-st-europa

Jonsdottir, A. et al. (2002) Ahættugreining vegan innflutnings a hundum og køttum til Islands (Risk analysis regarding the import of dogs and cats to Iceland). Report September 2002, 32 pp.

Lind, P. DTU Veterinærinstituttet (2014) Vurdering af smitterisici ved indførsel af hunde (FVST-sagsnr.: 2014-14-81-00074), 36 pg. Available at: *https://www.foedevarestyrelsen.dk/SiteCollectionDocuments/Dyresundhed/Sygdomsgruppen/Hunde%20smitterisici%20ved%20indf%C3%B8rsel,%20DTU%20Veterin%C3%A6rinstituttets%20rapport.pdf*

Liu, Q. et al. (2008) Paragonimiasis: an important food-borne zoonosis in China. Trends in Parasitology vol 24 No. 7, 318 – 323.

MacDiarmid, S. C. & K. C. Corrin (1998) Case study: The risk of introducing rabies through the importation of dogs [online]. In: Elms, David (Editor). Owning the Future: Integrated Risk Management in Practice. Christchurch, New Zealand: Centre for Advanced Engineering, 1998: 221-226. Availability: https://search.informit.com.au/documentSummary;dn=079872305819366;res=IELENG

MacDiarmid, S.C. & H.J. Pharo (2003) Risk analysis: assessment, management and communication. Rev. Sci. Tech. Off. Int.Epiz. 2003, 22, 397 – 408.

McGarvey, S.T. et al. (2006) Cross-sectional associations between intensity of animal and human infection with Schistosoma japonicum in Western Samar province, Philippines. Bull. WHO, 84, 446 – 452.

Menn, B. et al. (2010) Imported and travelling dogs as carriers of canine vector-borne pathogens in Germany. Parasites & Vectors 3:34, 7 pp.

Merck Veterinary Manual: Available at: https://www.merckvetmanual.com/

New Zealand Government Ministry of Primary Industries (9 March 2018): Import Health Standard, 19 pp. Available at: https://www.mpi.govt.nz/dmsdocument/1574/loggedIn

OIE Handbook on Import Risk Analysis for Animals and Animal Products, vol. 1 & 2, 2004.

OIE Technical Disease Card on AHS (Last updated April 2013). Available at: http://www.oie.int/fileadmin/Home/eng/Animal_Health_in_the_World/docs/pdf/Disease_cards/AFRICAN_HORSE_SICKNESS.pdf

OIE Technical Disease Card on Trypanosoma evansi infections (Surra) Last updated April 2013. Available at: *http://www.oie.int/fileadmin/Home/eng/Animal_Health_in_the_World/docs/pdf/Disease_cards/TRYPANO_EVANSI.pdf*

OIE Terrestrial Animal Health Code (2017) Chapter 8.5. *Echinococcus multilocularis*; Chapter 8.6. *Echinococcus granulosus*; Chapter 8.14.6. Recommendations for importation of dogs, cats and ferrets from countries considered infected with rabies. Available at: http://www.oie.int/index.php?id=169&L=0&htmfile=chapitre_rabies.htm

OIE Terrestrial Manual (2014) Chapter 2.1.11. Leishmaniosis, 12 pp. Available at: *http://www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.01.11_LEISHMANIOSIS.pdf*

Parasitipedia.net Available at: *http://parasitipedia.net/index.php?*

Policy and Risk, MAF Biosecurity New Zealand (2 November 2009): Import risk analysis: Cats, dogs and canine semen - FINAL, 262 pp. Available at: https://www.mpi.govt.nz/dmsdocument/6058/loggedIn

Ready, P.D. (2010) Leishmaniasis emergence in Europe. Euro Surveillance 15(10). Available at: *http://www.eurosurveillance.org/ViewArticle.aspx?Articleid=19505*

Reperant, L.A., et al. (2016) Companion animals as a source of viruses for human beings and food producing animals. J. Comp. Path.155, S41 – S53.

Rijks, J.M. et al (2016) Disease risk assessments involving companion animals: an overview for 15 selected pathogens taking a European perspective. J. Comp. Path.155, S75 – S97.

Sigurdarson, S. (2010) Dogs and echinococcosis in Iceland. Acta Vet. Scand. 52(Suppl 1): 56.

Skirnisson, K., J.H. Olafsson & H. Finnsdottir (1997) Dermatitis in cats and humans caused by Cheyletiella mites reported in Iceland. Læknabladdid 83, 30 – 34.

Skirnisson, K., G.R. Palsdottir, & M. Eydal (2018) Parasites of dogs and cats imported to Iceland during 1989 – 2017 with remarks on parasites occurring in the native populations. Icel. Agric. Sci. 31, 49 – 63.

Thordarson, T.H. (2018) Personal communication: Heartworm in a dog in quarantine in Iceland.

Yamamoto, M., M.T. Lopez, & L.A. Hart (2015) Registrations of assistence dogs in California for identification tags: 199 – 2012. PLOS One, August 19, 2015, 20 pp.

Wieland B, Dhollander S, Salman M, Koenen F (2011). Qualitative risk assessment in a data-scarce environment: A model to assess the impact of control measures on spread of African Swine Fever. Prev. Vet. Med. 99, 4 – 14.

Wikipedia: https://en.wikipedia.org/wiki/Canine_distemper

Willesen, J.L. et al (2014) Occurrence of *Angiostrongylus vasorum i*nfections in dogs with a relevant clinical picture in Denmark. 4th European Dirofilaria and Angiostrongylus Days, 2014, 67.