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Haemotrophic *Mycoplasma* spp

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Yellow discolouration

Sheep and lamb carcasses displaying yellow discolouration at slaughter are likely to be condemned simply because they are aesthetically objectionable. Not much effort is expended on making an aetiological diagnosis. The yellow discolouration can be the result of a genetic beta-carotene metabolism defect, or the accumulation of abundant bile pigments (jaundice or icterus). Causes of bilirubin build-up can be before, within, or beyond the liver (pre-, hepatic, and post-hepatic). Hepatic jaundice is mostly the result of toxic liver damage, with pyrrolizidine alkaloids probably the best-known culprit. Post-hepatic jaundice can be attributed to reduced excretion, as can happen with bile duct occlusion in facial eczema. In pre-hepatic jaundice, overproduction of bilirubin overwhelms the liver's metabolic and excretory capacity. The excess bilirubin is invariably caused by accelerated breakdown of erythrocytes, or haemolysis. Animals suffering from chronic copper poisoning and subject to a stressful event can suffer such a haemolytic crisis. Or erythrocytes can be destroyed, e.g., by *Babesia*, *Anaplasma*, or haemotrophic *Mycoplasma* infection. In the latter case, haemolysis occurs extra-vascularly via erythrophagocytosis, making haemoglobinuria a rare occurrence. (Schröder & Lloyd 2026).

From *Eperythrozoon* and *Haemobartonella* to *Mycoplasma*

The first *Eperythrozoon* was seen in thin blood smears of mice in 1928. The organisms were closely associated with, but not intracellular to, red blood cells. They didn't look nor behaved like bacteria and were thought to be protozoa (Schilling 1928). When similar organisms were found in blood smears of sheep and cattle in 1934, they were named *E ovis* and *E wenyoni*, respectively (Neitz et al. 1934; Adler & Ellenbogen 1934). Taxonomically, Eperythrozoon was first grouped in the family Bartonellaceae (Peters & Wigand 1955) and later classified as a rickettsia (Kreier & Ristic 1968, cited by Baker *et al*. 1971) with *Anaplasma* and *Haemobartonella*.

Electron microscopy raised the suspicion that *E ovis* and *E wenyoni* were related to *Mycoplasma* spp (Avakian *et al*. 1973). Molecular biology enabled genomic and phylogenetic analysis, which revealed that four wall-less bacteria (*Haemobartonella felis*, *Haemobartonella muris*, *Eperythrozoon suis* and *E wenyonii* [sic]) were not rickettsiae, but mycoplasmas (Neimark & Kocan 1997; Rikihisa *et al*. 1997; Neimark *et al*. 2001, 2002). They began to be collectively referred to as haemotrophic mycoplasmas or haemoplasmas, due to their epierythrocytic nature (Neimark *et al*. 2002, 2005). *M ovis* should not be confused with *Anaplasma ovis*, which is an obligate intracellular bacterium (Dumler *et al*. 2001). *M ovis* infections in sheep can occur concurrently with *A ovis* (Hornok *et al*. 2009) and other haemoplasmas (Tagawa *et al*. 2012).

Significance

The clinical significance of haemoplasma infection stems from the haemolytic anaemia, the severity of which is related to the bacteraemia (Sykes et al. 2010). Infections in sheep might be associated with the condemnation of jaundiced sheep carcasses at slaughter. In an investigation of abattoir data from New South Wales in 1984, 65% of jaundiced carcases were infected with *Eperythrozoon* [*Mycoplasma*] *ovis* , as well as 12% of non-jaundiced carcases from the same consignment/lot/line (Archer & Littlejohns 1984).

The apparently common occurrence of long-term and subclinical infections makes quantification of their impact in livestock production difficult (Neitz et al. 1934, Harbutt 1969, Girotto et al. 2012, McFadden et al. 2016). Ill-thrifty lambs, with or without prolonged severe anaemia, in the Eastern Cape province of South Africa were found to be consistently infected with *E [M] ovis* (Burroughs 1988). This subclinical 'erosive disease' ('eroding' profitability without apparent clinical effect) is often discovered only once other potential causes of ill thrift have been eliminated. Acute clinical outbreaks characterised by progression to anaemia, icterus, haemoglobinuria and some mortality are rare and often associated with distinct events, such as vaccination and lamb marking (Urie *et al*. 2019, Windsor 2022).

References to the zoonotic potential of haemoplasma infection feature rarely in published literature and only since the turn of the century (six individual case reports from 2008 to 2021 with infection by at least five different genotypes) (Schröder *et al*. 2026). Poor sanitary conditions, frequent animal contact, and exposure to biting arthropods are cited as predisposing factors (Yang *et al.* 2000; Sykes *et al.* 2010; Maggi *et al*. 2013). The first report of human hemoplasmosis in an animal carer in Australia (Alcorn *et al.* 2021) lends weight to the risk of zoonotic infection posed by close association with animals. The prevalence of haemoplasma infection in 'at-risk' human populations surveyed in the USA (Maggi et al. 2013), a coastal community in Brazil (Kmetiuk et al. 2026), farm workers in China (Yuan et al. 2009), and bed-ridden Nigerian patients (Grétillat & Konarzewski 1978) seems to vary widely. PCR surveys of humans with a history of arthropod and/or animal contact (Yuan et al. 2009, Maggi et al. 2013) revealed high prevalences of *M suis* and *M ovis* infection, with at least one other potentially novel species. Frequent exposure to animals is a significant risk factor for human infection (Schröder & Lloyd 2026), and synanthropic rodent populations could pose a significant zoonotic risk in resource-poor urban communities (Retief 2022).

Occurrence and host preference

Haemotrophic *Mycoplasma* spp have been described on every continent except Antarctica. A multitude of genotypes have been isolated from a variety of domestic and wild terrestrial and flying mammalian host species. Despite earlier naming conventions, the haemoplasmas do not seem to be particularly host-specific, with many genotypes having been isolated from more than one host.

Due to their small genome, the Mycoplasmas lack the metabolic pathways for energy production, thus making them fully host-dependent. This does not directly translate into host-specificity (Citti & Blanchard 2013), because these pathways are conserved. Slovakian red foxes infected with more than four haemotrophic

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Mycoplasma spp (Koneval et al. 2017) seemed to settle the argument against host specificity. There are instances where haemoplasmas display host opportunism. Phylogenetic analysis of 33 species of bats and the 29 haemoplasma genotypes isolated from them seemed to support host specificity, but the bats fell into three classes based on their feeding habits (frugivory, carnivory, and sanguivory) (Becker et al. 2020). This apparent host opportunism is not dissimilar from the notion that parasite infestations are shared by a community of host species with shared functional traits (Dallas et al. 2019). In contrast, the *Mycoplasma haemomuris*-like bacterium infecting two gerbil species in the Negev Desert, but not a third sympatric species, was interpreted as a clear indication of host specificity (Kedem et al. 2014).

Transmission

Haemotrophic Mycoplasma is transmitted in infected blood. This transfer can be mechanical, biological, or vertical.

Mechanical transfer has been demonstrated for several arthropods with biting or sucking mouthparts, such as lice, fleas, biting flies, mosquitoes, and ticks. Aggressive behaviour among rodents is cited as the reason for positive buccal swabs (Conrado et al. 2015, Cohen et al. 2018, Millán et al. 2021, Retief 2022), but whether this signifies the presence of the infection in saliva or contamination with the blood of an infected bite victim is unclear. Sheep husbandry procedures such as vaccination, ear-tagging, and surgical castration are considered to be high-risk iatrogenic and mechanical vector transmission possibilities, with clinical outbreaks seen up to six weeks later (Windsor 2022). The evidence is circumstantial, but the higher prevalence in flocks where needles were reused for vaccination makes a strong case for iatrogenic infection (Urie *et al*. 2019).

Attempts to find haemoplasma DNA in questing tick larvae collected by flannel dragging have failed (Kakimori *et al.* 2023), but experimental infestation of mice with the 3-host *Haemaphysalis longicornis* and the presence of haemoplasma DNA in eggs, larvae, nymphs, and adult ticks provided evidence of biological transmission (Shi *et al.* 2026).

Haemoplasma DNA has been detected in foetuses and neonates, suggesting vertical transmission, without proving pathogenic involvement in foetal or neonatal outcomes (Girotto-Soares et al. 2016, Žilić et al. 2025). Early evidence of transplacental transfer of haemoplasma was based upon PCR confirmation of infection with *M wenyonii* in newborn calves (Hornok et al. 2011, Niethammer et al. 2018). A survey of 651 aborted foetuses in Croatia by PCR found *M wenyonii* and *M haemobos* in calf; *M ovis* in lamb; and *M haemobos*, *M ovis* and *M haemominutum* in goat kid foetuses (Žilić et al. 2025). In South Africa, vertical transmission of rodent-associated haemoplasmas was demonstrated in the sampling of foetuses from 30% of positive *Mastomys natalensis* dams (Retief 2022).

Diagnosis

Clinical signs elicited by haemotrophic *Mycoplasma* infections are non-specific, often vague and transient, and not reliable for a definitive diagnosis. Similarly, abnormalities seen at necropsy, e.g., anaemia, icterus, splenomegaly, and liver histopathology, can be attributed to several causes.

For many decades since its first description, diagnosis of haemoplasma infection relied on the observation of the organisms in thin blood smears stained either with Giemsa or acridine orange (the latter examined under UV light and not specific to *Mycoplasma* spp.) (Schilling 1928, Neitz et al. 1934, Adler & Ellenbogen 1934, Littlejohns 1960, Brun-Hansen *et al.* 1997). Care had to be taken with Giemsa because of the risk of mistaking stain deposits on the smear as proof of infection.

Serology introduced a new diagnostic modality, with tests such as Coombs, complement fixation, indirect fluorescent antibody, and antibody ELISA (Sheriff 1967, Daddow 1977, Nicholls & Veale 1986, Kabay *et al.* 1991). Serological tests can be done in batches, which greatly speeds up the process. A positive serological test proves exposure to the antigen, but not necessarily a current infection.

The advent of molecular biology and the polymerase chain reaction (PCR) for amplification of extracted nucleic acid revolutionised not only the taxonomy of the haemoplasmas, but also their accurate diagnosis. In addition to the small subunit 16S rRNA locus used for bacterial identification, other gene regions have been described for finer distinction between the haemotrophic *Mycoplasma* spp. (Schröder *et al.* 2026)

Pathophysiology

Haemoplasma infection seems to shorten the lifespan of erythrocytes by accelerating the rate of erythrophagocytosis, e.g., in the spleen (Neitz 1937), and possibly by scavenging and competing for host nutrients (Guimarães *et al*. 2011). In addition to the icterus, the haemolysis can also result in haemosiderosis, evident in the liver and kidney cortex (Foggie & Nisbet 1964; Sutton 1974; Fitzpatrick *et al.* 1998).

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QUESTION 1

Is jaundice due to the build-up of haemosiderin in tissues?

a. Yes, especially in the liver and kidney cortex
b. No, it is due to an accumulation of unmetabolized carotene
c. No, it is the accumulation of bilirubin in tissue due to bile duct occlusion
d. No, it is due to liver damage caused by toxic plants
e. No, it is due to the accumulation of bilirubin and can have pre-hepatic, hepatic and post-hepatic causes

QUESTION 2

Is *Eperythrozoon* a blood parasite?

a. Yes, it parasitises red blood cells in cattle, sheep, and pigs
b. Yes, it is transmitted by ticks, like Babesia and Anaplasma
c. No, it is a Mycoplasma and can cause arthritis, mastitis, and pneumonia
d. No, it is a bacterium classified as a haemotrophic Mycoplasma
e. Yes, it is transmitted by biting insects, similarly to Trypanosoma spp

QUESTION 3

How are haemotrophic Mycoplasma species transmitted?

a. By close contact between animals in intensive husbandry systems
b. From males to females during the breeding season
c. By migrating birds
d. In infected blood, either mechanically, biologically, or vertically
e. By biting behaviour in bats and rodents

QUESTION 4

Where do haemotrophic Mycoplasma spp occur?

a. Only in marine mammals (polar bears, walruses) in Arctic environments
b. Only in rodents in urban settings
c. Only in deer in the northern hemisphere.
d. On all continents (not yet described in Antarctica) in a variety of mammals, including humans
e. Only in livestock, e.g., cattle, pigs, and sheep

QUESTION 5

How can haemoplasma infection be diagnosed?

a. By the appearance of the colonies grown on McConkey agar
b. By the clinical signs displayed by infected animals – anaemia, jaundice, and weight loss
c. By the post-mortem appearance of anaemia, icterus, and splenomegaly
d. In Giemsa-stained thin blood smears (look out for stain deposit), or PCR, using the correct primers
e. By serological tests, such as the antibody ELISA

QUESTION 6

Can humans contract haemoplasmosis?

a. No, it is a veterinary problem
b. Yes, it is a venereal disease frequently seen in international travellers
c. Yes, from eating meat from infected animals
d. Yes, from close contact with infected animals
e. Yes, especially from needle sticks when vaccinating animals

QUESTION 7

Is haemotrophic Mycoplasma infection an important cause of jaundice in sheep?

a. Yes, infected animals suffer from haemolytic anaemia, which leads to icterus
b. Yes, one in three jaundiced sheep carcasses condemned at slaughter is infected with a haemoplasma
c. The true extent of jaundice due to haemoplasma is unknown and probably variable
d. No – jaundice is mostly the result of liver damage due to plant poisoning
e. No – jaundice mostly results from the haemolytic crisis in chronic copper poisoning

QUESTION 8

How do Anaplasma and haemotrophic Mycoplasma differ from each other?

a. Anaplasma is transmitted by ticks and haemoplasma by close contact
b. Anaplasma is transmitted by ticks and haemoplasmas by arthropods with biting mouth parts
c. Anaplasma is a rickettsial bacterium, intracellular in red blood cells, while haemoplasmas occur on the external surface of the erythrocytes, or free in plasma
d. Anaplasma causes haemoglobinuria, but haemoplasmas do not
e. Splenomegaly is caused by Anaplasma, but not by haemotrophic Mycoplasma

QUESTION 9

Does an outbreak of haemotrophic mycoplasmosis cause an abortion storm in a sheep flock?

a. Yes – infected sheep develop a high fever and pronounced anaemia leading to a high rate of foetal deaths
b. Yes – a few pregnant ewes infected by a haemoplasma will carry the foetus to full term
c. Yes – haemotrophic Mycoplasma infection causes foetal death
d. No – haemoplasma infection has nothing to do with female reproduction
e. No – haemoplasmas have been isolated from foetuses and foetal membranes but not proven to be the cause

QUESTION 10

Does haemotrophic Mycoplasma infection interfere with beta-carotene metabolism?

a. Yes, and that is the cause of the yellow discolouration in carcases
b. Yes, because it inhibits the expression of the beta-carotene metabolism gene
c. No, but it makes animals more susceptible to the beta-carotene metabolism gene defect
d. No – haemoplasmas have no effect on animals' genetic ability to metabolise beta-carotene
e. No, because beta-carotene is metabolised in the liver and haemoplasmas infect the blood

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