Import risk analysis: Babesia gibsoni in dogs (*Canis familiaris*) and dog semen.

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Ministry of Agriculture and Forestry
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Approved for release

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1. EXECUTIVE SUMMARY

This document is a qualitative analysis of the biosecurity risks posed by *Babesia gibsoni* in dogs (*Canis familiaris)* and their semen imported into New Zealand.

B. gibsoni is a tick-transmitted blood borne parasite of wild and domestic dogs. The tick *Haemaphysalis longicornis,* which is known to be capable of transmitting the disease, is found in New Zealand. The disease is characterised by lethargy, fever and haemolytic anaemia. Treatment is not effective in eliminating the parasite, and recovered dogs commonly become chronic carriers, thereby posing a source of infection for other dogs and ticks.

There is no evidence that semen poses a risk of introduction of *B. gibsoni.*

Safeguards are recommended to manage the risk of introduction of *B. gibsoni* in imported dogs

2. INTRODUCTION

2.1 BACKGROUND

Babesia gibsoni is considered to be a potential hazard in the cat and dog import risk analysis currently being undertaken by MAF.

B. gibsoni is present in a number of countries, and has recently been reported in Australia (Hood, 2002). During 2001 a total of 2,530 dogs were imported into New Zealand, mostly (1,730) from Australia.

B. gibsoni is the subject of this separate risk analysis so that safeguards may be put in place as soon as possible.

2.2 RISK ANALYSIS METHODOLOGY

The steps in the risk analysis process are (OIE, 2002):

- hazard identification
- risk assessment -release assessment
  -exposure assessment
  -consequence assessment
  -risk estimation
- risk management
- risk communication

2.2.1 Hazard identification

In the hazard identification the epidemiology of *B. gibsoni* is examined to determine if the parasite could be introduced into New Zealand in imported dogs. The hazard identification concludes that *B. gibsoni* is a potential hazard.

2.2.2 Risk assessment

The risk assessment comprises four steps:

i) **Release assessment**

This step describes the biological pathways by which *B. gibsoni* could be introduced by imported dogs. Two pathways for introduction are discussed : dogs harbouring the organism and/or dogs harbouring infected ticks.

ii) **Exposure assessment**

This step describes the possible pathways by which susceptible hosts in New Zealand could be exposed to *B. gibsoni*.

iii) **Consequence assessment**

The human and animal health, environmental, cultural, social and economic consequences of introduction of *B. gibsoni* are discussed.

iv) **Risk estimation**

The release, exposure and consequence assessments are integrated to enable a conclusion to be drawn as to the risk imported dogs and dog semen pose with respect to
B. gibsoni.

2.2.3 Risk management

In this step risk mitigation measures (safeguards) are formulated to give an appropriate level of protection against the introduction of *B. gibsoni* by imported dogs and their semen.

3. HAZARD IDENTIFICATION

3.1 AETIOLOGIC AGENT

Family Babesiidae, Genus *Babesia* and Species *gibsoni.*

Individual *Babesia* species have historically been characterised by the size and morphological appearance of the intra-erythrocytic forms. All small canine *Babesia* were identified as *Babesia gibsoni.*

Genetic sequencing technology using the polymerase chain reaction (PCR) technique has allowed further subdivision into three strains *Babesia gibsoni* (Asia), *Babesia gibsoni* (California) and a recently identified small *Babesia* endemic in north-west Spain and is also reported in Germany (Zahler et al, 2000; Kjemtrup et al, 2000).

3.2 New Zealand's status

Babesia gibsoni has never been reported in New Zealand and is notifiable.

In view of the suggested link with American Pit Bull Terriers, which have been introduced to New Zealand from the United States, a survey of 155 adult dogs in New Zealand was carried out. This including 32 American Pit Bull Terriers, 47 Staffordshire Bull Terriers and 27 Bull Terrier cross dogs did not reveal any evidence of *B. gibsoni* infection, either from serology (indirect fluorescent antibody testing (IFAT) using the *B. gibsoni* (Asian) antigen) or examination of blood smears for parasites (Beban, 2003).

3.3 Epidemiology

B. gibsoni is a tick-transmitted intracellular haemo-protozoan parasite of wild and domestic canids (Yamane, Conrad & Gardner, 1993; Yamane et al, 1993).

Although it has been recognised for some time in Asia, it is now also recognised as being widely distributed in Asia, Africa, Europe, Middle East, Brazil, North America. In addition, it has recently been reported from Australia.

B. gibsoni (Asia) is endemic in Sri-Lanka, Japan, Malaysia, Korea and Egypt and, more recently, it has been recognised as present in the mid-west and south-eastern United States of America and Australia (Yamane et al, 1994; Birkenheuer et al, 1999; Macintire et al, 2002; Kocan et al, 2001; Hood, 2002). Its prevalence in Peninsular Malaysia and Taiwan has been reported to be 17% (Rajamanickam et al, 1985; Chang & Tu, 1992). In Japan, 40% of veterinarians surveyed reported diagnosing the disease in the previous year (Onishi et al, 1994). Many of the positive dogs from Japan, Taiwan and mid-west and south-eastern United states have been fighting breeds such as American Pit Bull Terriers or American Staffordshire Terriers.

B gibsoni (California) is endemic in California and is closely related to isolates from wildlife and humans from the western United States. There appears to be no increased prevalence in fighting breeds for this strain (Conrad et al, 1991; Yamane, Conrad & Gardner, 1993; Jerant & Arline, 1993; Persing et al, 1995).

Although *Babesia canis volgeli* has been known to be present in Australia for some time, *B. gibsoni* (Asia) has only recently been reported from that country, in three American Pit Bull Terriers belonging to the same owner at a single premise in Victoria (Hood, 2002). No direct link with imported dogs was found, suggesting that the agent is endemic.

3.3.1 Methods of transmission

3.3.1.1 Tick transmission

The principal vector for *B. gibsoni* (Asia) in endemic areas of India and Sri Lanka is the tick *Haemaphysalis bispinosa* and both stage-to-stage and transovarial transmission have been found in this species (Otsuka, 1974). Some early studies referred to *H. longicornis* as *H. bispinosa,* until methods to differentiate between them structurally and biologically were identified (Hoogstraal et al, 1968).

Rhipicephalus sanguineus is reported to be a vector for B. gibsoni (Asia) in India (Otsuka 1974). Stage-to stage (larvae to nymphs) transmission was found and development of B. gibsoni in the salivary glands of R. sanguineus has been observed (Higuchi et al, 1995).

In Australia, both *H. longicornis* and *R. sanguineus* are believed to be vectors (Leggoe, 1998). While *R. sanguineus* is widespread in Australia, *H. longicornis* has been reported in Queensland, New South Wales, South Australia and Western Australia (Leggoe, 1998).

The natural tick vector for *B. gibsoni* (Asia) in the USA is not known. It is thought to be vectored by ixodid ticks that feed on dogs such as *R. sanguineus* and *Dermacentor variabilis* (Conrad et al, 1991) but definitive transmission studies have not been done (Macintire, 2002). . *H. bispinosa* and *H. longicornis* are not present in the USA.

H. longicornis has been shown to be capable of transmitting *B. gibsoni* (Asia) (Otsuka, 1974) and is thought to be the vector in Japan.

H. longicornis is endemic in New Zealand. It occurs mainly in the North Island but is now also found in areas in the South Island. The tick is found in greater numbers on hosts at certain times of the year depending on the district. In Northland numbers of larvae on the host are greatest in February, nymphs in August and adult females in December (Heath, 1985).

H. longicornis is a three host tick; each stage (larval, nymph and adult) must attach to separate hosts during the course of development. The hosts may be the same or different species. The main hosts for *H. longicornis* in New Zealand are sheep, cattle and deer although they will attach to virtually all mammals including dogs, as well as some birds (Tenquist & Charleston, 1981; Heath et al, 1988).

The major source of infection for dogs are carrier ticks (the infective merozoites are in the tick's saliva). Ticks generally need to feed on hosts for 2 to 3 days before transmission of *Babesia* spp. can occur (Kraje, 2001). Each of the feeding periods lasts for about 4 to 7 days. The detachment periods may last weeks or months, with the effect that ticks spend about 80% of their total lives off hosts. Moreover, ticks may survive about a year without a blood meal (Heath, 1985). Most recovered dogs develop a state of premunition that is a balance between the hosts immune response and the parasite's ability to induce clinical disease (Birkenheuer et al, 1999). In this state of premunition, dogs are at risk of recrudescence and are a potential reservoir for tick infection.

3.3.1.2 Direct transmission

Direct blood transmission may occur through blood transfusions containing *B. gibsoni*, through the sharing of instruments for such procedures as ear cropping and tail docking, and through the reuse of vaccination needles (Macintire et al, 2002).

Direct transmission may also occur by dog bites (Macintire et al, 2002). This has been suggested by the reportedly high incidence of the disease in American Pit Bull Terriers (Birkenheuer et al, 1999; Chang & Tu, 1992; Onishi et al, 1994), a breed commonly used for dog fighting, and by the high prevalence of *B. gibsoni* in American Pit Bull Terrier kennels in the USA (Macintire et al, 2002).

3.3.1.3 Other routes of transmission

Transplacental transmission of *B. gibsoni* is thought to occur, although this has not been confirmed under controlled conditions (Birkenheuer et al, 1999; Macintire, 2002).

None of the references sighted during the course of this risk analysis made any mention of the possibility of semen being a vehicle for transmission of *B. gibsoni*. On this basis, it is concluded that semen poses a negligible risk of introducing this organism.

3.3.2 Pre-patent period and incubation period

The prepatent period (time from infection to the appearance of *B. gibsoni* in the bloodstream) has been reported as 7-11 days for dogs infected by *H. longicornis* (Otsuka, 1974) and 2-40 days in experimental infections (Yamane, Conrad & Gardner, 1993; Meinkoth et al, 2002). The incubation period (i.e. time from infection to the onset of clinical signs) depends on the route of infection and number of parasites in the inoculum and varies from 7 to 21 days (Yamane, Conrad & Gardner, 1993; Lobetti, 1998; Macintire et al, 2002).

3.4 Clinical Signs

There are two forms of clinical disease caused by *B. gibsoni* infection (Kraje, 2001). The acute form is characterised by fever, lethargy, haemolytic anaemia and marked thrombocytopenia (Meinkoth et al, 2002). Animals that recover develop a chronic

carrier state, in which parasitaemia has been shown to persist for at least 38 months (Groves & Dennis, 1972; Groves & Yap, 1968; Farwell, LeGrand & Cobb, 1982). The chronic form presents as intermittent fever, lethargy and weight loss and may persist for years (Groves & Dennis, 1972).

The clinical signs of *B gibsoni* infection are variable. In some cases the disease is fulminative with multiple organ failure and death, and such cases are related not only to the degree of parasitaemia but also to the host's immune response (Kraje, 2001). However recent reports of *B. gibsoni (*Asia) in the south-eastern United States infections document cases of mild and, in some cases, even inapparent disease (Macintire et al, 2002; Meinkoth et al, 2002).

Infection with *B. gibsoni* (California) appears to be a more severe form than infection with *B. gibsoni* (Asia) in the United States (Meinkoth et al, 2002, Conrad et al, 1991). 6 of 15 cases of naturally infected *B. gibsoni* dogs in California died or were euthanased (a case fatality rate of 40%) (Yamane et al., 1994).

The Spanish strain appears to cause severe regenerative anaemia and evidence of renal failure in some cases (Camacho et al., 2001).

3.5 Treatment

No drugs have proven to be effective for the elimination of *B. gibsoni* from infected dogs (Birkenheuer et al, 1999). Some antibabesial drugs can reduce the severity of clinical signs and the mortality associated with the disease.

No vaccines for *B. gibsoni* are available.

3.6 Zoonotic potential

B. gibsoni's zoonotic potential is uncertain. Although two other *Babesia* species, *B. microti* (a rodent parasite which is associated with human babesiosis in the USA) and *B. divergens* (a ruminant parasite associated with human babesiosis in Europe) have been definitely identified as human pathogens (Persing et al, 1995), only two reports of serious illness in humans from the *B. gibsoni* (California) strain have been documented, both in splenectomised patients (Jerant & Arline, 1993). Nevertheless, serologic testing of people who were considered to have had possible exposure to ticks indicated a seroprevalence rate of 16% (8 of 51 persons) (Persing et al, 1995).

3.7 Hazard identification conclusion

B. gibsoni is a tick borne disease and one tick vector, *H. longicornis,* is found in New Zealand. Dogs that are incubating infection, or are in the acute or chronic phase of the disease can potentially transmit *B. gibsoni.* Of particular importance are dogs that are in the premunition phase (are clinically normal but harbour the organisms and may develop parasitaemia).

It is concluded that *B. gibsoni* is a potential hazard in imported dogs, but that dog semen is not.

4. RISK ASSESSMENT

4.1 Release Assessment

There are reports that, in both the USA and in Asia, the Asian strain of *B. gibsoni* is more prevalent in fighting breeds of dogs (Birkenheuer et al., 1999, Chang and Tu, 1992 and Onishi et al., 1994), but the parasite can affect dogs of all breeds and all ages (Rajamanickam et al, 1985). Wild canine species destined for zoos could also be infected.

As shown in Table 1, the vast majority of dogs imported into New Zealand originate from Australia. Since *B. gibsoni* has recently been identified in Australia, this trans-Tasman trade in live dogs is considered to now pose the greatest risk to New Zealand. A significant number of dogs are also imported from the USA and Canada. Although few dogs are imported from continental Europe, the ease of movement of dogs within the European Community with the Pet Travel Scheme means that dogs from the United Kingdom and Ireland are also potential hazards (Perkins, 2000).

There are two possible pathways for imported dogs to introduce *B. gibsoni:*

- dogs could carry the organism at the time of importation
- dogs could harbour infected ticks

In the year to December 2001, approximately 2500 dogs were imported into New Zealand. Table 1 shows that of those, only 5% come from countries which do not have *B. gibsoni*. About 70% of imported dogs come from Australia, 17% from the UK, and 6% from the USA.

Table 1: Dogs Imported into NZ for the 2001 calendar year

From countries where *B. gibsoni* is present	**Number of dogs**
Australia	1730
United Kingdom	426
USA	155
Canada	29
Continental Europe	26
Ireland	10
Japan	10
Korea -South	8
Malaysia	7
Taiwan	3
From countries where *B. gibsoni* is not present	126
TOTAL	2530

4.1.1 Carriers

Dogs may be chronic or incubatory carriers of *B. gibsoni.* Dogs that recover from clinical disease usually become chronic carriers, exhibiting bouts of illness and parasitaemia interspersed with periods of clinical normality and low or negligible parasitaemia. Incubatory carriers arise because infected dogs may remain clinically normal for 7-21 days after infection.

4.1.2 Dogs harbouring infected ticks

No acaricide will kill 100% of ticks. The recommended acaricide (Fiproil *Frontline,* Merial) is considered to be about 90% effective after 48 hours (McPherson, Merial, personal communication, 2002).

There have been 73 tick interceptions in New Zealand between 1980 and 2000 (Heath, 2001), the majority of which were on dogs. *R. sanguineus* was identified in 34 of the total interceptions.

From April 2000 to April 2002 there have been a further 8 interceptions (Matthew Stone, personal communication 2002), of which 6 were from Australia.

The recent introduction of tick inspections for all imported dogs and cats is expected to decrease the probability of exotic ticks being released into New Zealand on dogs and cats.

4.1.3 Release assessment conclusion

Many dogs are imported from countries where *B gibsoni* is present, and ticks have been found on imported dogs. Therefore the release assessment concludes that the likelihood of *B. gibsoni* being introduced with imported dogs is non-negligible.

4.2 Exposure Assessment

4.2.1 Spread by vectors

The most likely route of spread of *B. gibsoni* in New Zealand would be infected dogs being exposed to the endemic tick, *H. longicornis.* Since this tick has a limited distribution in New Zealand, it would be possible for an infected dog to be introduced into a part of the country free from ticks, in which case tick-borne transmission would not occur. However, *B. gibsoni* may persist in infected dogs for many years, so later movement within the country could spread infection.

Infected ticks introduced on dogs could spread *B. gibsoni* by biting other dogs. Further, many mammals and birds act as hosts for *H. longicornis* and as ticks survive for long periods, these animals could disperse infected ticks widely so that dogs could become infected with *B. gibsoni* far from the point of initial introduction.

If exotic ticks became established they would constitute a long term risk. *H. bispinosa* may establish in areas of New Zealand that are hot and wet, such as the Bay of Plenty

and Northland. *R. sanguineus* could establish in New Zealand inside houses as it requires warm, dry conditions (Heath et al, 1980).

4.2.2 Other routes

Other possible routes of spread are :

- iatrogenic (via blood transfusions using an infected carrier as a donor or by needle sharing),
- via dog bites while fighting
- transplacental transmission

A scenario tree for the exposure assessment of *B. gibsoni* from imported parasitaemic dogs can be seen in figure 1. The major pathway for spread is shown in bold.

4.2.3 Exposure assessment conclusion

There is a high likelihood of *B gibsoni* being exposed to susceptible species in New Zealand.

4.3 Consequence Assessment

4.3.1 Direct and indirect consequences

The consequences of *B. gibsoni* infection may be severe in dogs. Dogs that survive the acute illness are likely to become carriers and have periodic episodes of ill health. In addition, treatment is expensive and not always effective. Thus, *B. gibsoni* infection would have direct consequences (both emotional and financial) for dog owners.

If *B. gibsoni* became established in New Zealand, eradication would be unlikely to be successful as the tick vector is widespread. Preventive measures such as regular tick treatment and prevention of dogs entering tick infested areas, would have financial consequences for dog owners and prevent them freely enjoying their dogs and their environment. Export of dogs to some countries would become difficult.

New Zealand does not have any indigenous canine species. Thus the effect on native animal populations would be negligible. There is a potential for serious disease in splenectomised or immune-compromised humans.

Figure 1. Scenario tree for the exposure assessment outlining biological pathways necessary for susceptible dogs to become infected with *Babesia gibsoni* in New Zealand following importation of a parasitaemic dog

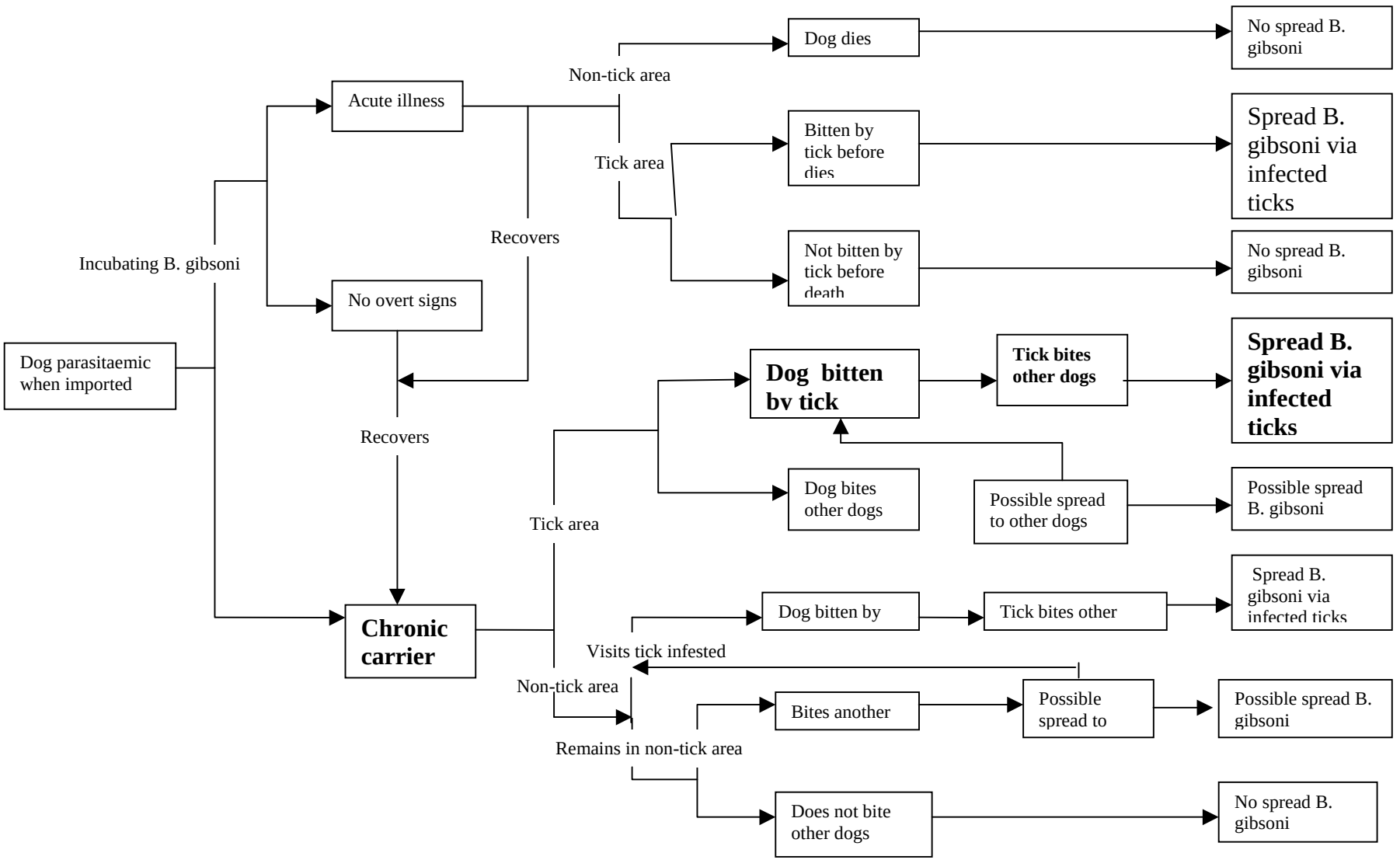

Table 2 Summary of the consequence assessment for *B.gibsoni*

Consequence		Significance	Likelihood
Biological	Dogs	Severe	High
	Other animals	Negligible	Negligible
	Humans	Possibly severe	Low
Environmental	(including cultural & social conditions)	Moderate to severe	High
Economic	(effect on national economy)	Low	High

4.3.2 Consequence assessment conclusion

The direct consequences of infection with *B. gibsoni* in New Zealand would be severe, it is likely to become widespread and eradication would be impossible. Control costs would be high for pet owners. Frequent tick treatment would be necessary to prevent exposure to disease. The establishment of *B. gibsoni* could have an adverse effect on dog exports.

Consequences in some humans cannot be ruled out, but as there are no native canine species the consequences for native fauna would be negligible.

4.4 Risk Estimation

Dogs are imported into New Zealand from a number of countries in which *B gibsoni* is endemic. Since infection with *B. gibsoni* is characterised by a prolonged sub-clinical carrier state, the likelihood of dogs imported from such countries being infected is non-negligible.

As the New Zealand cattle tick is a potential vector for *B. gibsoni*, it is likely that the organism could spread and become established in this country.

The likely consequences of *B. gibsoni* infection in dogs would be severe.

Therefore it is concluded that the risk estimate for *B. gibsoni* is non-negligible.

5. RISK MANAGEMENT

5.1 Risk Evaluation

Since the risk estimate for *B. gibsoni* is high, the imposition of sanitary measures is justified.

5.2 Option Evaluation

5.2.1 Objective

The objective of the recommended sanitary measures is to minimise[1] the likelihood that dogs will be carrying *B. gibsoni* when given a biosecurity clearance in New Zealand.

5.2.2 Options available

5.2.2.1 Country freedom

Proving country declaration of freedom from *B. gibsoni* would be difficult. At the very least this would require the demonstration of adequate surveillance systems in the country from which the dog was imported. However, in view of the prolonged carrier state in recovered animals, for a dog to qualify as coming from a free country, it would also be necessary to show that all countries in which the dog has resided were free.

5.2.2.2 Clinical signs and clinical pathology

The clinical signs of *B.gibsoni* infection are variable and non-specific. Anaemia and general ill health can be seen in many other diseases. Differentiation from auto-immune haemolytic anaemia is difficult as many *B. gibsoni* infections are Coomb's positive.

5.2.2.3 Tick control

Although tick control is valuable in reducing the risk of infection, acaricides that are appropriate for use on dogs are only partially effective.

Fipronil (*Frontline, Merial)* is currently the most appropriate acaricide for use in the import context. Fipronil has a label claim as an "aid in tick control", and the manufacturers claim greater than 90% efficacy in tick control after 48 hours (McPherson, Merial, personal communication, 2002). One trial found that
90% of *Ixodes holocyclus* ticks were killed by fiprinol spray, and that this reduction lasted for 3 weeks (Atwell et al, 1996). The reported kill rate at 48 hours was 96%, and at 72 hours was 100%. However, tick species vary in their response to acaricides and each of four dogs from Australia on which live ticks have been found in this country had been treated with fipronil prior to export (Heath, 2002, personal communication).

The manufacturer's recommendation for tick control with fipronil is monthly treatment (McPherson, Merial, personal communication, 2002), but at the height of the paralysis tick season

[1] Given the overarching MAF objective of safe and free trade, "minimise" in this context means to reduce the likelihood of introduction to the lowest level possible using the technologies currently available.

in Australia, animals are treated every two weeks (Wilson, Merial, personal communication, 2002). However, fiprinol treatment is not intended to prevent ticks attaching but it is claimed that they will usually be killed within 24-48 hours.

Tick control in the pre-export period may be achieved by treatment with fipronil at 14 day intervals, under veterinary supervision, at a dose according to the label recommendation. To minimise the likelihood of ticks being on dogs at the time of importation, the final treatment prior to shipment would have to be no earlier than 14 days and no later than 72 hours prior to export.

5.2.2.3 Diagnostic tests

The diagnostic tests that are of potential value as screening tests for *B. gibsoni* are:

- detection of the parasite on thin blood smears
- serological testing using IFAT
- detection of genetic material by the PCR test

None of these methods is completely sensitive.

a) Blood smears

The value of blood smears are limited by the fact that in the early stages of the disease and in the carrier state there may be very low or negligible parasitaemia. The parasites are not detectable on blood smear for up to 40 days post infection (Yamane, Conrad and Gardner, 1993) and detectable parasitaemia is transient; it is only apparent for 21 to 28 days although dogs remain carriers (Meinkoth et al, 2002). Groves and Dennis (1972) found that during relapses in carrier dogs, there were seldom more than 1% of red blood cells infected. Even in cases of severe anaemia, only 1-2% of erythrocytes are likely to have organisms (Conrad et al, 1991).

Using PCR as the ‘gold standard’ the sensitivity of microscopy is about 90% when highly trained operators are used (Birkenheuer, personal communication, 2002). Even then, blood smears must be very carefully examined, for up to one hour. Sensitivity of blood smear examination is maximised by making the blood smear from a drop of blood taken from an area with readily accessible capillary beds, such as the ear margin (Perkins, 2000).

b) Serology

The indirect fluorescent antibody test (IFAT) is considered to be relatively effective in diagnosing chronic disease, as IFAT titres remain high for prolonged periods even though the level of detectable parasitaemia is low or negligible (Anderson et al, 1980).

However, the IFAT gives low or negligible titres when dogs are in the early stages of the disease (Yamane et al, 1993). Combining IFAT with blood smear examination will increase sensitivity but there is a period of up to 40 days post-infection when parasitaemia may not be apparent and IFAT is likely to give false negatives.

Moreover, the sensitivity and specificity of the IFAT have not been precisely determined. While sensitivity is reported to be 'high' (Anderson et al, 1980; Yamane et al, 1993), there may be considerable serologic cross-reactivity among *Babesia* species (Anderson et al, 1980) and to other protozoal parasites (Yamane et al, 1993). To increase test accuracy, the antigen used in the IFAT

should be that of the *B. gibsoni* strain(s) in the countries in which the dog has resided (Meinkoth et al, 2002).

For the purpose of managing the risk in imported animals, the requirement is to maximise test sensitivity. A cut off titre of 1:40 (Farwell et al, 1982 , Yamane et al, 1993) has been recommended for this purpose.

c) PCR

PCR is more expensive and the protocol is more difficult than the IFAT. It can be used to confirm a positive result from the IFAT and to detail the genotype, however the IFAT is generally recommended for import screening.

5.2.2.4 Quarantine and testing

Yamane, Conrad and Gardner (1993) have suggested that quarantine of imported dogs is necessary to prevent the spread of *B. gibsoni* to countries free of the organism. Quarantine would have to minimise the likelihood of exposure to ticks, for example by use of concrete runs. The quarantine period must be at least 40 days (which is the maximum prepatent period), and subsequent testing by blood smear examination and IFAT to maximise the sensitivity of such a programme.

5.2.2.5 Using intensive tick control in place of quarantine

Notwithstanding the imperfections of acaricide treatment, high frequency treatment (fipronil every two weeks) for at least 40 days prior to testing, together with measures to minimise exposure to ticks, might be considered as alternative to quarantine. However, there are difficulties associated with certification of minimised tick exposure outside quarantine.

5.2.2.6 General measures

In the case of pre-export quarantine or intensive tick control, in order to minimise the opportunity for re-infection dogs should be exported as soon as possible after testing. Dogs would have to be kept in an environment to prevent or reduce tick exposure until the time of export.

5.2.3 Recommended sanitary measures

In order to achieve the stated risk management objective, imported dogs must either:

i) have resided in countries which can demonstrate freedom from *B. gibsoni*

or

ii) a) undergo a period of pre-export quarantine in the country from which they are being exported. The dog would be treated with appropriate acaricides upon entry into quarantine and again 14 days later, and the quarantine facility must be able to

prevent any new tick infestations occurring during the period of quarantine, which will be long enough to allow testing at the end of the maximum prepatent period for *B. gibsoni.* After 40 days in quarantine, the dog will be tested by thin blood smear[1] and IFAT[2] (samples to be taken on the same day), and will remain in quarantine until negative results to both tests are received, at which time it will be eligible for export direct from quarantine. A positive result to either test will disqualify the dog for export to New Zealand.

and

b) be inspected for ticks on arrival in New Zealand[3]. If ticks are found, the dog will be subjected to a period of post-arrival quarantine. Following 40 days in quarantine, the dog will be tested by blood smear and IFAT (samples to be taken on the same day), and will remain in quarantine until negative results are received from the overseas laboratory doing the testing. In the case of a positive result from either test, the dog will be re-shipped or destroyed.

[1] Examination of thin blood smear taken from a drop of blood from an ear margin, carried out by a laboratory approved by MAF Animal Biosecurity.
[2] In all cases, the IFAT must be carried out using antigens appropriate for the strains of *B. gibsoni* likely to be present in the country where the dog has been resident. The cutoff for the IFAT will be 1:40.
[3] The focus of this risk analysis is *B. gibsoni*, not exotic ticks, so the measures recommended are to manage the risk of *B. gibsoni*. The important issue of exotic ticks is outside the scope of this risk analysis, and is being considered separately by MAF.

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