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Lung health assessment of common dolphins (*Delphinus delphis*) in Aotearoa, New Zealand

A thesis presented in partial fulfilment of the requirements for the degree of

Master of Veterinary Studies

at Massey University, Palmerston North, New Zealand



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2026

ABSTRACT

Respiratory health is fundamental to individual fitness in cetaceans, with respiratory compromise able to impair survival in air-breathing aquatic marine mammals. Pneumonia and lungworm infection are the most prevalent post-mortem respiratory diagnoses observed globally in cetaceans. However, systematic lung health assessment has rarely been applied in cetaceans. This study provides the first application of a lung health scoring system to common dolphins (*Delphinus delphis*), using an adapted lung health scoring system and the equivalent lung health grade (LHG) that quantified parasitism, pneumonia, and irreparable tissue damage. A total of 122 stranded and bycaught dolphins examined between 1999 and 2025 were assessed using this lung health system, with a wider subset of 141 individuals assessed specifically for the prevalence of lungworm infection. Lung health scores ranged from 0 to 13, with less than half of the examined dolphins (44.3%, n=54) exhibiting normal lungs, and with most pathological change scored as mild (36.1%, n=44). Lung health grade differed significantly among age classes, with perinates showing the highest proportion of moderate to severe lesions, while immature and sexually mature individuals exhibited predominantly mild or no pathology. No associations between LHG and sex, body condition or animal source were found.

More than half of the examined dolphins displayed pneumonia (51.6%), most scored as mild (58.7%; n=37). Notably, 70% of the pneumonia cases (n=44) were attributable to lung parasitism. However, lungworm prevalence in this study was relatively low (20.6%) compared to global populations of cetaceans, with most infections considered mild. Parasite burden was significantly associated with age class, with perinates exhibiting the highest prevalence and severity. Lung nematodes identified anatomically and molecularly included *Halocercus lagenorhynchi and Stenurus ovatus*. The relatively high proportion of lungs with no or only mild changes (80.3%), and the low prevalence of parasites suggest a comparatively low burden of respiratory pathology in this sample population. This study establishes the first baseline lung health data for any cetacean species in Oceania, serving as a reference for future monitoring of marine ecosystem health under a One Health framework.

ACKNOWLEDGEMENTS

I would like to express my sincere gratitude to my supervisor, Prof. Karen Stockin (Cetacean Pathology Unit, Massey University), for welcoming me into her research group and giving me the opportunity to undertake this project. Her guidance, encouragement, and thoughtful advice throughout this work have been invaluable, and I am deeply grateful for the support she has provided. I am also thankful to my co-supervisor, Prof. John Munday (School of Veterinary Science, Massey University), for agreeing to co-supervise me and for the constructive comments and suggestions that greatly strengthened this thesis.

I would also like to thank Dr Luca Schick and Prof. Ursula Siebert (Institute for Terrestrial and Aquatic Wildlife Research, University of Veterinary Medicine Hannover) for generously sharing the methodological framework that helped shape this thesis, Dr Bronwen Presswell (University of Otago) for her assistance with parasite identification, and Dr Cathy Harvey and Dr Emma Gulliver (Awanui Veterinary) for conducting the histopathology readings for all cases in this study. Their support contributed meaningfully to this work.

My thanks extend to the Department of Conservation and iwi partners, without whom the data for this study would not have been possible. I also acknowledge the CERG team for their assistance, support, and camaraderie during this project.

To my New Zealand family, thank you for providing me with a home away from home. You made daily life far from home easier and more enjoyable, and I am truly grateful for the warmth and stability you offered during this time.

Finally, I am deeply thankful to my family for providing every support I needed from the very beginning. Your care, thoughtfulness, and love made this decision less difficult and allowed me to focus fully on my studies, knowing everything at home was taken care of.

And to God, thank you for guiding my steps, for supporting me through every challenge, and for being present when I needed strength the most.

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Chapter 1. Introduction

1.1 Wildlife Health

Wildlife health is increasingly recognised as an important component of biodiversity conservation and ecosystem stability (Acevedo-Whitehouse & Duffus, 2009; Bossart, 2010). The health of wild populations reflects natural disease dynamics, environmental change and anthropogenic pressures, serving as an indicator of ecosystem health (Bossart, 2010; Cortés-Hinojosa & Seguel, 2024). Surveillance of wildlife health, therefore, provides an insight into the health of the environment, while also serving as an early warning system for wider environmental disturbance (Ijsseldijk et al., 2020; Obusan et al., 2021; Stephen, 2016). This perspective is consistent with the One Health framework, which stresses the interrelation of human, animal and environmental health (Bossart, 2010; Garcia-Bustos et al., 2025; Obusan et al., 2021).

Pathological investigations of wildlife have become essential tools for understanding both species-specific vulnerabilities and ecosystem-level processes (Cortés-Hinojosa & Seguel, 2024; Durden et al., 2023; Ijsseldijk et al., 2020; Kuijpers et al., 2022). Postmortem examinations, disease surveillance and long-term monitoring programs can expose patterns of pathogen emergence, environmental contaminants, and stress-related mortality that may otherwise remain undetected (Dierauf & Gulland, 2001; Fenton et al., 2017). These findings do not only provide information for conservation strategies but also on potential risks to human health, particularly in cases where zoonotic pathogens are involved (Bossart, 2010; Neimanis et al., 2022; Obusan et al., 2021). By integrating pathology, epidemiology and ecological monitoring, wildlife health research forms the basis for conservation and management decisions based on the One Health framework (Dierauf & Gulland, 2001; Garcia-Bustos et al., 2025; Obusan et al., 2021).

Marine mammals have emerged as sentinel species for assessing the health of aquatic ecosystems (Acevedo-Whitehouse & Duffus, 2009; Bossart, 2010; Ijsseldijk et al., 2020; Kuijpers et al., 2022). Their long lifespans, elevated position within the food chain, and reliance on coastal and pelagic habitats, make them vulnerable to cumulative environmental

stressors, including pollution, climate change, and infectious disease (Arbelo et al., 2013; Bossart, 2010; Dierauf & Gulland, 2001). As such, they function as “barometers” of ocean health, with their morbidity and mortality patterns able to reflect broader ecological conditions (Bossart, 2010; Cortés-Hinojosa & Seguel, 2024; Durden et al., 2023). An understanding of marine mammal pathology not only supports species conservation but also provides information on the health of the ecosystem and potential implications for human communities (Bossart, 2010; Obusan et al., 2021; van Elk et al., 2014).

Wildlife mortality events represent important opportunities for species and ecosystem health assessment (Kuijpers et al., 2022). The global interest in the study of marine mammals, particularly cetaceans (whales, dolphins and porpoises), as bioindicators has positioned them at the forefront for monitoring ocean ecosystem health (Bossart, 2010; Fenton et al., 2017). The examination of health trends and causes of mortality is critical to environmental monitoring and population management (Durden et al., 2023; Ijsseldijk et al., 2020; Tomo & Kemper, 2022).

1.2 Cetacean Lung Health

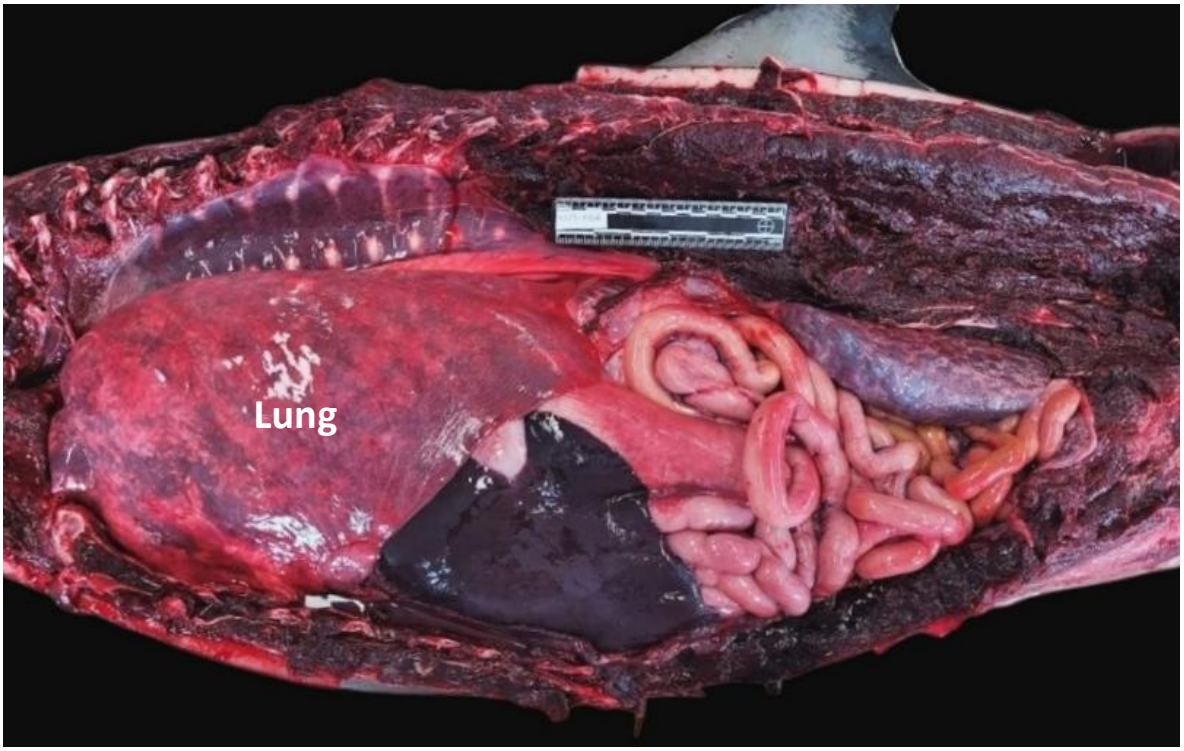


Figure 1.1. Common dolphin (*Delphinus delphis*): In-situ observation of visceral organs with rib cage removed showing left lung with areas of congestion and atelectasis. Photo: Cetacean Pathology Unit, Massey University.

Marine mammal stranding occurs when an animal is found outside of its natural aquatic environment, typically run aground on a beach, or in a helpless position where it is ill, weak, or disoriented, and often leads to its death. A marine mammal that is cast ashore already dead or beachcast is also considered stranded (Geraci & Lounsbury, 2005). The cause of stranding or death is sometimes categorized based on prominent findings observed at postmortem examinations and the results of histopathology and ancillary testing (Fenton et al., 2017; Giorda et al., 2017; Neimanis et al., 2022). Histopathological assessment is a practical and informative tool that provides pathological evidence and reinforces the necropsy conducted in dead cetaceans as part of the stranding response (Obusan et al., 2021).

The pulmonary system has consistently been reported as the organ system most commonly affected in stranded and bycaught cetaceans, with lesion prevalence ranging from 39% to 100% (Cornaglia et al., 2000; di Guardo et al., 1995; Gonzales-Viera et al., 2011; Greenwood & Taylor, 1977; Groch et al., 2020a; Rodrigues et al., 2018). Although other organs, such as the liver and intestines also show lesions (Cowan et al., 1986; di Guardo et al., 1995; Siebert et al., 2006), the disproportionately high frequency of lung pathology highlights the importance of respiratory disease in cetacean stranding and morbidity (Cornaglia et al., 2000; Rodrigues et al., 2018; Venn-Watson et al., 2012).

This trend has been observed across different decades and geographic regions, from early Pacific and European studies (Cowan et al., 1986; di Guardo et al., 1995) to more recent investigations in the Mediterranean, Atlantic, and South America (Cornaglia et al., 2000; Domiciano et al., 2016; Greenwood & Taylor, 1977; Groch et al., 2020a; Rodrigues et al., 2018), suggesting that it is not an artefact of a single population or study design but a consistent feature of cetacean health. Moreover, the variation in reported prevalence - ranging from one third to 100% of examined animals, likely reflects the health status of local populations (di Guardo et al., 1995; Siebert et al., 2020). Therefore, because the cetacean respiratory system is both highly specialised and vulnerable to natural disease and chemical pollutants, it can be utilised as an "*indicator organ*" for monitoring population health (Siebert et al., 2020).

1.3 Anatomy and Physiology of Cetacean Lungs

Cetaceans have an upper and lower respiratory anatomy that enables the rapid exchange of large volumes of air, control buoyancy, echolocation, and vocalisation in the aquatic environment (Fahlman et al., 2017; Otero-Sabio et al., 2021; Venn-Watson et al., 2012). The blowhole, which is analogous to the nostrils, is dorso-centrally positioned on a dolphin's head (Piscitelli et al., 2013). From the blowhole, air during inspiration passes through their elongated larynx, called the "goosebeak," a short and wide trachea, then ends in the non-lobulated lungs that are oblong to pyramidal in shape (Bernd et al., 2018; Piscitelli et al., 2013; St. Leger et al., 2018).

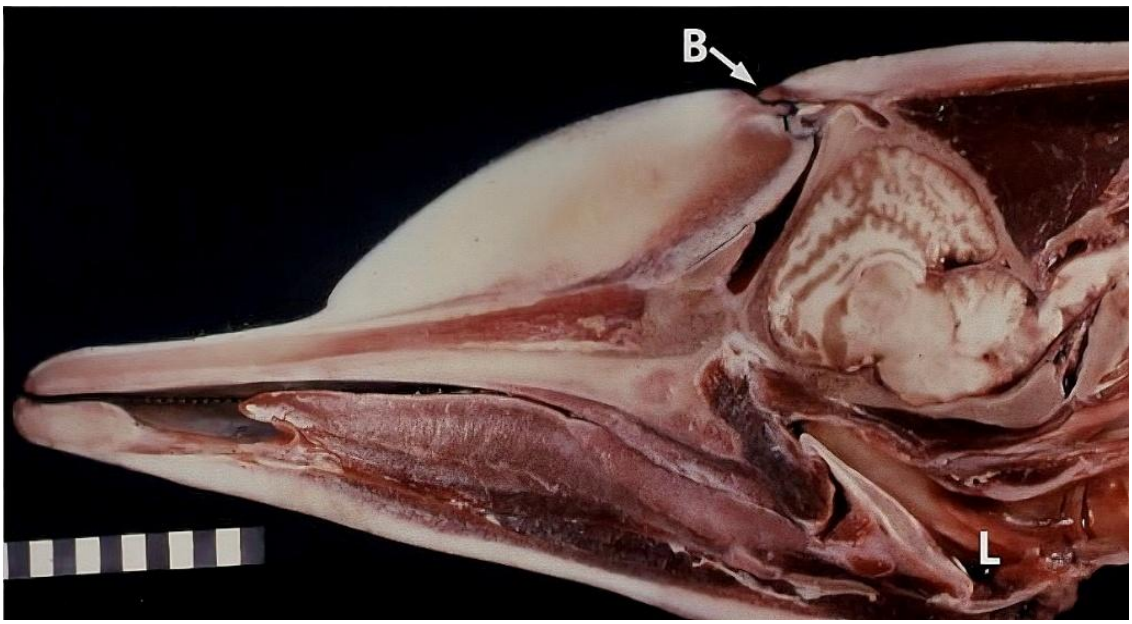


Figure 1.2. Common dolphin (*Delphinus delphis*): Midsagittal section of the head illustrating the blowhole (B) and larynx (L). Photo: Reidenberg and Laitman (2025).

Dolphins do not have upper airway filters or turbinates to warm, filter, or humidify air (St. Leger et al., 2018; Venn-Watson et al., 2012). This means that inhaled pathogens encounter minimal anatomical resistance before entering deep into the lungs (Venn-Watson et al., 2015). Their cartilaginous airways extend from the trachea down to the terminal bronchioles at the entrance of the alveoli or alveolar sac (Piscitelli et al., 2013). They also do not have respiratory bronchioles, which are non-cartilaginous airways lined with ciliated epithelial cells that normally follow the terminal bronchioles in terrestrial mammals (Fahlman et al., 2017; Murata, 1951). Instead, the terminal opening of each bronchus into the alveoli is surrounded by a muscular elastic sphincter. The alveoli appear as ellipsoid, ovoid, or elongated sacs when

inflated (Piscitelli et al., 2013; St. Leger et al., 2018). Scholander (1940) proposed that the cartilaginous reinforcement prevents the compression of the airway, facilitating alveolar collapse or atelectasis. This stops gas exchange in the alveoli, which also prevents the uptake of nitrogen and formation of gas emboli during ascent from a dive (Fahlman, 2024; Fahlman et al., 2017).

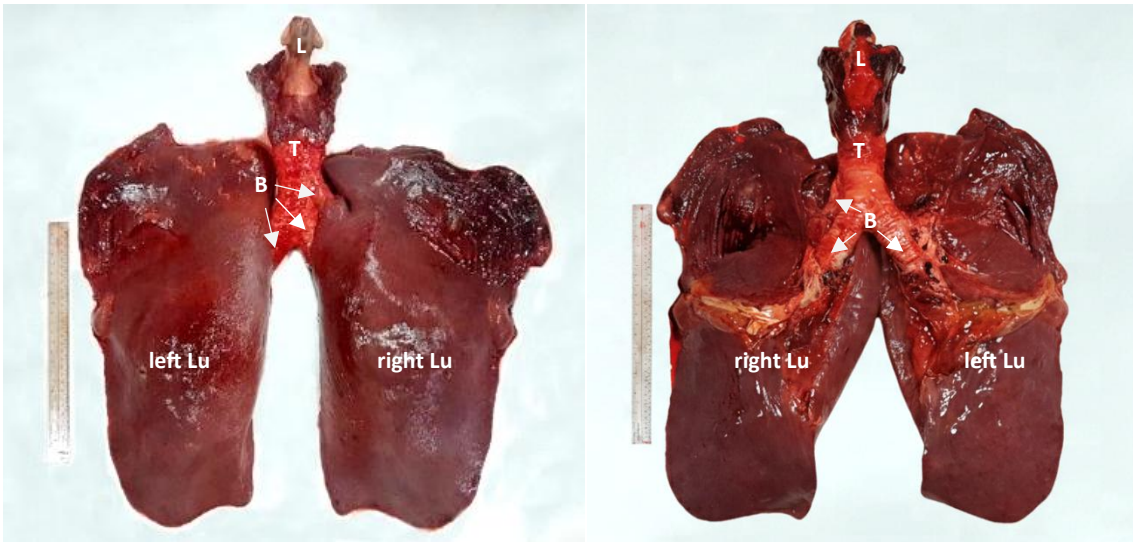


Figure 1.3. Common bottlenose dolphin (*Tursiops truncatus*): Dorsal (*left image*) and ventral (*right image*) aspect of the lungs showing the larynx (L), trachea (T), bronchi (B), and lungs (Lu). Scale bar = 30cm

Histologically, alveolar walls are lined by Type I and Type II pneumocytes and capillary endothelial cells. The Type I cells, which are elongated and tightly interconnected, form the lining of the alveolar septum where the gas exchange occurs, while the Type II cuboidal cells protrude into the alveolar lumen and secrete pulmonary surfactant (Piscitelli et al., 2013; St. Leger et al., 2018). Unlike terrestrial mammals, cetacean alveolar walls contain a double layer of capillary beds separated by a central connective tissue membrane, making each capillary in contact with only one alveolus rather than two (Piscitelli et al., 2013; St. Leger et al., 2018). This double capillary bed is often associated with a thicker alveolar septum, which increases the lung's mechanical compliance and structural resistance from extreme pressure fluctuations experienced during deep dives (Fahlman et al., 2017; Otero-Sabio et al., 2021; Piscitelli et al., 2013; St. Leger et al., 2018).

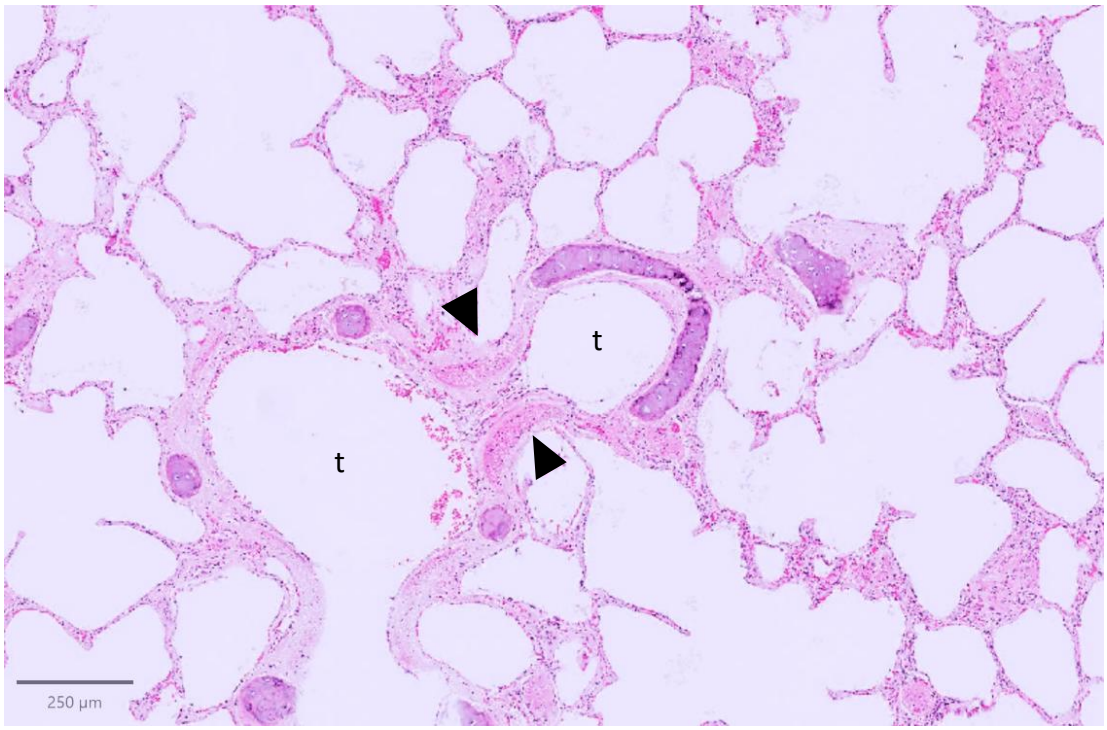


Figure 1.4. Common dolphin (*Delphinus delphis*)*:* Muscular elastic sphincter (arrows) lies between cartilaginous rings of a terminal bronchus (t), which extend to the alveolar sac entrance. Scale bar = 250µm.

Cetaceans depend heavily on their cardiovascular and pulmonary capacity to perform extended foraging dives beneath the water surface (Fahlman et al., 2015; Fischbach & Seguel, 2023). Before taking a breath, they normally begin exhaling just before their blowhole emerges from the water. This strong expiration clears any residual water from the blowhole and upper airways before the animal inhales (Fahlman et al., 2017; St. Leger et al., 2018). Delphinids complete the entire breathing process in less than a second, maintaining explosive and high velocity airflow reaching up to 200m/s during expiration and 44m/s at inspiration (Bernd et al., 2018; Fahlman et al., 2015; Piscitelli et al., 2013).

Humans generally exchange approximately 15 to 20% of their total lung volume during tidal breathing at rest (Fahlman, 2024; Fahlman et al., 2017). In contrast, dolphins take quick, deep breaths that replace 75% to 90% of the air in their lungs in less than a second (Cieri et al., 2024; Irving et al., 1941; Olsen et al., 1969; Venn-Watson et al., 2012). The exchange of air at the water's surface places dolphins in direct contact with aerosolised seawater and respiratory droplets from nearby conspecifics (Collier et al., 2025; Gupta et al., 2026). This rapid, high velocity, and efficient exchange of large volumes of air further increases exposure to

pollutants and pathogens at the ocean's surface and facilitates deep penetration of inhaled material into the lungs, increasing their vulnerability to respiratory infections (Durden et al., 2023; Venn-Watson et al., 2015; Venn-Watson et al., 2012). It is therefore not surprising that pulmonary pathologies represent one of the most commonly reported findings in cetaceans (Rodrigues et al., 2018; Sweeney & Ridgway, 1975; Tomo & Kemper, 2022).

1.4 Cetacean Lung Diseases

1.4.1 Pneumonia

Pneumonia is the most frequently diagnosed respiratory disease among stranded dolphins worldwide (Durden et al., 2023; Rodrigues et al., 2018; Venn-Watson et al., 2015). Broadly defined as an inflammation of the lung parenchyma, it results in consolidation (increased firmness) of the lung tissue as a result of the influx of fluid (oedema), red blood cells, inflammatory cells, or fibrin in the alveolar spaces, which impairs the gas exchange capacity of the lungs (Caswell & Williams, 2016; Côté & Silverstein, 2009; Jain et al., 2025; St. Leger et al., 2018; Torres et al., 2021). Pneumonia in cetaceans is caused by various pathogens including bacteria, viruses, parasites, and fungi (Jain et al., 2025; Mackenzie, 2016; St. Leger et al., 2018; Venn-Watson et al., 2012).

Bacterial lung infections, which often follow conditions that cause immunosuppression, are the most common cause of respiratory disease and death in many dolphin populations (Giorda et al., 2017; Groch et al., 2020a; Sweeney & Ridgway, 1975). Bacteria commonly involved in these infections include *Staphylococcus aureus*, *Streptococcus* spp., *Escherichia coli*, and *Pseudomonas aeruginosa* (St. Leger et al., 2018; Sweeney & Ridgway, 1975; Venn-Watson et al., 2012). Recent studies have also identified rare infections such as *Rhodococcus equi,* which is a terrestrial bacterium causing pyogranulomatous pneumonia (Oliviero et al., 2024), *Nocardia cyriacigeorgica* causing suppurative necrotising bronchopneumonia (Ito et al., 2021), and the first reported case of fatal fibrinonecrotising bronchopneumonia associated with *Morganella morganii* (Elfadl et al., 2017).

Cetacean Morbillivirus (CeMV) is the most significant viral pathogen, often causing acute systemic disease characterised by broncho-interstitial pneumonia and profound

immunosuppression that leads to secondary infections (di Guardo et al., 1995; Giorda et al., 2017; Groch et al., 2020a). The histopathological hallmark of this infection is the formation of syncytial cells within the alveolar and bronchial spaces and hyperplasia of the type II pneumocyte cells (di Guardo et al., 1995; Domingo et al., 1992; Groch et al., 2020b; St. Leger et al., 2018; Zafra et al., 2015). Parainfluenza virus has also been isolated from dolphins with pyogranulomatous broncho-interstitial pneumonia, while Influenza A subtypes have been associated with haemorrhagic pneumonia in some whales (Nollens et al., 2008; St. Leger et al., 2018; Venn-Watson et al., 2012).

Although less common than bacterial or viral infections, opportunistic fungal infections are increasingly identified as major contributors to dolphin mortality, particularly in individuals with compromised immune systems (Groch et al., 2018; St. Leger et al., 2018). Aspergillosis, which is caused by *Aspergillus fumigatus,* is the most frequently reported fungal infection, mainly because of its high virulence and thermotolerance (Garcia-Bustos et al., 2025; Meegan et al., 2012). Blastomycosis, caused by *Blastomyces dermatitidis,* and cryptococcosis, caused by *Cryptococcus gattii* or *C. neoformans* can also cause severe, fatal pulmonary disease, especially in debilitated animals (Cates et al., 1986; Groch et al., 2018; Venn-Watson et al., 2012).

1.4.2 Parasitic Lung Infection

Parasitic infection of the respiratory system is nearly universal in wild odontocetes, with some studies finding nematodes in nearly 100% of small cetaceans (Fauquier et al., 2009; Gonzales-Viera et al., 2011; Stockin et al., 2009). Lungworms such as *Halocercus* spp. and *Parafilaroides* spp. are frequently found in the bronchi, bronchioles and lung parenchyma, inducing parasitic (verminous) pneumonia (Guimarães et al., 2015; Measures, 2001). These infections often present as multifocal nodules or granulomas. In severe cases, the parasites cause thickening of the alveolar septa and muscles, reducing the size of the alveoli (Cornaglia et al., 2000; Demarque et al., 2020). Chronic cases often progress to dense fibrous scarring or dystrophic calcification, and the formation of firm, parasitic nodules where tissue has been destroyed (Guimarães et al., 2015). Further, evidence suggests that species like *Halocercus lagenorhynchi* can be transmitted transplacentally, which can lead to infections in stillborn or

very young calves (Fauquier et al., 2009). Air sinus flukes such as trematodes of the genus *Nasitrema*, typically found in the nasal sacs and pterygoid sinuses, can be aspirated into the lungs. Their presence can cause severe granulomatous eosinophilic bronchopneumonia (Arbelo et al., 2013; Gonzales-Viera et al., 2011).

1.4.3 Fluid Aspiration (Drowning)

Drowning among cetaceans is most often a result of bycatch, which is considered a major global threat to cetaceans (Bernaldo de Quirós et al., 2017; Read et al., 2006). These animals often exhibit stable white or red-tinged froth in the airways (Arbelo et al., 2013; McFee & Lipscomb, 2009). The seawater aspirated during drowning causes acute alveolar damage and a change in the osmotic gradient, leading to the rapid accumulation of proteinaceous fluid and froth in the airways (Jepson et al., 2013; Knieriem & García Hartmann, 2001). Histopathology of the lungs of bycaught cetaceans regularly show non-specific findings that include severe pulmonary congestion, pulmonary oedema, alveolar haemorrhage, alveolar hyperinflation, and emphysema (Ijsseldijk et al., 2021; Jepson et al., 2013; Knieriem & García Hartmann, 2001; Siebert et al., 2006).

Newborn and foetal dolphins are susceptible to respiratory conditions related to birth stress or in utero infection (Colegrove et al., 2016). Foetal distress during prolonged labour or asphyxiation triggers premature gasping *in utero*, resulting in meconium aspiration syndrome (Arbelo et al., 2013; Colegrove et al., 2016; Lair et al., 2015). Pathogens like *Brucella ceti*, which is frequently associated with reproductive failure and meningoencephalitis, can cross the placenta, causing *in utero* pneumonia that results in late-term pregnancy failure or death shortly after birth (Colegrove et al., 2016; St. Leger et al., 2018).

1.4.4 Barotrauma

Barotrauma refers to the physical injury to tissues caused by the pressure difference between an internal gas-filled space, such as the lungs or sinuses, and the surrounding environment (Cieri et al., 2024; Ioannidis et al., 2015). Uncontrolled rapid ascents from deep foraging dives induce extreme pressure changes that exceed lung tissue compliance causing tears in pulmonary tissues (Cieri et al., 2024; Fahlman, 2024; St. Leger et al., 2018). Although

cetaceans possess physiological adaptations to prevent barotrauma, such as alveolar collapse at depths to limit nitrogen uptake in the blood (Fahlman et al., 2017), intense acoustic stimuli, and other anthropogenic disturbances can override these protections by causing the animals to surface rapidly or by causing tissue damage through tissue resonance (Fahlman et al., 2021; Olsen et al., 1969; Ridgway et al., 1969).

Barotrauma in stranded cetaceans is most often associated with extreme anthropogenic events, most notably mid-frequency active sonar used during military activities, underwater explosions, and other high-energy acoustic sources, and remains an uncommon finding (Birkun Jr, 2002; Cox et al., 2006; Velázquez-Wallraf et al., 2021). Affected animals frequently show a complex "decompression-like" syndrome rather than simple hearing loss (Arbelo et al., 2013). The pathology of these events is often characterised by a "gas and fat embolic syndrome" that closely resembles decompression sickness in humans. In the lungs, heavy congestion, haemorrhages, and emphysema are often observed in animals that strand alive but succumb to cardiovascular failure (Fahlman et al., 2021; St. Leger et al., 2018; Velázquez-Wallraf et al., 2021).

1.4.5 Toxins

Toxin-related lung pathology in cetaceans occurs after exposure to petroleum hydrocarbons (Schwacke et al., 2014; Venn-Watson et al., 2015; Venn-Watson et al., 2012), harmful algal bloom (HAB) biotoxins (Fire et al., 2020) and persistent organic pollutants (POPs), all of which damage the normal structure and protective function of the respiratory system and weaken immune defences. Since a dolphin’s blowhole is at the surface of the water during inspiration, chemicals and toxins can be readily inhaled and cause irritation of the airway mucosa, peribronchiolar inflammation, necrosis of airway and alveolar tissues, severe secondary bacterial pneumonia, and systemic toxicity (Bossart, 2010; St. Leger et al., 2018; Venn-Watson et al., 2015). HAB toxins such as brevetoxins, domoic acid, and saxitoxins can also induce pulmonary congestion, oedema, haemorrhage, and acute cardiovascular collapse without inflammatory lesions (Bossart, 2010; St. Leger et al., 2018). “Chuffing”, which is a behaviour equivalent to coughing, has been observed to increase up to tenfold after being exposed to

Karenia brevis algal blooms, showing considerable respiratory irritation from aerosolised brevetoxins (Fire et al., 2020).

Long-lived contaminants like dichlorodiphenyltrichloroethanes (DDTs), polychlorinated biphenyls (PCBs), and mercury exacerbate the susceptibility of an animal to infection by suppressing immune function and increasing vulnerability to viral and opportunistic pathogens (Beineke et al., 2009; Cornaglia et al., 2000; Lahvis et al., 1995). These effects are stronger among cetaceans because of their respiratory physiology, making them particularly prone to toxin-induced respiratory disease (Irving et al., 1941; Olsen et al., 1969; St. Leger et al., 2018; Venn-Watson et al., 2015; Venn-Watson et al., 2012). However, toxin-related lung pathology in cetaceans is rare. Studies from the Canary Islands (Arbelo et al., 2013; Beineke et al., 2009; Díaz-Delgado et al., 2018), North America (Durden et al., 2023; McFee & Lipscomb, 2009; Venn-Watson et al., 2015), Canada (Lair et al., 2015), British waters (Baker & Martin, 1992; Jepson et al., 2000), Mediterranean waters (Cornaglia et al., 2000; Domingo et al., 1992), and the Amazon River (Rodrigues et al., 2018) all showed that infectious and parasitic diseases were the predominant respiratory lesions, with toxin-associated pulmonary injury rarely identified.

1.4.6 Neoplasia

Neoplasia in the lungs and other organs is rare in most cetacean populations previously studied (Newman & Smith, 2006; Suárez-Santana et al., 2016). One notable exception, however, is an isolated population of beluga whales (*Delphinapterus leucas*) in the St. Lawrence Estuary, Quebec, Canada (Lair et al., 2015). Neoplasia was determined as the second most frequent cause of death after infectious disease in the population. Adenocarcinoma of the gastrointestinal mucosa was the most common neoplasm identified. However, adenocarcinoma, chondroma, and lipoma of the lungs were also reported. The unusually high rate of cancer in these beluga whales is hypothesized to be linked to chronic exposure to carcinogenic environmental contaminants, such as polycyclic aromatic hydrocarbons (PAHs) and PCBs from industrial sources (Bossart, 2010; Lair et al., 2015; Newman & Smith, 2006; St. Leger et al., 2018).

Table 1.1. Summary of common cetacean lung pathologies from the published literature.

Pathology	Prevalence	Lung Features	Reference
Pneumonia	Very common	Inflammatory cell infiltration, consolidation, congestion, oedema, alveolar septal thickening, froth in airways	Birkun Jr (2002); Caswell and Williams (2016); Cornaglia et al. (2000); Durden et al. (2023); Gonzales-Viera et al. (2011); Lair et al. (2015); Sweeney and Ridgway (1975); Venn-Watson et al. (2012)
Parasitic Lung Infection	Very common	Parasite on airways, nodules/granuloma, congestion, oedema, focal emphysema	Arbelo et al. (2013); de Oliveira Carvalho Demarque et al. (2020); Fauquier et al. (2009); Neimanis et al. (2022); Venn-Watson et al. (2015); Zafra et al. (2015)
Drowning	Common	Oedema, congestion, froth in airways, haemorrhages, emphysema, hyperinflation	Arbelo et al. (2013); Attia El Hili and Maatouk (2024); Bernaldo de Quirós et al. (2017); Birkun Jr (2002); Ijsseldijk et al. (2021)
Barotrauma	Uncommon	Gas and fat emboli, congestion, haemorrhage, emphysema	Cox et al. (2006); Fahlman et al. (2021); St. Leger et al. (2018); Velázquez-Wallraf et al. (2021)
Toxins	Rare	Congestion, oedema, haemorrhage	Cornaglia et al. (2000); Díaz-Delgado et al. (2018); Durden et al. (2023); Rodrigues et al. (2018); St. Leger et al. (2018)
Neoplasia	Rare	Focal masses or nodules	Newman and Smith (2006); Suárez-Santana et al. (2016)

1.5 Pneumonia

The aetiology of pneumonia in dolphins is frequently multifactorial, with many cases resulting from polymicrobial infections (Cornaglia et al., 2000; Durden et al., 2023; Gonzales-Viera et al., 2011; Neimanis et al., 2022). In temperate North American waters, Durden et al. (2023) identified respiratory disease as the most common pathological finding in common bottlenose dolphins (*Tursiops truncatus*) over an 18-year monitoring period in the Indian River Lagoon system. Coastal common bottlenose dolphin populations in South Carolina also exhibited substantial mortality attributable to respiratory diseases, especially pneumonia (McFee & Lipscomb, 2009).

Mediterranean populations show similar patterns, with Cornaglia et al. (2000) documenting a high incidence of bacterial pneumonia in Italy between 1990 and 1997 among stranded common bottlenose dolphins, striped dolphins (*Stenella coeruleoalba*), and Risso's dolphins (*Grampus griseus*), with secondary infections frequently developing following initial viral or parasitic insults—a pattern consistent with earlier observations by Dierauf and Gulland (2001). Harbour porpoises (*Phocoena phocoena*) in British waters show similar aetiologies,

with bacterial and parasitic pneumonia representing major mortality factors (Baker & Martin, 1992; Siebert et al., 2001).

Cold-water environments show similar findings, as beluga whale populations in the St. Lawrence Estuary exhibit pneumonia as the primary cause of mortality (Lair et al., 2015), indicating that even cold-adapted species remain highly susceptible to respiratory pathogens. Tropical freshwater systems also present similar pathological patterns. For example, Rodrigues et al. (2018) reported pneumonia in 53.8% (28/52) of Amazon River dolphins (*Inia geoffrensis*), with verminous pneumonia and bacterial pneumonia each accounting for 25% of cases.

These examples show that the prevalence of pneumonia in cetacean stranding and mortality goes beyond geographic boundaries and species differences. Although regional differences in aetiology reveal important ecological insights, the universal pattern represents a significant cause of morbidity and mortality and highlights the susceptibility of dolphins to lower respiratory tract infections (Durden et al., 2023; Rodrigues et al., 2018; Venn-Watson et al., 2012).

Characterisation of pneumonia can be made according to the morphological pattern of the lesion in the affected lungs, its underlying aetiology, and the predominant type of inflammatory response or tissue reaction (Carvallo & Stevenson, 2022; Caswell & Williams, 2016; Jain et al., 2025; Venn-Watson et al., 2015; Zafra et al., 2015).

1.5.1 Morphologic Pattern (*anatomical spread and localization within the lung*)

Bronchopneumonia

Bronchopneumonia is marked by suppurative inflammation occurring at the bronchiolar-alveolar junction (Carvallo & Stevenson, 2022; Panciera & Confer, 2010). It is triggered by opportunistic bacterial pathogens, with the disease developing when there is either heightened bacterial exposure to the lungs, a compromise in pulmonary defence mechanisms, or a combination of both (Caswell & Williams, 2016; Côté & Silverstein, 2009; Jain et al., 2025). The cranioventral regions of the lungs are typically the most affected, particularly in terrestrial mammals, with bronchiolar and alveolar spaces filled with

neutrophils and, in some cases, fibrin or macrophages (Panciera & Confer, 2010). In severe and acute cases, pneumonia can progress to form lung abscesses. Localised tissue damage forms pockets that are filled with pus within specific areas of the lung (Clayton et al., 2012; Cusick & Bullock, 1973; Ito et al., 2021; Jain et al., 2025; Morick et al., 2023). Severe forms can involve complete loss of the normal pulmonary structure with diffuse infiltration of degenerated or necrotized neutrophils into the air spaces, and infiltration into destroyed bronchial cartilages, and multifocal fibrinous pleuropneumonia (Elfadl et al., 2017; Ito et al., 2021; St. Leger et al., 2018).

Interstitial Pneumonia

This type of pneumonia primarily affects the alveolar septa rather than the airspaces (Carvallo & Stevenson, 2022; Panciera & Confer, 2010). It is characterised by the hyperplasia of the alveolar septa due to an increase in type II pneumocytes and an intense inflammatory infiltrate composed of macrophages, lymphocytes, and plasma cells (Panciera & Confer, 2010; Zafra et al., 2015). Alveolar spaces may contain small amounts of proteinaceous fluid or scattered inflammatory cells but are generally less exudate-filled than in bronchopneumonia (Caswell & Williams, 2016; Zafra et al., 2015). This type of pneumonia can result from numerous causes, including viral infections, migrating parasite larvae, immune-mediated reactions, toxic gas inhalation and potentially adverse environmental conditions (Fahlman et al., 2017; Panciera & Confer, 2010; Venn-Watson et al., 2015; Zafra et al., 2015). In cetaceans, interstitial pneumonia is often associated with CeMV and may also include multinucleated cells, intranuclear, and/or intracytoplasmic inclusion bodies in the infiltrate (di Guardo et al., 1995; Groch et al, 2020a; Lipscomb et al., 1994; Zafra et al., 2015).

Broncho-interstitial Pneumonia

Broncho-interstitial pneumonia involves concurrent inflammation of the airway epithelium in the bronchi and alveolar septa within the lung parenchyma (Carvallo & Stevenson, 2022; St. Leger et al., 2018). This condition differs from bronchopneumonia, which involves only the bronchioles and alveoli and is typically due to bacterial infection (Caswell & Williams, 2016; Jain et al., 2025). Broncho-interstitial pneumonia is primarily caused by aerogenous viral infections or inhaled or ingested toxins that impact both bronchiolar and alveolar epithelial

cells (Caswell & Williams, 2016; Nollens et al., 2008; Venn-Watson et al., 2015). Lungs affected by broncho-interstitial pneumonia are typically firm, heavy, oedematous, and reddened, failing to collapse (Caswell & Williams, 2016; Domingo et al., 1992; Panciera & Confer, 2010; St. Leger et al., 2018). Histologically, broncho-interstitial pneumonia is identified by a combination of inflammation and/or necrosis of bronchiolar epithelial cells, type II pneumocyte hyperplasia, thickening of alveolar septa by mononuclear inflammatory cells, variable neutrophil infiltration within alveolar spaces, and syncytial cell formation and viral inclusion bodies in viral cases (Carvallo & Stevenson, 2022; Caswell & Williams, 2016; Di Guardo et al., 2005). In cetaceans, this type of pneumonia with type II pneumocyte hyperplasia is also associated with CeMV that leads to severe lesions (Domingo et al., 1992; Groch et al., 2020a; Venn-Watson et al., 2012).

Table 1.2. Morphologic patterns of pneumonia in cetaceans.

Pneumonia Type	Key Histologic Features	Common Causes / Aetiologies
Bronchopneumonia	Inflammation centred on bronchioles and adjacent alveoli; patchy consolidation; neutrophils and macrophages in airways	Bacterial infections, aspiration, secondary to viral disease
Interstitial	Thickened alveolar septa with lymphocytes, plasma cells, macrophages; type II pneumocyte hyperplasia; minimal alveolar exudate; syncytia possible	Viral infections (cetacean morbillivirus), toxins, immune-mediated processes
Broncho-interstitial	Bronchiolar epithelial necrosis; type II pneumocyte hyperplasia; septal thickening by mononuclear cells; neutrophils in alveoli; syncytia, viral inclusions possible	Viral (cetacean morbillivirus), mycoplasma, bacterial, parasitic, toxins

1.5.2 Aetiology

Bacterial

Pulmonary bacterial infection is the most prevalent disease and cause of death in small cetaceans (Sweeney & Ridgway, 1975). Documented bacterial pathogens implicated in dolphin pneumonia include *Aeromonas hydrophila* (Cusick & Bullock, 1973), *Proteus* spp*., Pseudomonas aeruginosa* (Venn-Watson et al., 2012), *Streptococcus* spp*., Escherichia coli* (Venn-Watson et al., 2015)*, Morganella morganii* (Elfadl et al., 2017), *Burkholderia*

pseudomallei, Mycobacterium spp*. (St. Leger et al., 2018), Actinobacillus delphinicola, Brucella* spp*., Enterococcus faecalis* (van Elk et al., 2019)*, Klebsiella pneumoniae* (Groch et al., 2020a), *Nocardia* spp. (Ito et al., 2021), *Erysipelothrix rhusiopathiae, Salmonella enterica, Shewanella putrefaciens (Neimanis et al., 2022), Staphylococcus aureus* (Meegan et al., 2012; Tomo & Kemper, 2022), *Vibrio alginolyticus, V. parahaemolyticus* (Tomo & Kemper, 2022), *Photobacterium damselae (Morick et al., 2023),* and *Rhodococcus equi (Oliviero et al., 2024).*

di Guardo et al. (1995) documented that diffuse or lobar, generally severe bilateral pneumonia—predominantly bacterial in nature—was the most frequently observed lesion among dolphins stranded in Italy between 1990 and 1993. The condition was diagnosed in 17 individuals (68%), including 11 of the 16 striped dolphins, and three bottlenose dolphins, the latter of which exhibited suppurative bronchopneumonia. A similar pattern emerged from examinations of stranded dolphins between 2007 and 2014 in the Pelagos Sanctuary, Italy (Giorda et al., 2017). These findings are consistent with previous reports that identified bacterial pneumonia as a common cause of mortality among wild cetaceans (Baker & Martin, 1992; Dierauf & Gulland, 2001; Neimanis et al., 2022; Siebert et al., 2001; Sweeney & Ridgway, 1975; Tomo & Kemper, 2022). More recent studies have confirmed that pneumonia, mostly bacterial and frequently associated with parasitic infections, reinforce the role of bacterial infections as a significant pathological factor in cetacean strandings (Giorda et al., 2017; Neimanis et al., 2022).

Viral

Viral pathogens have also been implicated in the aetiology of respiratory disease among cetaceans, notably CeMV (Domingo et al., 1992; Lipscomb et al., 1994) and parainfluenza virus (Nollens et al., 2008). CeMV is a highly significant global pathogen responsible for severe morbidity and mortality in dolphins (Bossart, 2010; St. Leger et al., 2018; Van Bressem et al., 2014). Both viruses are known to compromise immune function, making affected individuals more susceptible to secondary infections and contribute to chronic pulmonary pathology (Di Guardo et al., 2005; Nollens et al., 2008; Venn-Watson et al., 2012). Concerning CeMV, 53% of the striped dolphins examined by Cornaglia et al. (2000) exhibited chronic broncho-interstitial pneumonia characterised by the presence of endoalveolar syncytia, a hallmark of

morbillivirus infection. Additionally, 41.2% of these dolphins presented with non-purulent encephalitis, which emphasizes the virus's systemic impact after the initial infection in the respiratory system.

Parasitic

Bronchopneumonia is widely recognised as the most frequent pathological consequence of lungworm infections in marine mammals, as well as in domestic and terrestrial animals (Fischbach & Seguel, 2023). Parasites in the airways or pulmonary parenchyma can directly cause tissue damage and trigger an inflammatory response. This inflammation does not only compromise respiratory function but also creates an environment that is favourable for bacterial colonisation that further increases the severity of the disease (Measures, 2001).

In many studies, parasitic or verminous bronchopneumonia is frequently complicated by secondary bacterial infections, illustrating the dual threat of direct parasitic damage and indirect bacterial complications (Fischbach & Seguel, 2023). For example, *H. brasiliensis* infections were associated with chronic interstitial and fibrinopurulent bronchopneumonia, serving as the probable cause of mortality in 40% of the histopathologically evaluated Guiana dolphins (*Sotalia guianensis*) (Marigo et al., 2010). Altogether, these findings highlight the pathogenic significance of lungworms in cetaceans and their contribution to respiratory compromise and death.

Nematodes have similarly been documented in the pulmonary tissue of stranded dolphins in Italy, where Giorda et al. (2017) reported parasitic pneumonia ranging from mild to severe, with several cases complicated by secondary bacterial infections. Although lungworm infection is prevalent among cetaceans, documented outcomes vary substantially ranging from benign incidental lesions to fatal bronchopneumonia (Fauquier et al., 2009; Fischbach & Seguel, 2023; Measures, 2001).

The clinical signs and pathological alterations induced by pulmonary nematode infestations can vary according to the severity of the parasitic burden (Cowan et al., 1986; Measures, 2001; Tomo et al., 2010). In certain populations, such as the common bottlenose dolphins in

Southwest Florida, pneumonia is observed predominantly in neonates and calves, whereas infections in older age classes often present as chronic, resolving nodules that are of little to no pathologic significance (Fauquier et al., 2009; Geraci & St. Aubin, 1987).

Fungal

Fungal pneumonia in dolphins, categorised as a mycotic disease, is not common for cetaceans in the natural environment (Bossart, 2010; Venn-Watson et al., 2012). However, when it occurs, it is often associated with severe underlying conditions, particularly immunosuppression or chronic debilitation, leading to fatal secondary infections (Domingo et al., 1992; Groch et al., 2018; St. Leger et al., 2018). Pathogenic fungi implicated in pulmonary fungal infections in these cases include *Blastomyces dermatitidis* (Cates et al., 1986), *Coccidioides immitis* (Groch et al., 2018), *Cryptococcus gattii* (Gonzales-Viera et al., 2011; St. Leger et al., 2018), and *Aspergillus fumigatus* (Garcia-Bustos et al., 2025; Neimanis et al., 2022). In a case of an Atlantic spotted dolphin (*Stenella frontalis*) in Brazil, the severity and extent of lesions associated with *A. fumigatus* lesions was diagnosed as the likely cause of death (Groch et al., 2018).

1.5.3 Histologic Character (*type of inflammatory response and tissue reaction*)

Suppurative

Suppurative pneumonia is characterised by the accumulation of pus, composed predominantly of neutrophils, within the bronchioles and alveolar spaces (Panciera & Confer, 2010; Siebert et al., 2020; Venn-Watson et al., 2015). It is most often associated with bacterial infections in dolphins and other mammals (Caswell & Williams, 2016; Jain et al., 2025; Venn-Watson et al., 2015). Bacteria may be present extracellularly or within inflammatory cells, and extensive tissue necrosis is common (Clayton et al., 2012; Gonzales-Viera et al., 2011). In severe cases, dense aggregates of inflammatory cells can completely obscure the normal pulmonary architecture (Morick et al., 2023; St. Leger et al., 2018; Venn-Watson et al., 2015). Lesions often feature multifocal abscesses and areas where inflammatory cells and bacteria are surrounded by fibrous connective tissue (Elfadl et al., 2017; Oliviero et al., 2024). In some cases, it presents as granulomatous suppurative or necrotising pneumonia and is sometimes accompanied by abscess formation (Jain et al., 2025; Venn-Watson et al., 2015). Several

bacterial pathogens have been identified in association with suppurative pneumonia, including *Staphylococcus aureus* (Arbelo et al., 2013; Siebert et al., 2001), *Vibrio parahaemolyticus* (Bossart et al., 2019; Garcia-Bustos et al., 2025), *V. alginolyticus* (Tomo & Kemper, 2022), *Nocardia cyriacigeorgica* (Ito et al., 2021), *Nocardia farcinica* (Arbelo et al., 2013), *Photobacterium damselae* (Morick et al., 2023), *Rhodococcus equi* (Oliviero et al., 2024) and β-haemolytic *Streptococcus* species (Elfadl et al., 2017; Gonzales-Viera et al., 2011; Ito et al., 2021; Siebert et al., 2001; Tomo & Kemper, 2022).

In small cetaceans, lungworm infections are strongly associated with severe suppurative bronchopneumonia (Baker & Martin, 1992; Fauquier et al., 2009; McFee & Lipscomb, 2009; Measures, 2001). Parasitic migration and feeding activity within conducting airways and lung parenchyma induce mechanical mucosal trauma, impair mucociliary clearance, and introduce pyogenic bacteria into deep alveolar spaces (Fischbach & Seguel, 2023; Guimarães et al., 2015; Measures, 2001). It appears that the synergy between parasitic tissue destruction and opportunistic bacterial colonisation results in severe infections that exacerbate pulmonary disease (di Guardo et al., 1995; Fischbach & Seguel, 2023; Giorda et al., 2017; Siebert et al., 2001).

Fibrinous

Fibrinous pneumonia is defined by the presence of abundant fibrin exudation into the alveoli, bronchioles, and interlobular septa, often accompanied by serous fluid and inflammatory cells (Carvallo & Stevenson, 2022; Caswell & Williams, 2016; Elfadl et al., 2017). It is normally observed as a bilateral, cranioventrally distributed consolidation where affected lungs are firm, heavy, and frequently display yellow-white fibrin deposits on pleural surfaces (Panciera & Confer, 2010; Siebert et al., 2020; St. Leger et al., 2018). Histologically, prominent strands of eosinophilic fibrin are present in alveolar spaces and along septa, and the pleura is often covered by a thick fibrinous layer (fibrinous pleuritis) (Elfadl et al., 2017; St. Leger et al., 2018). This pattern is typically associated with severe, toxin-producing bacterial infections and is highly fatal (Caswell & Williams, 2016; Panciera & Confer, 2010). In cetaceans, fibrinous pneumonia has been documented in association with both bacterial and viral infections, including CeMV, and may occur concurrently with other exudative patterns (Domingo et al., 1992; Giorda et al., 2017; Groch at al., 2020a; Siebert et al., 2001; Venn-Watson et al., 2012).

Granulomatous

Granulomatous pneumonia is a chronic inflammatory lung disease characterised by the presence of granulomas (organised aggregates of macrophages) often including multinucleated giant cells, lymphocytes, plasma cells, and fibrous tissue (Rosen, 2022). These nodular lesions may be multifocal or coalescing, vary in size, and sometimes exhibit central caseous necrosis or mineralisation. (Caswell & Williams, 2016; Geraci & St. Aubin, 1987; Mackenzie, 2016). In dolphins, it frequently results from parasitic, bacterial, or fungal infections (Domiciano et al., 2016; Groch et al., 2018; Siebert et al., 2020; Venn-Watson et al., 2012). However, the appearance of granulomatous lesions is non-specific and does not indicate the aetiology unless the causative agent is detected within the granuloma or confirmed by culture. This makes it challenging to identify the cause or disease link of the lesions (Mackenzie, 2016; Oliviero et al., 2024; Rosen, 2022).

Necrotising

Necrotising pneumonia is a severe pulmonary condition that is characterised by extensive areas of tissue necrosis and complete destruction of the normal alveolar architecture (Carvallo & Stevenson, 2022; Kapania & Cavallazzi, 2024). Histologically, the alveoli contain macrophages and large amounts of necrotic cellular debris, and are lined by proliferating type II pneumocytes, with thickening of alveolar septa by inflammatory cells (Sarli et al., 2021). Cases of necrotising pneumonia in dolphins are most often associated with highly pathogenic bacteria, including *Nocardia cyriacigeorgica, Klebsiella* spp., *Streptococcus* spp., *Corynebacterium* spp*., E. coli, E. rhusiopathiae, Pseudomonas* spp. and *S. aureus* (Ito et al., 2021; Siebert et al., 2001; Venn-Watson et al., 2012). Lesions frequently overlap with suppurative or fibrinous patterns and, in chronic cases, can progress to abscess formation or cavitation (Clayton et al., 2012; Kapania & Cavallazzi, 2024; Morick et al., 2023).

Table 1.3. Inflammatory response of pneumonia in cetaceans.

Inflammatory Response	Key Histologic Features	Common Causes / Aetiologies
Suppurative	Neutrophil-rich exudate filling bronchioles & alveoli; bacteria intra/extracellular; necrosis; abscesses	Bacteria (*Vibrio parahaemolyticus*, *V. alginolyticus*, *Staphylococcus aureus*, *Nocardia cyriacigeorgica*)
Fibrinous	Abundant fibrin in alveoli, bronchioles, and septa; fibrinous pleuritis	Severe bacterial infections (*Pasteurella*, *Mannheimia* spp.); morbillivirus co-infection
Granulomatous	Granulomas with macrophages, multinucleated giant cells & lymphocytes; fibrosis; necrosis/mineralisation possible	Chronic fungal (*Aspergillus*, *Blastomyces*), mycobacterial, parasitic infections
Necrotising	Extensive tissue necrosis; loss of alveolar architecture; debris, fibrin, microbial colonies; haemorrhage/oedema	Highly virulent bacteria (*Nocardia*, *Klebsiella*, *Streptococcus* spp.)

1.6 Parasitic Lung Infection

Helminth parasitism of the respiratory and gastrointestinal system is common in stranded cetaceans and has been documented in animals from the North Sea (Baker & Martin, 1992; Siebert et al., 2001), eastern Pacific Ocean (Gonzales-Viera et al., 2011), the Gulf of Mexico (Fauquier et al., 2009), South Atlantic Ocean (Demarque et al., 2020; Guimarães et al., 2015; Marigo et al., 2010). Cornaglia et al. (2000) reported that parasitic infections were more prevalent in the lungs (29.2%) than in the stomach (20.8%) of examined cetaceans in their study. Moreover, the ulcerative lesions and chronic inflammation induced by pulmonary parasites were more severe than those caused by alimentary-tract parasites (Cornaglia et al., 2000; Marigo et al., 2010). This difference in clinical impact demonstrates the importance of respiratory parasitism in the health of cetaceans. Indeed, gastrointestinal parasitic lesions are generally considered to be of limited clinical importance in cetaceans (Baker & Martin, 1992; Siebert et al., 2006; Sweeney & Ridgway, 1975), in contrast to the more pathological consequences observed in the respiratory system (Geraci & St. Aubin, 1987; Measures, 2001).

Metastrongyles, also known as lungworms, are helminth parasites commonly found in the respiratory and cardiovascular systems of mammals (Measures, 2001). In marine mammals,

these parasites can significantly impair these body systems and affect their respiratory capacity and diving (de Oliveira Carvalho Demarque et al., 2020; Geraci & St. Aubin, 1987). Several nematode species belonging to the family Pseudaliidae have been documented in the lungs and lower respiratory tract of cetaceans. Specifically, *Pseudalius inflexus, Stenurus* spp*., Torynurus convolutus* and *Halocercus* spp. are frequently observed in pulmonary infections (Fauquier et al., 2009; Fenton et al., 2017; Fischbach & Seguel, 2023; Measures, 2001). These nematodes, which have long been recognised as a source of severe pulmonary pathology in cetaceans. In particular, the genus *Halocercus* has a widespread occurrence and pathogenicity in the lungs among odontocetes (Measures, 2001; Rodrigues et al., 2018), implicated in causing bronchitis, bronchopneumonia, and tissue necrosis (Geraci & St. Aubin, 1987).

Lungworm infections associated with stranding or mortality have been documented in harbour porpoises*,* striped dolphins, and finless porpoises (*Neophocoena phocaenoides*) (Cornaglia et al., 2000; McFee & Lipscomb, 2009; Siebert et al., 2001). In common bottlenose dolphins, *H. lagenorhynchi* represents a major pathogen responsible for verminous pneumonia (Sweeney & Ridgway, 1975). Lungworms feed on epithelial–endothelial cells, host debris, and occasionally blood in the airways and respiratory sinuses or cardiovascular system (Fauquier et al., 2009; Geraci & St. Aubin, 1987).

de Oliveira Carvalho Demarque et al. (2020) provided compelling evidence of the pathological impact of *H. brasiliensis* in Guiana dolphin from Brazil. All examined individuals were infected by the parasite, and 60% exhibited notable pulmonary lesions. These included emphysema and atelectasis, always observed together, alongside pulmonary oedema, and thickening of the septal and alveolar wall. The latter resulted in reduced alveolar space and a structurally massive lung. Mineralisation of bronchiolar cartilage and mononuclear inflammatory infiltrates further illustrated the chronic and multifocal nature of the damage.

Assessing the clinical significance of pulmonary parasitism in odontocetes presents considerable challenges (Lair et al., 2015; Measures, 2001). Progression from subclinical infestation to clinically significant respiratory disease, which is often complicated by secondary bacterial infections, may be influenced by a range of immunosuppressive factors

(Fenton et al., 2017; Geraci & St. Aubin, 1987). Contaminants, infectious diseases, and stress make odontocetes more prone to acquiring severe lung diseases from pseudaliid infection (Measures, 2001). These include exposure to persistent organic pollutants (POPs) and HAB toxins (Jepson et al., 1999; St. Leger et al., 2018), co-infection with viral pathogens (Groch et al., 2020a), and environmental stressors such as increased marine traffic and anthropogenic noise (Birkun Jr, 2002; Siebert et al., 2020). The reasons for these variable outcomes are not fully understood, but the site of infection, burden, and host immune status have been indicated as drivers of pathogenic outcomes (Bossart, 2010; Forbes, 2021). Indeed, although lungworms commonly cause bronchopneumonia in cetaceans, the association of the parasitism and mortality is not definitive (Fischbach & Seguel, 2023).

1.7 Non-specific Lung Pathologies

1.7.1 Oedema and Congestion

Pulmonary oedema and congestion are commonly observed postmortem in cetacean lungs and generally regarded as non-specific or perimortem changes rather than indicators of primary disease (Bernaldo de Quirós et al., 2017; Jepson et al., 2013; Siebert et al., 2006). Pulmonary oedema is defined by the abnormal accumulation of transudate fluid within the pulmonary interstitium and alveolar spaces (Caswell & Williams, 2016; Diana et al., 2009). In stranded cetaceans, particularly in drowning cases, the fluid appears blood-tinged due to the leakage of erythrocytes and proteinaceous fluid into the alveoli associated with hypoxic damage to the integrity of the alveolar membranes and osmotic gradient created by saltwater in the alveoli (Jepson et al., 2013; Knieriem & García Hartmann, 2001; Lunetta & Modell, 2005). Affected lungs grossly appear wet, heavy, and fail to collapse (Carvallo & Stevenson, 2022; Ijsseldijk et al., 2021). Microscopically, the oedema appears as eosinophilic or proteinaceous fluid filling the alveoli and is often accompanied by congested capillaries and thickened alveolar septa (Caswell & Williams, 2016; Jepson et al., 2013; Knieriem & García Hartmann, 2001).

Pulmonary congestion is defined by the abnormal accumulation of blood within the pulmonary vasculature and parenchyma (Carvallo & Stevenson, 2022; Caswell & Williams, 2016). Grossly, congested lungs in cetaceans typically appear dark red in colour and wet,

heavy, and glistening, as it is often associated with pulmonary oedema (Arbelo et al., 2013; Jepson et al., 2013). Histologically, it is characterised by engorged alveolar capillaries filled with red blood cells and distended septal vasculature (Ijsseldijk et al., 2021; Knieriem & García Hartmann, 2001; Torres et al., 2021).

These lesions are commonly documented in bycaught and stranded cetaceans and often observed with alveolar haemorrhage and hyperinflation, regardless of the underlying cause of death (Jepson et al., 2013; Siebert et al., 2006; Siebert et al., 2001). They are frequently interpreted as an agonal or perimortem event, reflecting physiological disturbances associated with hypoxia and terminal cardiopulmonary dysfunction (Lunetta & Modell, 2005; Siebert et al., 2020). Furthermore, pulmonary transudation can occur postmortem from continued atrio-ventricular and sino-atrial node contractions in the heart that may persist for 5 to 10 minutes (Ijsseldijk et al., 2021). This elevates pulmonary microvascular pressure, causing fibrin and fluid to leak into the alveoli (Arbelo et al., 2013; Jepson et al., 2013). Further, Bernaldo de Quirós et al. (2017) found no difference in the prevalence of pulmonary oedema between dolphins that succumbed from drowning and those that were euthanised after stranding, which emphasizes its non-specific nature.

Although other studies also note that alveolar oedema and congestion often occur with pneumonia and parasitic lung disease, their presence alone does not reliably distinguish primary pulmonary disease from terminal or post-mortem processes (Domiciano et al., 2016; Siebert et al., 2020). Similar patterns have also been described in dolphins exposed to high-pressure blasts or sonar, where the lesions tend to be most pronounced on the side of impact (Cox et al., 2006; St. Leger et al., 2018; Velázquez-Wallraf et al., 2021). Overall, because pulmonary oedema and congestion are common but non-diagnostic findings in cetacean lungs, documenting them is essential for contextual interpretation of necropsy findings, but should be interpreted cautiously and not used in isolation to infer primary pulmonary disease (Arbelo et al., 2013; Ijsseldijk et al., 2021; Kuijpers et al., 2022).

1.7.2 Emphysema

Emphysema is a pathological condition that is defined by the abnormal expansion of lung tissue due to trapped air or gas, leading to local or lobular distension of the pulmonary

parenchyma (Carvallo & Stevenson, 2022; Caswell & Williams, 2016). Affected regions appear grossly voluminous, pale and puffy, reflecting excessive air accumulation within the lungs (Meyerholz et al., 2017; Velázquez-Wallraf et al., 2021; Venn-Watson et al., 2015). Histologically, emphysematous lungs show destruction of alveolar septa and the formation of enlarged, coalescing airspaces (Caswell & Williams, 2016; Colegrove et al., 2016; Guimarães et al., 2015). While its presence is common among bycaught and stranded cetaceans, pathologists often interpret emphysema as an agonal finding or a non-specific change that is associated with the process of death (Birkun Jr, 1994; Cowan et al., 1986; Siebert et al., 2006).

1.7.3 Atelectasis

Atelectasis, defined as the collapse or the incomplete expansion of the alveoli resulting in the loss of effective gas exchange, is both a critical physiological adaptation and a common pathological finding in stranded cetaceans (Caswell & Williams, 2016; Fahlman et al., 2017). It often occurs as a consequence of a disease process, such as severe pneumonia that causes airway obstruction (e.g., exudates, mucus, or pulmonary nematodes) (Fauquier et al., 2009; Measures, 2001; Rodrigues et al., 2018), compression of the lungs by space-occupying lesions within the thoracic cavity (e.g., hydrothorax, pleural effusions, or large tumours) (Caswell & Williams, 2016; St. Leger et al., 2018), or failure of initial lung inflation in neonates and stillborn calves (Colegrove et al., 2016; Díaz-Delgado et al., 2018). Collapsed regions grossly appear purple-red in colour and consolidated (Caswell & Williams, 2016). Apart from the collapsed alveoli, amniotic squames, and meconium are also noted in neonates (Arbelo et al., 2013; Colegrove et al., 2016). Atelectasis, which commonly occurs with emphysema and congestion in animals with acute respiratory or circulatory failure, produces a patchy “checkerboard” pattern of alternating collapsed and over-inflated tissue (Birkun Jr, 1994; Díaz-Delgado et al., 2018).

1.8 Thesis Rationale and Objectives

While the significance of respiratory diseases within cetaceans is recognised as a major health concern in dolphins globally, there remains a lack of systematic lung assessment in most species and populations. However, if a lung health score could be devised to monitor lung health in a population over time (for example, in the context of environmental contaminants

e.g., microplastics) this would fill a critical knowledge gap, particularly given the role of dolphins as sentinel species for marine ecosystem health (Domiciano et al., 2016).

This study explored lung health in 122 common dolphins, utilizing a 26-year tissue archive and associated data sets collected from stranded and bycaught animals in New Zealand. Specifically, the primary objective of this thesis is to apply a preexisting lung health scoring system devised for Northern Hemisphere harbour porpoise to (1) instigate baseline data collection on respiratory health, (2) examine the prevalence and aetiology of pneumonia, and (3) detail the prevalence of lung parasitism. This will enhance current understanding of lung health and aetiology of pneumonia in common dolphins, while providing a baseline from which lung health can be monitored in the context of ocean health markers (e.g., persistent contaminants, ocean acidification). Ultimately, these findings can then be used to monitor changes within this population as indicators of marine ecosystem health.

1.9 Thesis Structure

This thesis follows a traditional thesis format as follows:

- Chapter 1 – Introduction: Summarises existing literature on cetacean lung anatomy and physiology, lung health, pneumonia, lung parasitism, and other lung pathologies.
- Chapter 2 – Materials and Methods: Describes the full methodology used to select the cases in the datasets, determine lung health scores, criteria used to classify severity of pneumonia and lung parasitism, identification of parasites, data processing, and statistical method used.
- Chapter 3 – Results: Outlines the findings of this thesis, quantifying the lung health scores, prevalence, and aetiology of pneumonia, and prevalence of lung parasitism in common dolphins.
- Chapter 4 – Discussion: Discusses the main findings of this thesis in the context of existing literature and outlines the study limitations and recommendations for future research.

Chapter 2. Materials and Methods

2.1 Postmortem Sampling

Postmortem records, tissues and parasites of common dolphins examined from 1999 to 2025 were accessed from BIOCET – Cetacean Pathology Unit (CPU), Massey University, for use in this study. Necropsies were performed by the CPU team over this period. I took part in the necropsies of some of the most recent cases and otherwise worked from the archived necropsy records, photographs, tissues and parasites. Data for sex (male or female), age (years), sexual maturity (immature or sexually mature), and body condition score (good, moderate, or poor) were extracted from the CETPATH database (CPU, Massey University). Following Ijsseldijk et al. (2019), only dolphins coded 1 to 3 for decomposition condition coding (DCC) were included (Table 2.1), where tissue preservation was deemed acceptable for histopathological assessment by a board-certified veterinary pathologist.

A total of 122 dolphins, of which 78 were single strandings, 30 were from mass strandings and 14 were bycaught, all of which met the DCC and tissue preservation criteria for inclusion in the study. Animal source was classified as single stranding, mass stranding or bycatch. A single stranding was defined as an animal or a cow–calf pair found stranded alone, and a mass stranding as two or more animals, excluding a cow–calf pair, stranded together at the same time and place (Geraci & Lounsbury, 2005). Both live-stranded animals that subsequently died or were euthanised and animals found beachcast were included in these categories. Bycatch was defined as animals recovered from fishing gear or showing evidence of net entanglement. The 30 mass-stranded dolphins came from six mass stranding events.

At necropsy, the lungs were serially sectioned transversely to expose the parenchyma, and the tissue was grossly described. Samples were collected from predetermined regions of both the left and right lung (the pleural surface and underlying parenchyma of the cranial, middle, caudal, and hilar regions), with additional samples taken from any gross lesion, as part of routine histopathological assessment. Samples were fixed in 10% buffered formalin and submitted, together with samples from other organs, to Awanui Veterinary, which processed the tissues and prepared the histological slides. Some slides therefore contained lung tissue

together with other organs. The number of slides per animal was not recorded in this study. Histopathological findings were taken from the reports of the pathologists who examined the slides (Section 2.2.2).

Table 2.1. Decomposition condition coding (DCC) based on Ijsseldijk et al. (2019).

DCC	Definition
Code 1 Extremely fresh carcass, just dead	Carcasses that live stranded and have died or were euthanised very recently and show no post-mortem changes (e.g. no bloating or sloughing of skin), fresh smell, clear glassy eyes, firm white blubber, well-defined dark red muscles, intact and well-defined viscera, no to minimal gas in the GIT (unless pathogenic), and a firm, non-discoloured brain with distinct surface features that can be easily removed intact.
Code 2 Fresh Carcass	Carcasses with a normal external appearance, fresh smell, and only minimal drying and wrinkling of skin, eyes, and mucous membranes. There is no bloating, and the tongue and genitalia are not protruded. Blubber remains firm and white, but occasionally blood-tinged.
Code 3 Moderate Decomposition	Carcasses with noticeable bloating, often with a distended tongue or penis; skin cracking and beginning to slough; a mild characteristic odour; sunken eyes and dry mucous membranes. Blubber is oily and blood-tinged, muscles are soft and poorly defined, gut segments contain gas, and the brain has a soft consistency. Organs remain largely intact, identifiable, and easily removed and assessed. However, colours and consistencies are becoming more uniform, and some tissues (e.g., kidneys, pancreas) are increasingly friable.
Code 4 Advanced Decomposition	Carcasses may be intact but collapsed with extensive skin sloughing and epidermal loss, exposing blubber. A strong odour is present. Blubber is soft and often with gas pockets and pooled oil; muscles are nearly liquefied and separate from the bone with little effort; blood is thin and dark. Viscera are still identifiable, but friable, easily torn and difficult to dissect; the gut is gas-filled, and the brain is liquefied, dark red, and contains gas pockets.
Code 5 Mummified or skeletal remains	Skin can cover skeletal remains, and the remaining tissues are desiccated. Organs may be absent or unrecognizable.

2.1.1 Body Condition Score (BCS)

The body condition score (BCS) or the nutritional status of each animal was classified as – BCS 1 (good), BCS 2 (moderate), or BCS 3 (poor) following Stockin et al. (2009). Individuals with rounded cranial and caudal dorsal flanks were classified as BCS 1. Individuals with sloping cranial and caudal dorsal flanks were classified as BCS 2. Individuals with hollow or concave cranial and caudal dorsal flanks, visible transverse processes of the lumbar vertebrae, and

indentation or concave caudo-dorsal aspect of the head were classified as BCS 3.

2.1.2 Sex Determination

Sex was determined anatomically based on the presence of sex-specific gonads - testes were identified in males, and ovaries and uterus in females. This was confirmed grossly during dissection.

2.1.3 Sexual Maturity and Age Class

Animals were classified as immature or mature based on gonadal development, following Palmer et al. (2022) and Palmer et al. (2023). In males, sexual maturity was confirmed by the presence of active spermatogenesis in the seminiferous tubules on histopathology. In females, sexual maturity was confirmed if the animal was pregnant (foetus present in the uterus), lactating, or had at least one *corpus albicans* or *corpus luteum* in either ovary during gross examination. Since foetuses and neonates may display different levels of pulmonary parasitism, a subclass of sexually immature animals called “perinates” was further included. Perinates included all foetuses extracted *in utero* or aborted before their first breath as confirmed by pulmonary atelectasis, and neonates as defined by the presence of vibrissae and/or unhealed navel, foetal folds, soft and folded dorsal fin and tail flukes, or non-erupted teeth.

2.1.4 Age Determination

Age was determined using decalcified, stained thin sections of teeth following Murphy et al. (2014). Teeth were sectioned and stained at the School of Veterinary Science of Massey University (Dr Matthew Perrott). Sections were examined at 10× to 40× magnification and ages estimated by counting annual growth layer groups (GLGs) in the dentine, as described by Myrick et al. (1983) and Lockyer (1995). Each tooth was read without prior biological information by two or three trained readers of the CPU (Emma Betty, Evi Hanninger, and Emily Palmer). If initial readings did not agree, additional teeth were sectioned and re-examined until consensus on the best age estimate or an age range was reached, following Westgate and Read (2007). Individuals with teeth that were too worn or damaged for reliable GLG counts were excluded from age-based analyses. Animals in which the neonatal line was absent or only just forming were assigned an age of 0 years.

2.1.5 Parasitism

To determine the prevalence of lung parasitism, carcasses of DCC 4 were added to the sample set since nematodes remained grossly and histologically visible despite autolysis. Accordingly, a total of 141 dolphins (97 and 30 single and mass stranding respectively, and 14 bycaught cases) were included in the parasitism dataset.

2.2 Lung Health Parameters

The parameters used to assess lung health in this study were based on Schick et al. (2024). Three main pathological parameters were evaluated: parasitism, inflammation, and irreparable tissue damage. These parameters were selected since they are the most frequent markers and lesions encountered in cetacean respiratory pathology (Durden et al., 2023; Geraci & St. Aubin, 1987; St. Leger et al., 2018). Collectively, these represent both acute and chronic disease processes.

2.2.1 Parasitism

Pseudaliid nematodes can inhabit both the bronchial tree and pulmonary blood vessels of cetaceans (Measures, 2001). In this study, only the presence of lungworms in the bronchial tree and lung parenchyma was graded, since these were the only regions systematically examined during all dissections. At necropsy, lungworms were detected by visual observation in the trachea and bronchi (Figure 2.1) and by palpation of nodules within the lung parenchyma (Figure 2.2). Lungworms were also identified in histological sections (Figure 2.3a). Lungworms were collected opportunistically and preserved in 70% ethanol for identification. Severity was categorised based on gross and histologic findings using the criteria in Table 2.2.

The criteria were adapted from Marigo et al. (2010), Tomo et al. (2010) and van Elk et al. (2019), and the numerical thresholds were defined for this study in Table 2.2. Absence of lungworms was recorded as no parasitism (0 points). Mild infection was defined as fewer than 10 adult or larval worms in one or both lungs, less than 10% bronchial lumen obstruction, and fewer than 5 parasitic nodules or granulomas. Moderate infection was defined as 10-30 worms, 10–20% bronchial lumen obstruction, and 5–10 nodules or granulomas. Severe

infection was defined as more than 30 worms, more than 20% bronchial lumen obstruction, and more than 10 nodules or granulomas. Histologically, lungworms were categorised as rare to occasional (mild; 10 or fewer ova, larvae, or adult fragments per section), occasional to frequent (moderate), or frequent to numerous (severe). Histological frequency was assigned by the reporting pathologists as a semi-quantitative impression, without numerical thresholds beyond those for mild infection. Bronchial lumen obstruction was estimated visually at necropsy. Lungs meeting the criteria for multiple categories were classified according to the highest severity. In cases where gross and histologic assessments differed, the gross pathology grade was used as the final severity assignment**.**

Table 2.2. Summary of parasite infection criteria applied in this study.

Severity	Criteria	
	Gross Observation	**Histological Observation**
None	No lungworms	No lungworms
Mild	<10 worms in one or both lungs, <10% bronchial lumen obstruction, <5 parasitic nodules/granulomas	Rare to occasional (≤10 ova, larvae or adult fragments)
Moderate	10-30 worms in one or both lungs, 10-20% bronchial lumen obstruction, 5-10 parasitic nodules/granulomas	Occasional to frequent
Severe	>30 worms in one or both lungs, >20% bronchial lumen obstruction, >10 parasitic nodules/granulomas	Frequent to numerous

Parasite Identification

Parasite identification, both morphological and molecular, was carried out at the University of Otago in collaboration with Dr Bronwen Presswell.

Morphologic Methods

For microscopic examination, nematodes were stained with lactophenol and prepared as temporary mounts. Photomicrographs taken with an Olympus BX51 microscope fitted with a DP25 camera were analysed using ImageJ software (Wayne Rasband, NIH, USA) to assist with morphological evaluation. Specimens from 9 animals were examined and identified to the lowest possible taxonomic level based on the morphological keys of Anderson et al. (2009),

Gibbons (2010), and original species descriptions by Baylis and Daubney (1925) and Zylber et al. (2002).

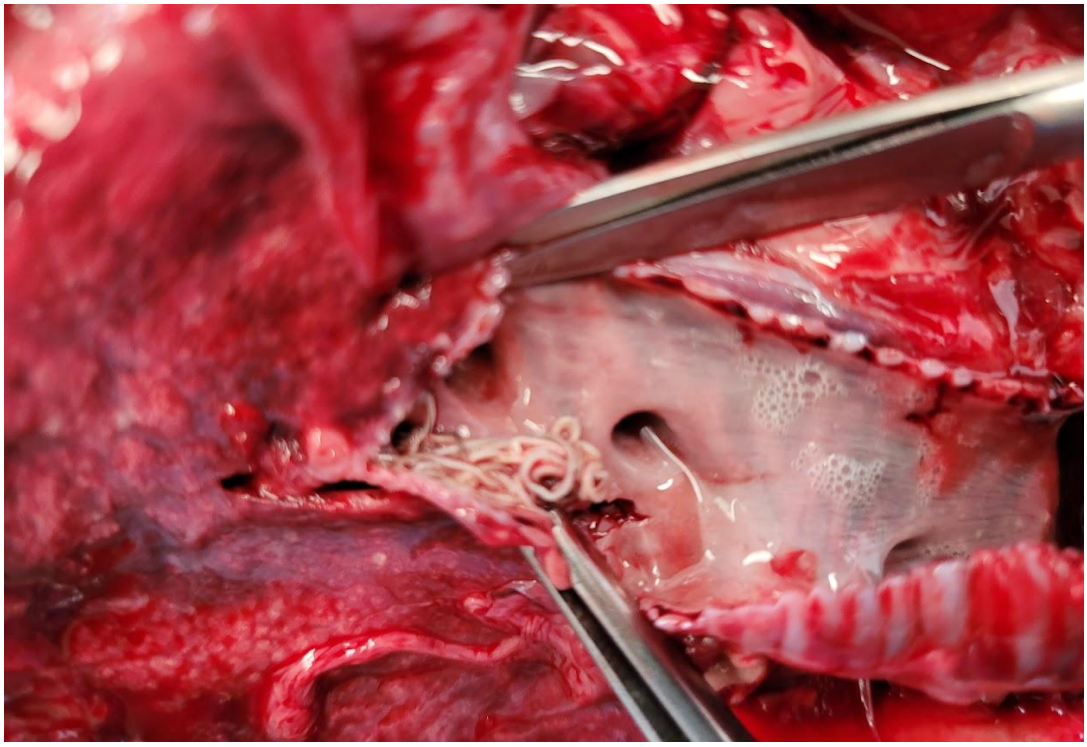


Figure 2.1. *H. lagenorhynchi* lodged in the bronchus of KS23-06Dd. Photo: Cetacean Pathology Unit, Massey University.

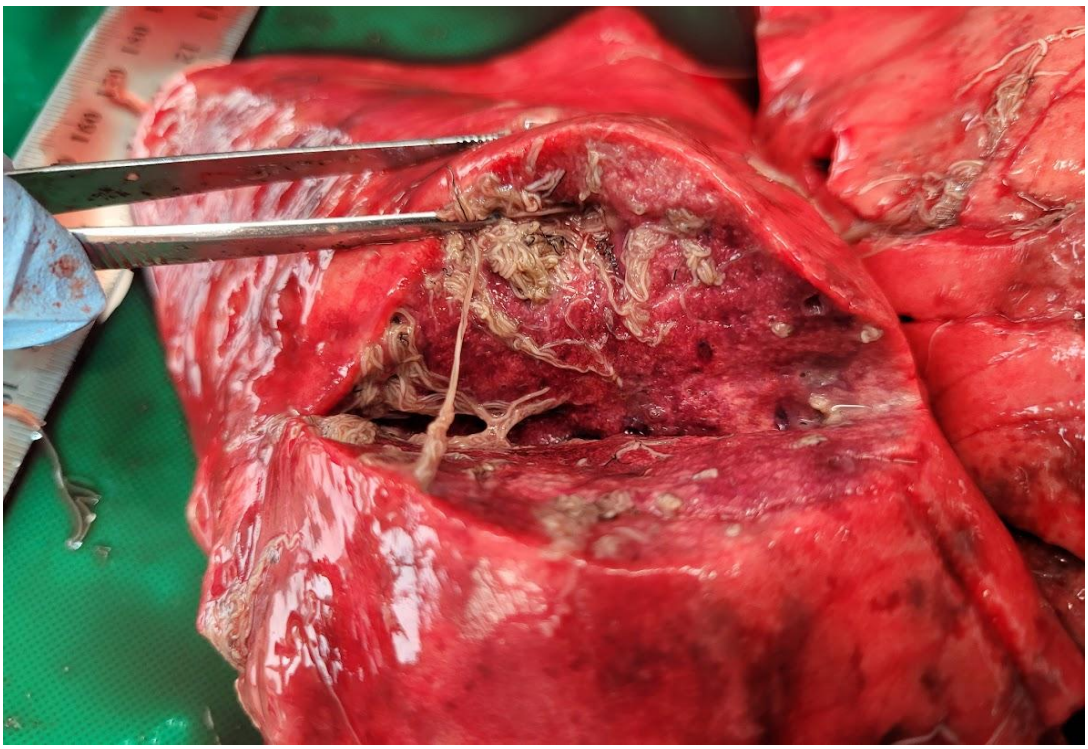


Figure 2.2. Severe *H. lagenorhynchi* exposed from the nodules in the lung parenchyma of KS25-04Dd. Photo: Cetacean Pathology Unit, Massey University.

Molecular Methods

Molecular identification was undertaken using two genetic markers: the nuclear small-subunit ribosomal RNA gene (18S rRNA) and the mitochondrial cytochrome c oxidase subunit I gene (CO1). Genomic DNA was extracted from nematode samples using the DNeasy® Blood & Tissue Kit (Qiagen, Hilden, Germany) according to the manufacturer's protocol. Partial fragments of the 18S gene were amplified and sequenced using primers Nem18SF and Nem18SR (Wood et al., 2013), and partial fragments of the CO1 gene using primers JB3 and JB4.5 (Bowles et al., 1992). Resulting sequences were compared with sequences in the NCBI GenBank database using BLAST (Carta & Wick, 2018). The 18S rRNA gene was used as the primary marker for species-level identification because of its high amplification success and conserved sequence characteristics. Pairwise sequence divergence among CO1 sequences was calculated in MEGA version 6 (Tamura et al., 2013) to assess genetic relatedness among specimens and to determine whether they were likely to represent the same or different species. Final taxonomic determinations were based on combined molecular and morphological evidence. Species were assigned when 18S sequences produced clear matches and morphological characteristics were consistent with published descriptions. Where molecular data were absent or ambiguous, morphology was used as the primary criterion. Specimens for which both molecular and morphological information were insufficient were classified as unidentified.

2.2.2 Pneumonia

Pneumonia was classified semi-quantitatively based on two parameters: the histologic severity of inflammation and the gross extent of lung involvement. Histopathology confirmed the presence and inflammatory pattern of pneumonia and was used to grade its severity. All histologic assessments were made by board-certified veterinary pathologists at Awanui Veterinary, including Dr Cathy Harvey and Dr Emma Gulliver, and I extracted the findings from the reports. I assessed the distribution of consolidation from necropsy photographs and necropsy reports (Section 2.2.2B). The histopathological findings recorded for each animal are listed in Appendix Table A.3.

A. Severity of Pneumonia

Pneumonia severity was graded as mild, moderate or severe by the pathologists who examined each case, and I used the grade as reported. The features characterising each grade - the degree and cellular composition of inflammatory exudation, the extent of alveolar and interstitial involvement, the presence or absence of necrosis or consolidation and the preservation of lung architecture, are summarised in Table 2.3, based on Passmore et al. (2018), Siebert et al. (2020) and Venn-Watson et al. (2015). Mild pneumonia displayed only small amounts of inflammatory cells or fluid in alveoli or bronchioles, minimal septal thickening, and preserved lung architecture, with consolidation absent or minimal. Moderate pneumonia exhibited a clear accumulation of inflammatory material in air spaces, noticeable thickening of alveolar septa, and involvement of larger areas of parenchyma, with consolidation present but architecture still partially preserved. Severe pneumonia displayed air spaces densely filled with inflammatory exudate, areas of fibrin deposition, necrotic debris, marked architectural distortion, and extensive consolidation replacing large regions of parenchyma.

B. Distribution of pneumonia

Pneumonia was identified from the histopathology reports (Section 2.2.2A), and its distribution was then assessed grossly based on the pattern and extent of pulmonary consolidation in both lungs. I classified the distribution for each animal from the necropsy photographs of both lungs, using the criteria in Table 2.4. Where the necropsy report described the distribution of lung changes, such as "patchy" or "throughout", or described changes that I interpreted as consolidation, I used these descriptions together with the photographs to assign the final category, and these descriptions took precedence over the photographs where the two differed. Necropsy descriptions were considered only in animals in which histopathology confirmed pneumonia. Classification from photographs does not allow palpation of the lung, which is addressed in Section 4.5. Focal distribution required one to two discrete foci of consolidation, together affecting no more than 10% of the total lung volume, separated by grossly normal parenchyma. Multifocal distribution was assigned when there were three or more discrete foci, or when consolidation affected more than 10% and up to 75% of the lung volume, with grossly normal parenchyma persisting between lesions.

Diffuse distribution was assigned when consolidation was coalescing or contiguous and affected more than 75% of the lung volume, with little or no intervening normal parenchyma. Where the criteria for number of foci and extent pointed to different categories, the more extensive category was assigned.

Table 2.3. Definitions of pneumonia severity applied in this study

Severity	Definition
Mild	Consolidation absent or minimal; small amounts of inflammatory cells or fluid within alveoli or bronchioles; alveolar septa mildly thickened; overall lung architecture remained largely preserved.
Moderate	Consolidation present; accumulation of inflammatory material in air spaces; thickening of alveolar septa; involvement of larger areas of parenchyma; architecture partially preserved.
Severe	Extensive consolidation; air spaces densely filled with inflammatory exudate and fibrin; necrotic debris, marked architectural distortion.

Table 2.4. Definitions of pneumonia distribution applied in this study.

Distribution	Definition
Focal	1 to 2 discrete foci of consolidation, together affecting ≤10% of the total lung volume, separated by grossly normal parenchyma
Multifocal	≥3 discrete foci of consolidation, or consolidation affecting >10% to ≤75% of the total lung volume, with grossly normal parenchyma between lesions
Diffuse	coalescing or contiguous consolidation affecting >75% of the total lung volume, with little or no intervening normal parenchyma

2.2.3 Structural Tissue Damage

Gross and histological examinations were used to identify additional pulmonary lesions from chronic or severe infections. Necrosis was recorded when coagulative or liquefactive necrosis was present, characterised by loss of normal alveolar architecture and accumulation of cellular debris. Fibrosis was identified by dense collagen replacing normal alveolar tissue, showing chronic reparative change. Mineralisation was noted when calcification was visible grossly or confirmed histologically in the affected regions of parenchyma (Figure 2.3b-d).

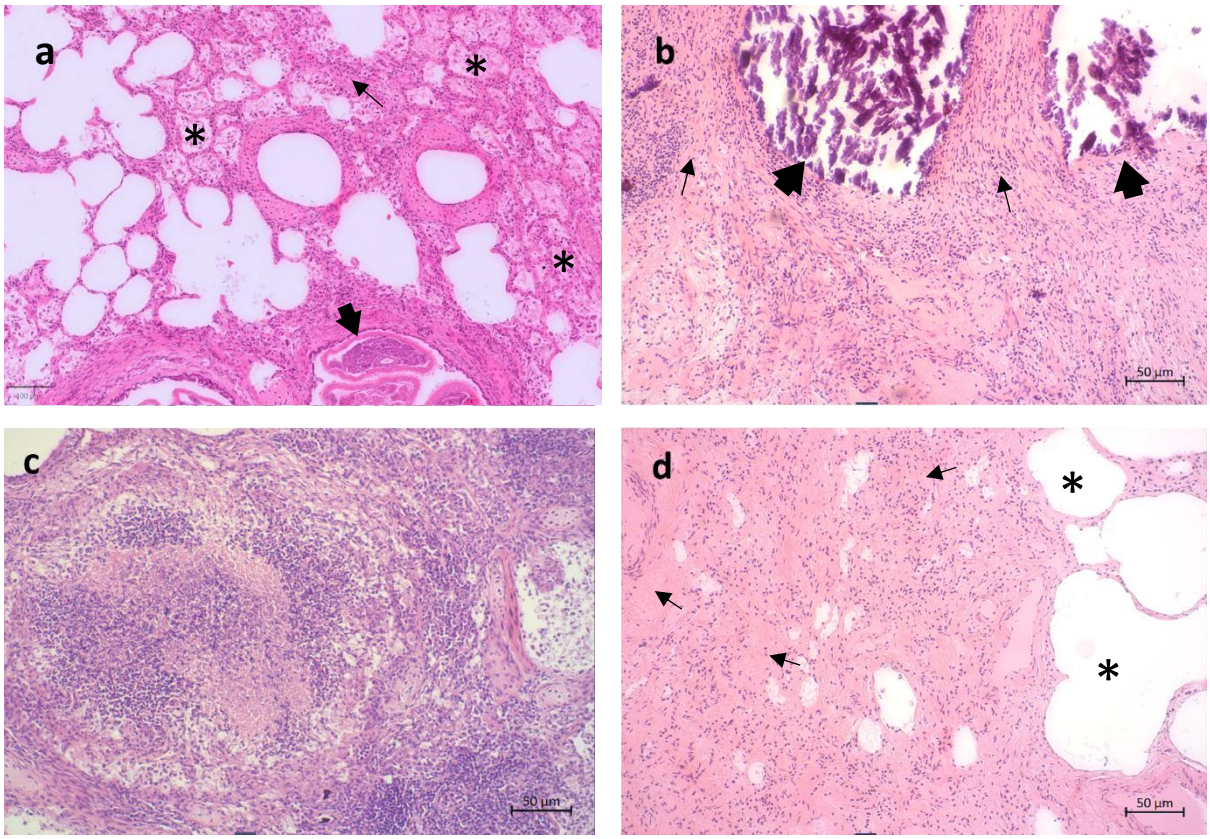


Figure 2.3. Representative H&E-stained lung sections from *Delphinus delphis*. (a) Moderate verminous pneumonia with alveoli filled with inflammatory cells and foamy material (*), thickened alveolar septa (*arrow*), and a nematode in a bronchiole (*arrowhead*; KS14-62Dd; scale bar 100 µm). (b) Extensive, fragmented basophilic mineralisation (*arrowhead*) within dense fibrotic tissue (*arrow*) (KS22-53Dd; scale bar 50 µm). (c) Focal necrosis with complete loss of cellular architecture (KS19-18Dd; scale bar 50 µm). (d) Marked fibrosis with dense collagen deposition (*arrows*), fibroblast proliferation and sparse chronic inflammatory cells adjacent to expanded alveolar spaces (*) (KS22-53Dd; scale bar 50 µm).

2.3 Lung Health Scoring System

I calculated all lung health scores from the parasitism, pneumonia and tissue damage categories described above. The lung health scoring system applied in this study was adapted from Schick et al. (2024) via quantified scores of parasitism, pneumonia, and irreparable tissue damage. Severity of parasitism (Table 2.2) and pneumonia (Table 2.3) was categorised as mild, moderate, or severe and assigned 1, 2, or 3 points respectively, with a maximum score of 3 points for each category (Table 2.5).

In the original scoring system of Schick et al. (2024), diffuse consolidation was given 2 points, so that pneumonia scores ranged from 0 to 6. To distinguish diffuse from multifocal

involvement, which differ in the extent of lung affected, the distribution score was modified in this study: focal consolidation was given 1-point, multifocal consolidation 2 points, and diffuse consolidation 3 points (Table 2.4). The pneumonia score was calculated by multiplying the severity score (1 to 3) by the distribution score (1 to 3), yielding a final score of 1 to 9. Higher scores indicated more severe inflammation and greater lung involvement. The presence of irreparable tissue damage (i.e., necrosis, fibrosis, and mineralisation) was given 1 point each, with a maximum of 3 points assigned. By summing the scores for parasitism, pneumonia, and irreparable tissue damage, the maximum possible lung score per animal was 15 (Table 2.5).

Table 2.5. Lung health grade with associated parasitism, pneumonia and irreparable tissue damage point allocations shown.

	Parasitism	**Pneumonia**		**Irreparable Tissue Damage**
		Severity	**Distribution**	
Mild	1 pt	1 pt	x1 if focal, x2 if multifocal, x3 if diffuse	1 pt for each of fibrosis, mineralisation, and necrosis
Moderate	2 pts	2 pts		
Severe	3 pts	3 pts		
TOTAL	**Max 3 pts**	**Max 9 pts**		**Max 3 pts**

For example, if an animal has a moderate parasitism (2 pts), moderate pneumonia (2 pts) that is multifocal in distribution (2 pts), and has fibrosis in the lung tissue (1 pt), the calculation of the score is as follows:

Parasitism	*=*	*2*
+		
Pneumonia (severity x distribution)	*=*	*2 x 2*
+		
Irreparable Tissue Damage	*=*	*1*
TOTAL	***=***	***7***

The lung scores were assigned a grade to indicate the degree of lung compromise. Schick et al. (2024) used grades of mild (1–3), moderate (4–8) and severe (9–12) for a maximum score

of 12. Because the maximum possible score in this study was 15, the scoring range was divided into three equal bands: lung scores of 1 to 5 were graded as mild, 6 to 10 as moderate, and 11 to 15 as severe. A lung score of 0 indicated that the lung showed no evidence of pathological change is referred to as normal. Because the distribution scores and grade bands differ from those of Schick et al. (2024), lung health scores in this study are not directly comparable with theirs.

Table 2.6. Lung Health Grade (LHG) with equivalent total lung score.

Normal	Mild	Moderate	Severe
0 points	1-5 points	6-10 points	11-15 points

Oedema, congestion, haemorrhage, atelectasis, and emphysema, as described in Sections 1.7.1 to 1.7.3, were recorded as additional lesions to provide context and support morphological diagnoses. However, these lesions were not included in the pneumonia severity score because they are non-specific, often agonal, and do not reliably reflect inflammatory response.

2.4 Characterisation of Pneumonia

Pneumonia was characterised based on histomorphological features following Caswell and Williams (2016) and according to the predominant aetiologic pattern, including parasitic, bacterial, viral, fungal, multifactorial, or non-specific inflammation. Bacterial pneumonias typically showed bronchopneumonia or fibrinous patterns with neutrophilic exudation and, when present, intralesional bacteria. Viral pneumonias were suspected when interstitial inflammation, diffuse alveolar damage, syncytia, or viral inclusions were observed. Parasitic pneumonias were identified by the presence of lungworms or larval stages accompanied by eosinophilic, or granulomatous inflammatory responses (Figure 2.3a). Because parasites in chronic lesions are often degraded or absent, pneumonia was also classified as verminous when histology showed granulomatous or chronic inflammation, with eosinophils and mononuclear cells, around necrotic or mineralised foci, even if no parasite was identified (Measures, 2001; Rodrigues et al., 2018). In these cases, the lungworm severity grade (Section 2.2.1) was recorded as none, because that grade requires parasites to be observed. Fungal

pneumonias were characterised by pyogranulomatous inflammation with multinucleated giant cells and identifiable fungal elements. Cases exhibiting overlapping features were classified as multifactorial, while those lacking identifiable organisms or specific aetiological indicators were categorised as non-specific. Detailed diagnostic criteria for each category are summarised in Table 2.7.

Table 2.7. Histopathological characterisation of pneumonia based on aetiology.

Aetiology	Histopathological Features
Bacterial	Bronchopneumonia: Neutrophil-rich exudate in bronchioles & alveoli. Suppurative/fibrinous inflammation abscesses possible. Necrosis of the airway epithelium, fibrinous pleuritis
Viral	Interstitial or broncho-interstitial pneumonia: Thickened alveolar septa with lymphocytes, plasma cells, and macrophages. Type II pneumocyte hyperplasia. Syncytial cells, viral inclusion bodies
Parasitic (Verminous)	Bronchopneumonia or granulomatous pneumonia: Eosinophilic and mononuclear infiltrates. Fibrosis, mineralisation, emphysema, atelectasis. Parasites or larvae may be visible.
Fungal	Granulomatous or pyogranulomatous pneumonia: Mixed macrophages, neutrophils, and multinucleated giant cells. Necrosis, mineralisation possible. Fungal hyphae or yeast forms within lesions
Multifactorial	Overlapping patterns and/or evidence of multiple agents.
Non-specific	Inflammatory changes without an identifiable associated agent. May resemble interstitial or bronchopneumonia

2.5 Statistical Analysis

All analyses were performed in R (version 4.4.3; R Core Team, 2025) using RStudio (version 2026.01.1, Build 403). Descriptive statistics (mean, standard deviation, median and range) were calculated for age and lung health scores. The prevalence and severity of lung health grades, pneumonia and lung parasitism were summarised using counts and percentages within sex, age class, BCS and animal source. Pneumonia prevalence and lung health scores were calculated for the 122 dolphins in the lung health dataset, and lungworm prevalence for the 141 dolphins in the parasitism dataset (Section 2.1.5). Associations

between categorical variables (lung health grade and parasitism severity against sex, age class, BCS, and source) were tested using Fisher's exact test, because expected frequencies were below 5 in several cells (e.g., 6 of 12 cells for lung health grade by BCS). Tests were two-sided, with significance set at $\alpha = 0.05$. Animals with no data (ND) for a variable were excluded from analyses involving that variable (for example, four animals without a BCS were excluded from the BCS analyses).

Chapter 3. Results

3.1. Sample Summary

A total of 122 common dolphins were assessed postmortem for lung health between 1999 and 2025. Dolphins originated from single strandings (n=78), mass strandings (n=30) and bycatch (n=14). Sex balance was relatively equal, comprising 45.9% (n=56) males and 54.1% (n=66) females. Age ranged from 0 to 31 years (mean = 7.77 years; SD = 7.05; median = 7 years), with most individuals (70%, n=86) aged 12 years or younger (Figure. 3.1).

Age classes included 14% (n=17) perinates, 41% (n=50) immature, and 45% (n=55) sexually mature. Most individuals exhibited a good (BCS 1) to moderate (BCS 2) body condition score – 61.9% (n=73) BCS 1, 30.5% (n=36) BCS 2, and 7.6% (n=9) BCS 3. Four individuals did not have a BCS recorded.

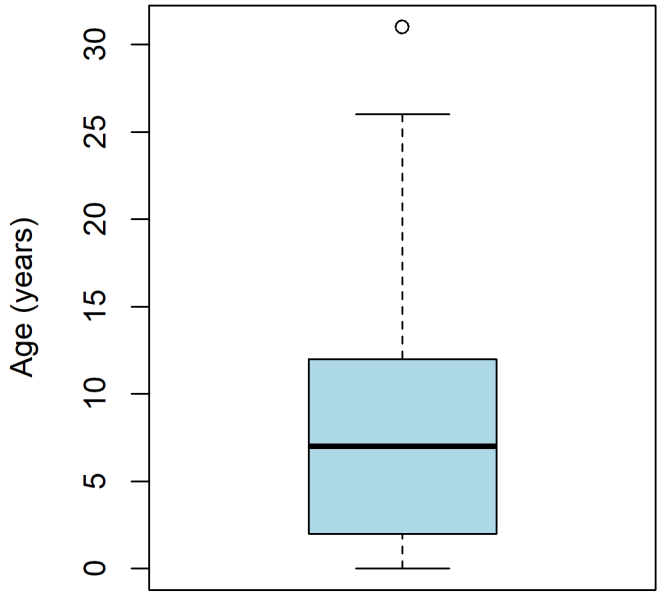


Figure 3.1. Age distribution of common dolphins examined between 1999 and 2025, with ages ranging from 0 to 31 years (median =7 years, SD = 7.05).

3.2 Lung Health Scores and Grades

Lung health scores ranged from 0 to 13, with a mean of 2.8 (±3.3) and a median of 2. Using the predefined LHG equivalent, 44.3% (n=54) of the animals recorded normal lungs. Among the 68 animals with compromised lungs, 36.1% (n=44) were scored as mild, 17.2% (n=21) as moderate (17.2%, n=21), and only a few as severe (2.5%, n=3) (Table 3.1 and Figure 3.2).

Table 3.1. Lung health grade with corresponding scores and observed counts.

LHG	Score Range	n	%
Normal	0	54	44.3
Mild	1-5	44	36.1
Moderate	6-10	21	17.2
Severe	11-15	3	2.5
Total	0-15	122	100

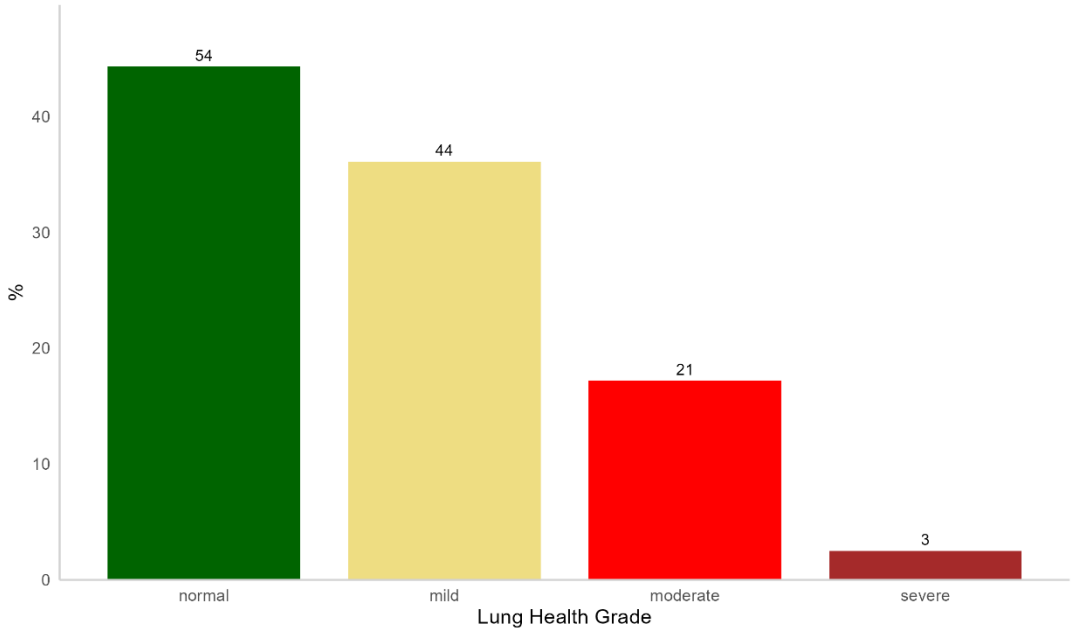


Figure 3.2. Distribution of lung health grades among examined animals (n=122). The x-axis displays four ordered categories (normal, mild, moderate, severe). The y-axis shows the proportion of animals in each category.

3.2.1 Lung Health Grade by Sex

Both males and females exhibited comparable proportions of normal, mild, moderate and severe LHG. The absence of statistically significant difference (Fisher's exact test, p = 1.0) provides no evidence of a difference in lung health between sexes (Table 3.2 and Figure 3.3).

Table 3.2. Percentages and counts of animals in each lung health grade per sex. Association tested with Fisher's exact test (p = 1.0).

LHG	Male % (n)	Female % (n)
Normal	44.6 (25)	43.9 (29)
Mild	35.7 (20)	36.4 (24)
Moderate	17.9 (10)	16.7 (11)
Severe	1.8 (1)	3.0 (2)

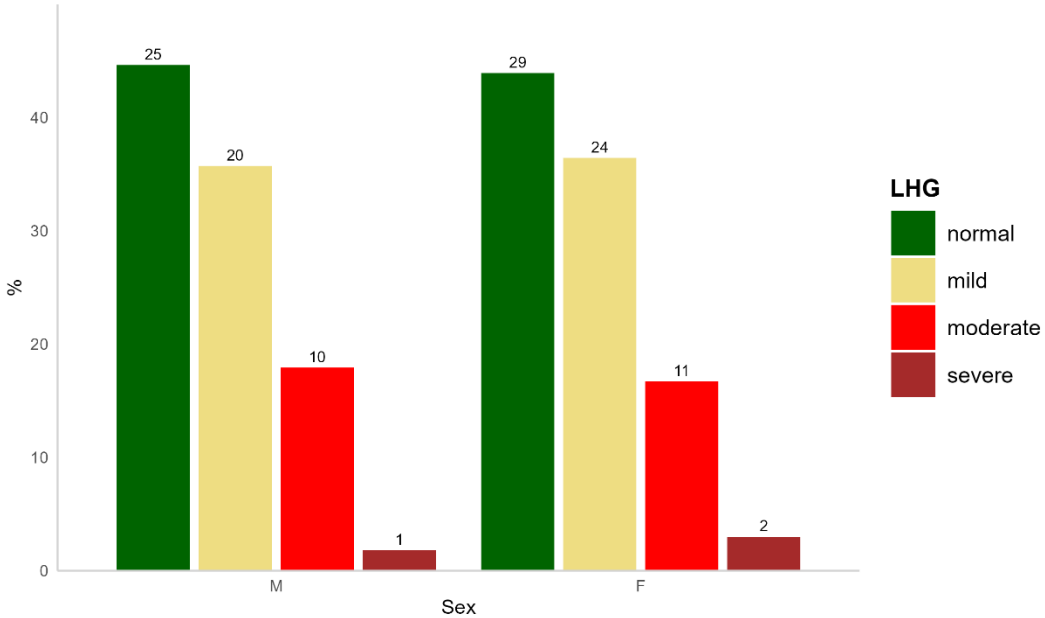


Figure 3.3. The distribution of lung health grades by sex. The x-axis displays four ordered categories (normal, mild, moderate, severe). The y-axis shows the proportion of animals in each category.

3.2.2 Lung Health Grade by Age Class

LHG differed significantly by age class (Fisher's exact test, p = 0.012). Perinates (n=17) recorded the highest proportion of moderate and severe grades, with 41.2% (n=7) classified as moderate and 11.8% (n=2) as severe, while only 35.3% (n=6) had normal lungs. Conversely, immature individuals displayed no (48.0%, n=24) or only mild pathological change (38.0%, n=19), with 14.0% (n=7) showing combined moderate and severe grades. Sexually mature individuals displayed the highest proportion of mild (41.8%, n=23) and the lowest proportion of severe (0) grades (Table 3.3 and Figure 3.4).

Table 3.3. Percentages and counts of animals in each lung health grade per age class. Association tested with Fisher's exact test (p = 0.012).

Severity	Perinates % (n)	Immature % (n)	Sexually Mature % (n)
Normal	35.3 (6)	48.0 (24)	43.6 (24)
Mild	11.8 (2)	38.0 (19)	41.8 (23)
Moderate	41.2 (7)	12.0 (6)	14.5 (8)
Severe	11.8 (2)	2.0 (1)	0

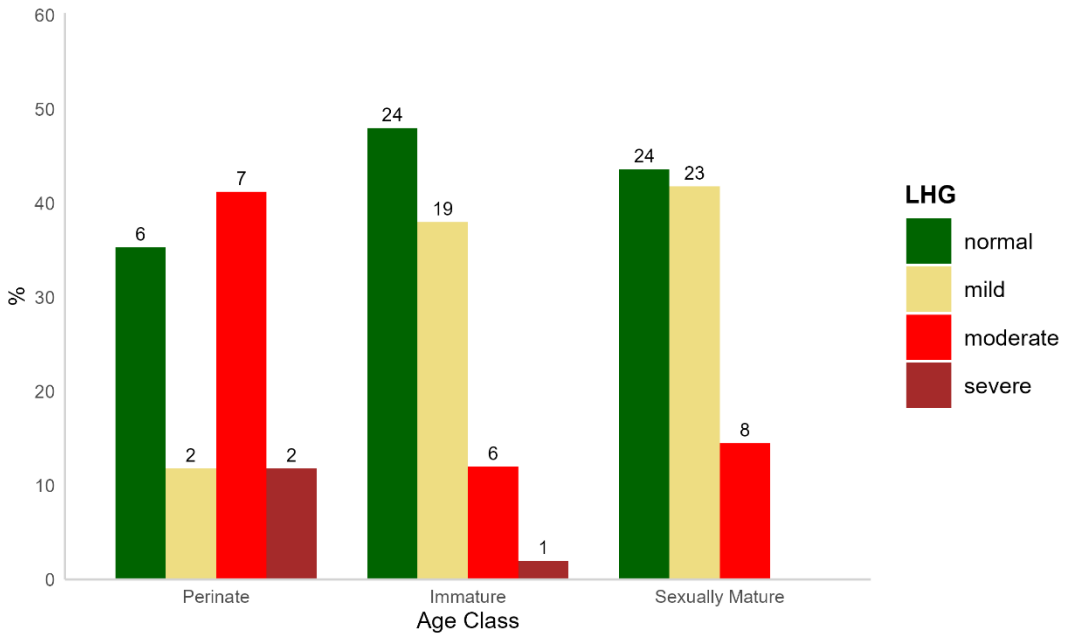


Figure 3.4. Distribution of lung health grades by age class. The x-axis displays four ordered categories (normal, mild, moderate, severe). The y-axis shows the proportion of animals in each category.

3.2.3 Lung Health Grade by BCS

The pattern of LHG by BCS suggests an inverse relationship where the proportion of animals with normal lungs increased and proportion of animals with mild, moderate and severe changes decreased as BCS declined (Table 3.4 and Figure 3.5). However, Fisher's exact test (p = 0.481) indicates that this trend is not statistically significant.

Table 3.4. Percentages and counts of animals by lung health grade across different body condition score (BCS). Association tested with Fisher's exact test (p = 0.481).

Severity	BCS 1 % (n)	BCS 2 % (n)	BCS 3 % (n)
Normal	35.6 (26)	52.8 (19)	66.7 (6)
Mild	42.5 (31)	27.8 (10)	22.2 (2)
Moderate	19.2 (14)	16.7 (6)	11.1 (1)
Severe	2.7 (2)	2.8 (1)	0

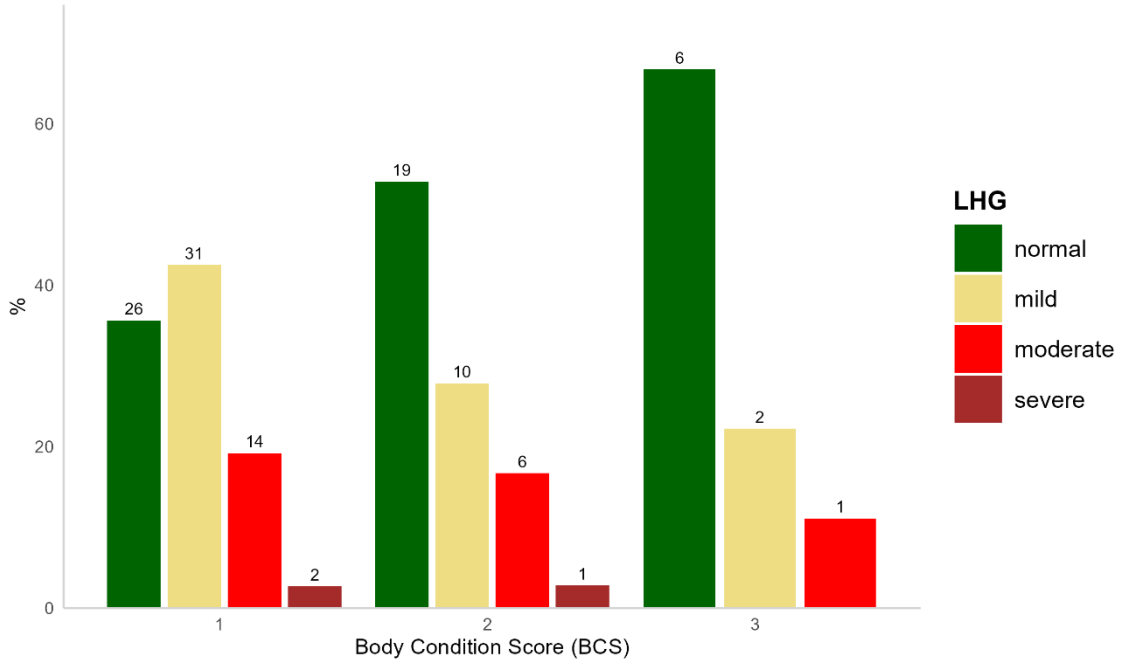


Figure 3.5. Distribution of lung health grades by BCS. The x-axis displays four ordered categories (normal, mild, moderate, severe). The y-axis shows the proportion of animals in each category.

3.2.4 Lung Health Grade by Animal Source

Lung health grade was not significantly associated with source (Fisher's exact test, p = 0.474). Normal lungs were found in 50.0% of bycaught dolphins, 48.7% of single strandings and 30.0% of mass strandings. Moderate or severe lesions were most frequent in mass strandings (30.0%), followed by single strandings (17.9%) and bycaught dolphins (7.1%, all moderate) (Table 3.5 and Figure 3.6).

Table 3.5. Percentages and counts of animals by lung health grade across different animal sources. Association tested with Fisher's exact test (p = 0.474).

Severity	Single % (n)	Mass % (n)	Bycatch % (n)
Normal	48.7 (38)	30.0 (9)	50.0 (7)
Mild	33.3 (26)	40.0 (12)	42.9 (6)
Moderate	15.4 (12)	26.7 (8)	7.1 (1)
Severe	2.6 (2)	3.3 (1)	0

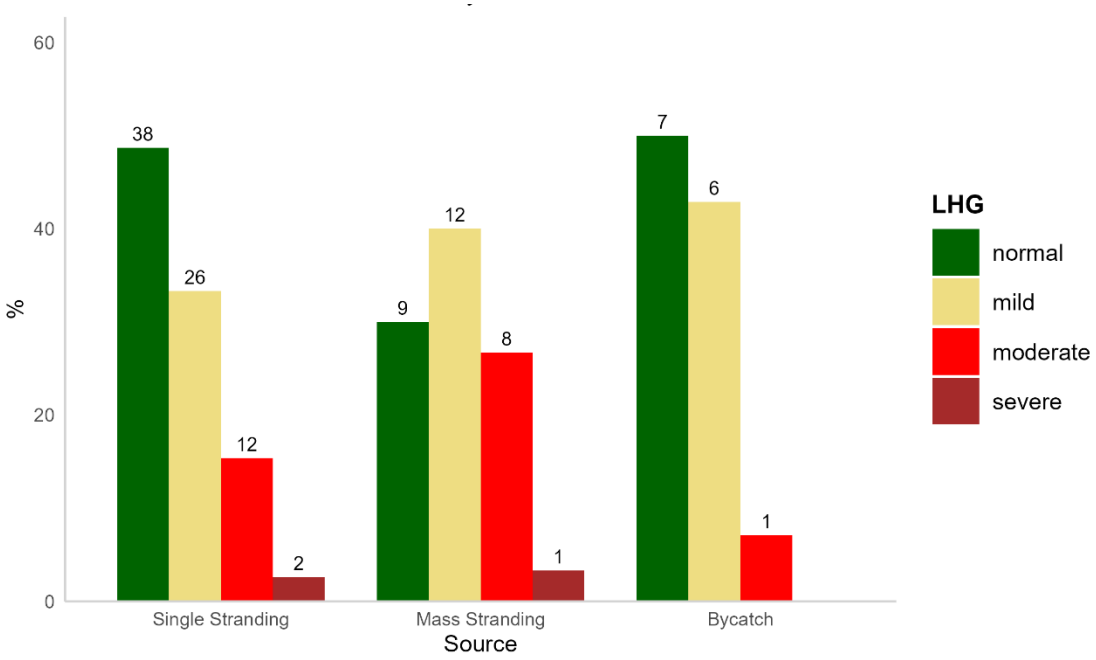


Figure 3.6. Distribution of lung health grades by animal source. The x-axis displays the animal source categories. The y-axis shows the proportion of animals in each category.

3.3 Prevalence and Aetiology of Pneumonia

Approximately half (51.6%, n=63) of the examined animals had pneumonia, with 58.7% (n=37), 22.2% (n=14), and 19.0% (n=12) classified as mild, moderate, and severe, respectively. Of these pneumonic cases, 69.8% (n=44) were attributed to parasitism, 7.9% (n=5) to bacterial infection, 4.8% (n=3) multifactorial (parasitic and bacterial infection), while 17.5% (n=11) were considered non-specific.

Table 3.6. Pneumonia prevalence by severity and aetiology (n=122).

Pneumonia Measure	Category	n	%
Prevalence (n=122)	None	59	**48.4**
	Present	63	**51.6**
Severity (n=63)	Mild	37	**58.7**
	Moderate	14	**22.2**
	Severe	12	**19.0**
Aetiology (n=63)	Parasitism	44	**69.8**
	Bacterial infection	5	**7.9**
	Multifactorial (parasitic + bacterial)	3	**4.8**
	Non-specific	11	**17.5**

Note: Severity and aetiology of pneumonia percentages are reported as the percentage of animals diagnosed with pneumonia (n=63).

3.4 Prevalence and Severity of Lung Parasitism

Of the 141 animals assessed for lung parasitism, only 20.6% (n=29) recorded parasitism (Table 3.7). Of these, all parasites identified were nematodes, with no cestodes identified (Table 3.8). Lungworm infections were mostly mild (10.6%; n=15), with moderate and severe infections accounting for only 6.4% (n=9) and 3.5% (n=5), respectively. Identified lungworm species included *Halocercus lagenorhynchi* (n=5), *Stenurus ovatus* (n=1), and *Halocercus* sp. (n=1).

Table 3.7. Lungworm prevalence and severity in common dolphins (n=141).

Category	Count (n)	%
None	112	79.4
Present	29	20.6
Mild	*15*	*10.6*
Moderate	*9*	*6.4*
Severe	*5*	*3.5*

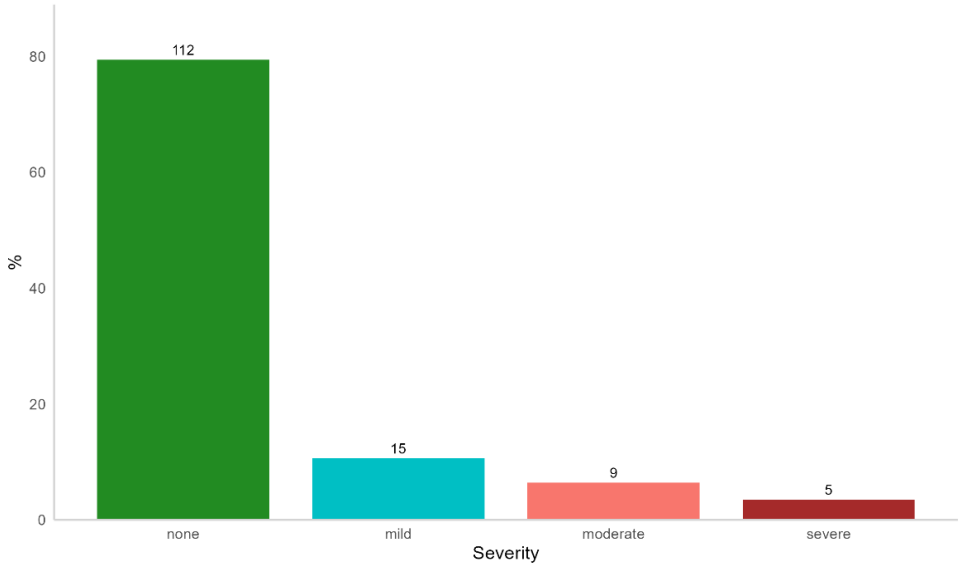


Figure 3.7. Distribution of lung parasitism among examined animals. The x-axis displays four ordered categories (none, mild, moderate, severe). The y-axis shows the proportion of animals in each category.

Table 3.8. Lungworm species identification based on morphological characteristics (Baylis and Daubney, 1925) and DNA sequencing.

Animal ID	Morphological identification	DNA identification
KS07-13Dd	Unknown	Unknown
KS14-62Dd	Unknown	Likely *Halocercus* sp.
KS19-18Dd	*Halocercus lagenorhynchi*	*Halocercus lagenorhynchi*
KS22-53Dd	*Stenurus ovatus*	Unknown
KS22-77Dd	*Halocercus lagenorhynchi*	*Halocercus lagenorhynchi*
KS23-04Dd	*Halocercus lagenorhynchi*	*Halocercus lagenorhynchi*
KS23-06Dd	*Halocercus lagenorhynchi*	*Halocercus lagenorhynchi*
KS23-58Dd	*Halocercus lagenorhynchi*	Unknown
KS25-11Dd	Unknown	Unknown

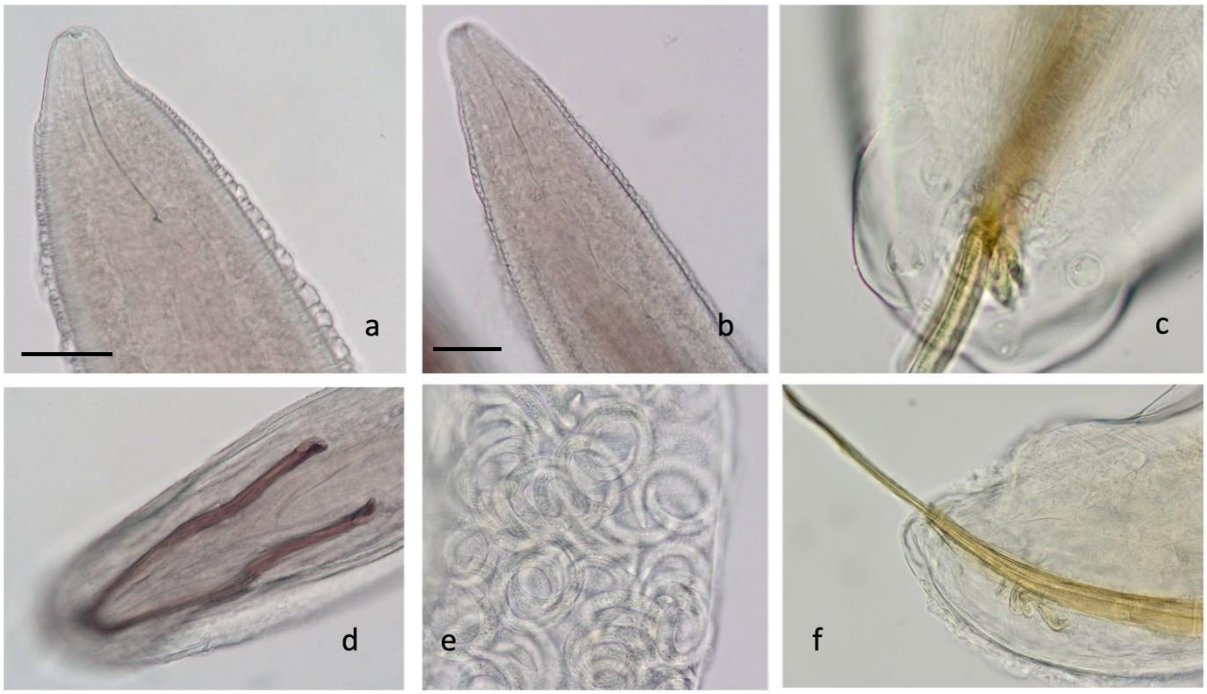


Figure 3.8. Light micrographs of *H. lagenorhynchi*. a) Female anterior end, b) male anterior end, c) male bursal membrane and rays, d) male spicule, e) larvae in utero KS23-04Dd, f) spicule showing folded gubernaculum. A) and b) scale bars = 100µm. Photo: Bronwen Presswell.

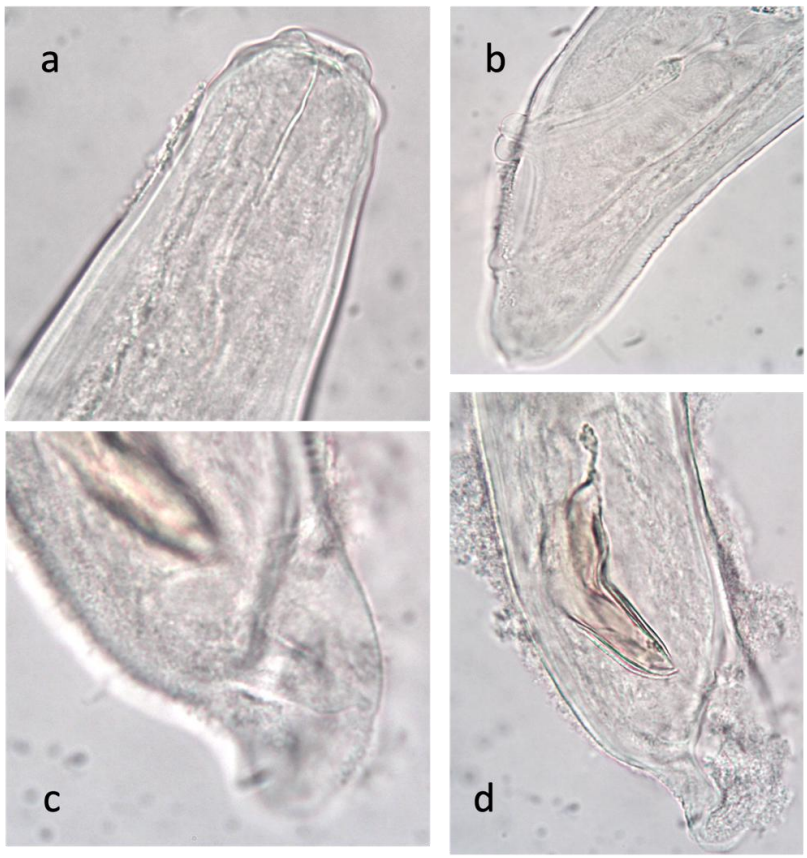


Figure 3.9. Light micrographs of *Stenurus ovatus* showing a) anterior end showing papillae, b) female posterior end showing anus and vulva with cuticular swellings, c) male bursa showing thick lateral and dorsal rays, d) spicule and gubernaculum. Photo: Bronwen Presswell.

3.4.1 Lungworm Prevalence and Severity by Sex

The proportion of individuals without evidence of lung parasitism was similar between sexes, at 76.5% (n=52) versus 82.2% (n=60) for males and females, respectively. Those individuals with parasitism demonstrated mostly only mild severity. Parasite severity was not statistically different by sex (Fisher's exact test, p = 0.263), although males displayed a higher proportion of mild (14.7%; n=10) and moderate (7.4%; n=5) infections, whereas females exhibited marginally more severe (5.5%; n=4) infestations (Table 3.9 and Figure 3.10).

Table 3.9. Parasite prevalence and severity by sex. Association tested with Fisher's exact test (p = 0.263).

Severity	Male % (n)	Female % (n)
None	76.5 (52)	82.2 (60)
Mild	14.7 (10)	6.8 (5)
Moderate	7.4 (5)	5.5 (4)
Severe	1.5 (1)	5.5 (4)

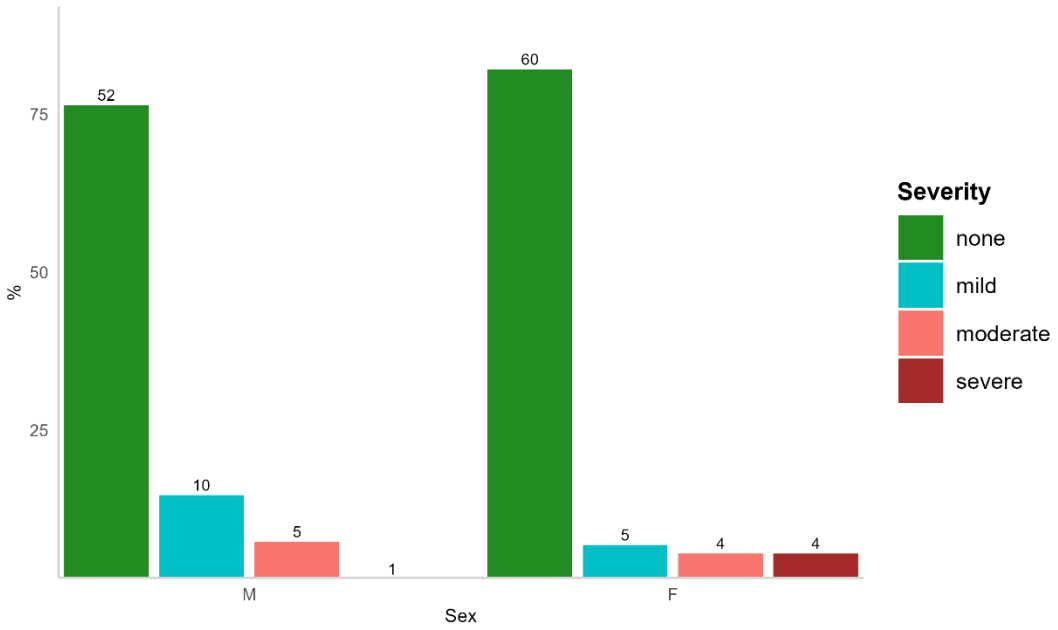


Figure 3.10. Lung parasitism prevalence by severity (normal, mild, moderate, severe) between sexes (n=141). Numbers above bars represent animals represented in each category.

3.4.2 Lungworm Prevalence and Severity by Age Class

Age class demonstrated a significant influence on infection severity (Fisher's exact test, $p < 0.001$), with perinates exhibiting greatest parasite prevalence (40.9%; n=9) and highest rates of moderate and severe lung parasitism, at 18.2% (n=4) and 13.6% (n=3), respectively. Conversely, sexually mature individuals demonstrated the lowest parasite prevalence, with 93.4% (n=57) showing no infection and 6.6% (n=4) have mild infection (Table 3.10).

Table 3.10. Parasite prevalence and severity by age class (Fisher's exact test, $p < 0.001$).

Severity	Perinate % (n)	Immature % (n)	Sexually mature % (n)
None	59.1% (13)	72.4% (42)	93.4% (57)
Mild	9.1% (2)	15.5% (9)	6.6% (4)
Moderate	18.2% (4)	8.6% (5)	0
Severe	13.6% (3)	3.4% (2)	0

3.4.3 Lungworm Prevalence and Severity by BCS

Most animals within each body condition grade did not present with lung parasites - BCS 1 at 75.9% (n=60), BCS 2 at 84.4% (n=38), and BCS 3 at 83.3% (n=10). For BCS 1, mild, moderate, and severe parasitism occurred in 11.4% (n=9), 7.6% (n=6), and 5.1% (n=4) of cases, respectively. For BCS 2, 11.1% (n=5) were mild, and 2.2% (n=1) were both moderate and severe. For BCS 3, one animal had a mild infection, and another had a moderate infection (8.3%), with no severe cases recorded.

No significant association between parasite severity and body condition score was found (Fisher's exact test, $p = 0.842$), despite animals with BCS 1 displaying the highest combined proportion of moderate and severe parasite loads (12.7%, n=10) (Table 3.10 and Figure 3.11).

Table 3.11. Parasite severity by body condition score (BCS). Percentages and counts are shown. Association tested with Fisher's exact test (p = 0.842).

Severity	BCS 1 % (n)	BCS 2 % (n)	BCS 3 % (n)
None	75.9 (60)	84.4 (38)	83.3 (10)
Mild	11.4 (9)	11.1 (5)	8.3 (1)
Moderate	7.6 (6)	2.2 (1)	8.3 (1)
Severe	5.1 (4)	2.2 (1)	0

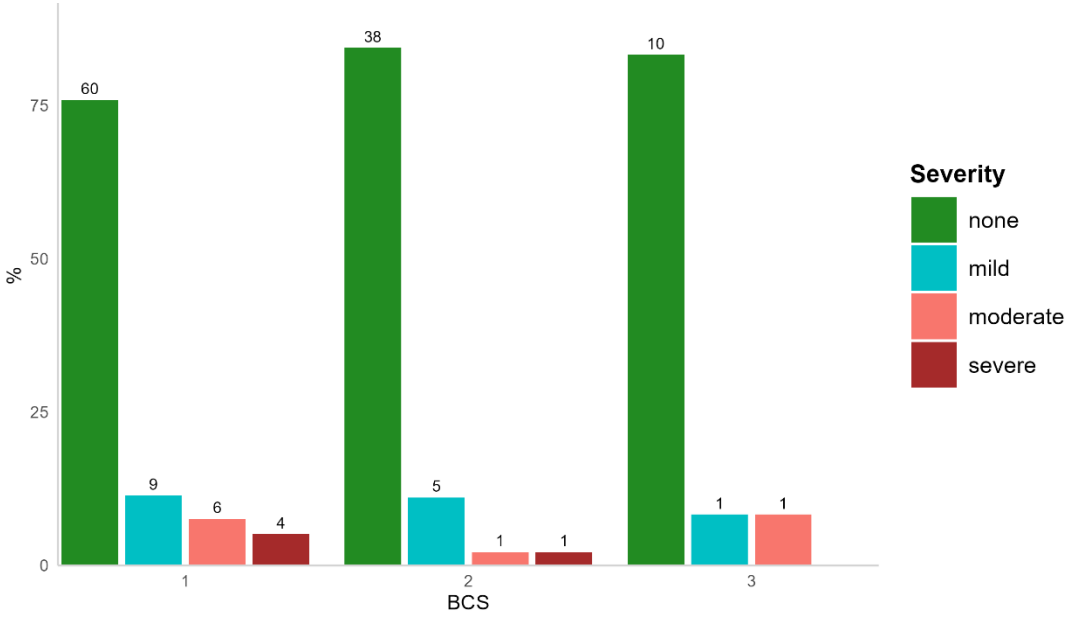


Figure 3.11. Distribution of lung parasitism among the different body condition scores (BCS). The x-axis displays four ordered categories (normal, mild, moderate, severe). The y-axis shows the proportion of animals in each category.

3.4.4 Lungworm Prevalence and Severity by Animal source

Parasitism severity did not differ significantly by source (Fisher's exact test, p = 0.375). No lungworm infection was detected in 80.4% of single strandings, 80.0% of mass strandings and 71.4% of bycaught dolphins. All infections in bycaught dolphins were mild (28.6%), whereas moderate or severe infections occurred only in single strandings (10.3%) and mass strandings (13.3%) (Table 3.12 and Figure 3.12).

Table 3.12. Parasite severity by animal source. Percentages and counts are shown. Association tested with Fisher's exact test (p = 0.375).

Severity	Single % (n)	Mass % (n)	Bycatch % (n)
None	80.4 (78)	80.0 (24)	71.4 (10)
Mild	9.3 (9)	6.7 (2)	28.6 (4)
Moderate	7.2 (7)	6.7 (2)	0
Severe	3.1 (3)	6.7 (2)	0

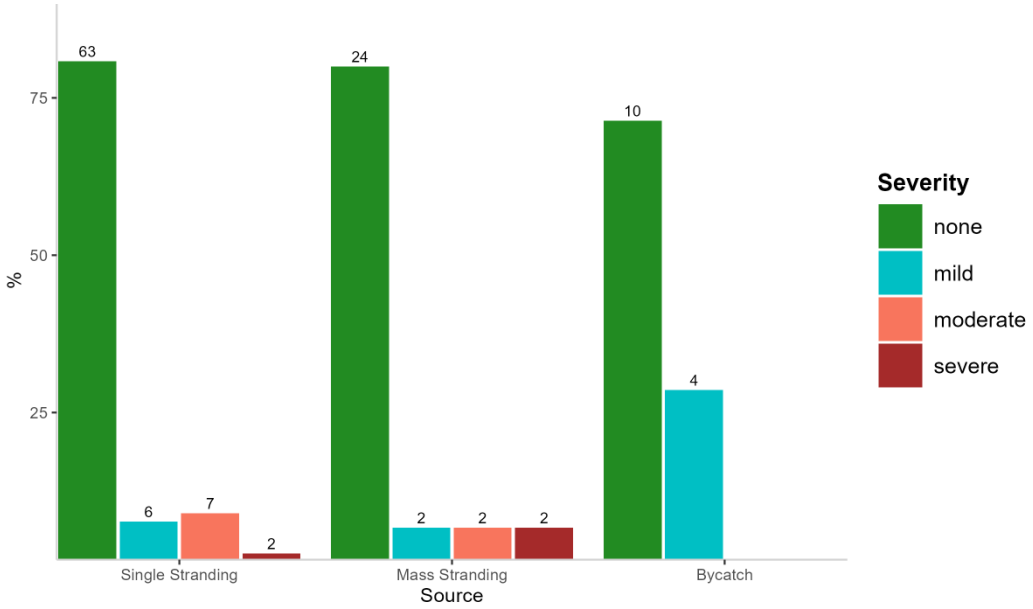


Figure 3.12. Distribution of lung parasitism by animal source. The x-axis displays the animal source categories. The y-axis shows the proportion of animals in each category.

Chapter 4: Discussion

4.1 Lung Health Grade

The finding that over 44% of examined dolphins presented with lungs free of parasitism, inflammation, or irreversible tissue damage, provides a critical epidemiological baseline for understanding respiratory health and disease in New Zealand cetacean populations. This proportion is notably higher than many international surveys of stranded and bycaught cetaceans where abnormal lungs affect 60% to 100% of examined cohorts (Birkun Jr, 2002; Cornaglia et al., 2000; di Guardo et al., 1995; Domiciano et al., 2016; Fauquier et al., 2009; Gonzales-Viera et al., 2011; Jauniaux et al., 2002; Jepson et al., 2000; Neimanis et al., 2022; Rodrigues et al., 2018; Siebert et al., 2020; Siebert et al., 2001; Tomo & Kemper, 2022; Venn-Watson et al., 2015), as well as targeted surveys of pulmonary parasitism (Conlogue et al., 1985; Guimarães et al., 2015; Marigo et al., 2010; Pool et al., 2024; Zafra et al., 2015) and studies investigating acute mortality events or perinatal cohorts (Colegrove et al., 2016; Groch et al., 2020a). Instead, results presented here are more comparable with findings reported from animals under managed care. For example, common bottlenose dolphins of the US Navy Marine Mammal Program, which lived in sea-pens and received frozen-thawed fish, preventive medical care, routine health monitoring, and nutritional supplementation, showed that over 30 years, 50% of necropsied animals had no histopathological evidence of pneumonia, no lungworms, and only low frequencies of permanent tissue damage: fibrosis (9.5%) and mineralisation (7.1%) (Venn-Watson et al., 2012).

Because the lungs are directly exposed to the external environment through the respiratory tract, normal pulmonary status often reflects low pathogen pressure and an absence of major environmental or physiological stressors (e.g. chemical pollutants and chronic disturbance) (Caswell & Williams, 2016; St. Leger et al., 2018). The high proportion of pathologically ‘normal’ lungs in the present study suggests that the New Zealand common dolphin population has not experienced widespread infectious respiratory outbreaks or epizootics that frequently decimate other wild cetacean populations (Birkun Jr, 2002; Colegrove et al., 2016; Domingo et al., 1992). Instead, the low rate of severe pulmonary lesions aligns more

closely with health profiles reported in managed or low-stress populations (Venn-Watson et al., 2012).

The age distribution of the examined dolphins also provides important context for interpreting these results. With a mean and median age of 7.77 ± 7.05 and 7 years, respectively, most of the animals in my dataset were subadults and young adults (Stockin et al., 2009). While immature cetaceans are often vulnerable to acute respiratory infections following weaning and decay of maternal antibodies (Cortese, 2009), most individuals in this study displayed either pathologically normal lungs or only mild changes. This suggests that these immature and sexually mature animals were not heavily exposed to respiratory pathogens or severe anthropogenic stressors such as significant pollution or chronic disturbance that could weaken immune responses and increase susceptibility to infection (Ross, 2002; Siebert et al., 2006; Wunschmann et al., 2001).

An alternate hypothesis is that the high proportion of normal lungs is driven by the immature cohort, which often represents a window of relative pulmonary health post-weaning (Fenton et al., 2017; Measures, 2001; Siebert et al., 2001). However, sexually mature animals had a similar proportion of normal lungs, so age composition alone is unlikely to explain the result.

Among the animals with compromised lungs, most exhibited only mild changes (36.1%), while moderate and severe changes were less common (17.2% and 2.5%, respectively). This predominance of mild pathology is consistent with incidental necropsy findings commonly reported in cetacean lung health surveys and often lacks clinical or functional significance (Fauquier et al., 2009; Geraci & Lounsbury, 2005; Rodrigues et al., 2018; Siebert et al., 2006; Sweeney & Ridgway, 1975). Cetaceans possess a remarkable pulmonary reserve capacity. This enables them to maintain diving performance and metabolic homeostasis despite having focal or low-grade lesions (Fahlman et al., 2017; Piscitelli et al., 2013; Venn-Watson et al., 2012). The mild changes observed in this study likely represent early-stage pathology, partial resolution of previous disease, or chronic low-grade immune responses that do not impair normal lung function (Fauquier et al., 2009; Schick et al., 2020; St. Leger et al., 2018). It is also possible that there are biological host factors, such as age-related immune function and

development, that influence susceptibility and inflammatory response, resulting in limited or focal lesions rather than diffuse and clinically significant disease (Cortese, 2009; Díaz-Delgado et al., 2018; Marsili et al., 2019).

In contrast, the smaller subset of animals displaying moderate and severe lung changes more likely represents animals that experienced clinical respiratory compromise (Clayton et al., 2012; Côté & Silverstein, 2009; Domiciano et al., 2016; Measures, 2001; Nollens et al., 2008; Rodrigues et al., 2018; Schwacke et al., 2014; St. Leger et al., 2018). Lesions of this magnitude can impair gas exchange, reduce lung compliance and make individuals susceptible to secondary complications, making them plausible contributors—or even primary causes—of mortality (Carvallo & Stevenson, 2022; Fahlman et al., 2017; Measures, 2001). This is consistent with regional studies in Australia, Brazil, and the Canary Islands, Spain that showed pulmonary disease, particularly pneumonia, is a major cause of death in several cetacean populations (Díaz-Delgado et al., 2018; Domiciano et al., 2016; Tomo & Kemper, 2022).

Sex

Lung health grades did not significantly differ between sexes, a finding consistent with published reports that showed respiratory lesions in stranded cetaceans occur across species without apparent sex-based susceptibility (Colegrove et al., 2016; Faulkner et al., 1998; Lair et al., 2015; Saldaña et al., 2022; Schwacke et al., 2014; Siebert et al., 2020; Siebert et al., 2001). For example, inflammatory respiratory diseases in common bottlenose dolphins from the Indian River Lagoon, were found to be independent of sex (Durden et al., 2023). Pneumonia was similarly reported as the most common lesion across multiple species in Southern Brazil, without sex-related differences (Domiciano et al., 2016). Even in the US Navy dolphin study, the presence of pneumonia at postmortem did not differ significantly between sexes (Venn-Watson et al., 2012). This pattern is consistent with cetacean respiratory anatomy and physiology, which inherently predisposes all individuals to respiratory infections regardless of sex. An evaluation of inhalation or exhalation air speeds and breath duration in male and female common bottlenose dolphins found no difference between sexes (Gupta et al., 2026), indicating that both sexes share comparable respiratory mechanics and thus.

Age Class

Lung health grade differed significantly among age classes ($p = 0.012$), with perinates disproportionately affected by moderate (41.2%) and severe (11.8%) pulmonary lesions, whereas immature and sexually mature individuals had lower and similar proportions of moderate and severe grades. This pattern is consistent with previous studies suggesting that the perinatal period represents a critical window of susceptibility in cetaceans, when developing immune competence and physiological adaptation to extrauterine life may increase vulnerability to respiratory disease (Cortese, 2009; Fauquier et al., 2009). In addition, the calving process triggers a surge in corticosteroid release in neonates, further reducing the systemic immune response during their first week of life (Cortese, 2009). Prenatal and perinatal factors, such as meconium aspiration (Martinez-Burnes et al., 2019; Rodrigues et al., 2018) and perinatal asphyxia (Alonso-Spilsbury et al., 2005) associated with foetal distress, further exacerbate their vulnerability and can induce diffuse inflammatory lesions that mimic or exacerbate infectious pneumonia (Arbelo et al., 2013; Colegrove et al., 2016; Díaz-Delgado et al., 2018; Fenton et al., 2017; Tomo & Kemper, 2022). In this dataset, however, most pneumonia in perinates was classified as parasitic (7/17) and multifactorial (parasitic and bacterial; 2/17), and lungworm infection was frequent (9/17) and moderate to severe in 7/17, suggesting that parasitic disease contributed to the high grades alongside perinatal factors (Fauquier et al., 2009) (Section 4.3).

Immature and sexually mature individuals had broadly similar lung health with majority of the animals exhibiting pathologically normal lungs or only mild changes. Once the perinatal period has passed, pulmonary health status remains relatively stable across subsequent age classes. Progressive immune maturation in immature animals and acquired immunity in sexually mature group enable individuals to mount faster, effective, and localised inflammatory responses, sequestering focal pathogens and preventing widespread lesions (Caswell & Williams, 2016; Cortese, 2009; Lair et al., 2015; Nussey et al., 2014; van Elk et al., 2019; Zafra et al., 2015). The significant association between lung health grade and age class, therefore, reflects age-dependent pathogenesis, in which immunologically naïve perinates are more susceptible to acute and severe pulmonary diseases due to physiological immaturity (Basha

et al., 2014; Fenton et al., 2017), whereas the older groups show age-related resilience that can tolerate low-grade pathology without a decline in overall lung health (Zafra et al., 2015).

Body Condition Score

The absence of a statistically significant association between lung health and BCS in this study is consistent with prior findings demonstrating that nutritional status is an unreliable predictor of lung health or proximate cause of death in stranded and bycaught cetaceans (Domiciano et al., 2016; Siebert et al., 2020; van Elk et al., 2019). For example, Arbelo et al. (2013) identified pneumonia as a major finding in both dolphins with significant loss of nutritional status and those with good nutritional status. In several studies of harbour porpoise from the Baltic Sea, researchers explicitly noted that the severity of pulmonary lesions was not reflected by changes in nutritional status, and significant findings such as pneumonia and pulmonary nematodiasis frequently occurred in individuals that were otherwise in relatively good nutritional status (Siebert et al., 2020).

Several reasons could explain the lack of association between lung health and BCS. First, the dolphins in this study may have predominantly stranded and died from acute causes (e.g., septicaemia, bycatch-related asphyxia, trauma, or toxin exposure) that do not allow an extended period of reduced food intake and progressive weight loss (Dierauf & Gulland, 2001). Because acute processes act quickly, affected animals often retain fat reserves and may present with pathologically normal lungs at necropsy despite having experienced a fatal systemic event (Díaz-Delgado et al., 2018; Martino et al., 2023; Siebert et al., 2001; van Elk et al., 2019). This hypothesis aligns with Stockin et al. (2009), who reported that 41.2% of mortality in New Zealand common dolphins was related to acute, human-induced causes.

Second, since low-grade or chronic inflammatory changes do not necessarily result in clinical respiratory disease, these animals likely continued to forage until a critical threshold was reached. For example, cetaceans commonly harbour lungworms without causing pulmonary disease and loss of body condition (Pool et al., 2024; Siebert et al., 2020; Siebert et al., 2006). Third, robust individuals can mount localised inflammatory responses that produce histologic lung changes while preserving overall nutritional status (Siebert et al., 2020; Tomo & Kemper,

2022). Fourth, severe malnutrition can reduce both innate and acquired immune activity under energy stress, inhibiting the inflammatory response. Emaciated animals may mount attenuated inflammation, producing mild lesions despite heavy pathogen loads (Acevedo-Whitehouse & Duffus, 2009; Cortese, 2009).

Finally, there is also the likelihood of carcass-recovery bias as sick and debilitated cetaceans that die offshore may sink or be scavenged. Emaciated animals, particularly those with chronic pulmonary disease, are more likely to sink because they have less body fat content and lung inflation (Moore et al., 2020; Williams et al., 2011). These animals are therefore automatically excluded from the data, resulting in an underestimation of population mortality.

In summary, these factors may explain why the proportion of lung normality increases with declining body condition, while the proportions of mild and moderate lung changes decline, and why no statistically significant association between lung health and body condition was detected in this study.

Animal Source

Animal source is an important potential driver of sampling bias in cetacean pathology studies (Domiciano et al., 2016; Neimanis et al., 2022; Tomo & Kemper, 2022). Single-stranded animals often include individuals that were debilitated before death, whereas bycaught dolphins typically represent actively foraging animals that are otherwise healthy (Bernaldo de Quirós et al., 2017; Domiciano et al., 2016; Siebert et al., 2006; Tomo & Kemper, 2022). In this study, LHG did not differ significantly among sources ($p = 0.474$), and the descriptive pattern did not follow this expected gradient. Bycaught dolphins were no more likely than single-stranded animals to have normal lungs (50.0% vs 48.7%), whereas mass strandings had the lowest proportion of normal lungs (30.0%) and the highest prevalence of moderate to severe lesions (30.0%). This finding does not support the expectation that mass strandings mainly involve otherwise healthy, socially cohesive pods that strand inadvertently (Cowan et al., 1986; Geraci & Lounsbury, 2005). It is possible that this reflects shared exposure to contagious respiratory pathogens such as CeMV, which is highly infectious and can spread rapidly through close-knit pods via aerosolised inhalation (Birkun Jr, 2002; St. Leger et al., 2018). The 30 mass-

stranded dolphins came from six events, and animals from the same event are not independent observations, so the apparent excess of pathology in mass strandings could partly reflect a few events. This pattern, however, should be interpreted with caution, as the small sample sizes for mass-stranded and bycaught animals reduce statistical power and limit broader generalisations (Bernaldo de Quirós et al., 2017; Domiciano et al., 2016).

4.2 Pneumonia Prevalence

Half of the common dolphins examined in this study presented with pneumonia. Among the affected animals, most lesions were classified as mild (58.7%), a finding that is often subclinical or incidental, and frequently associated with well-adapted parasitic infections (Guimarães et al., 2015; Lair et al., 2015). Comparable surveys on harbour porpoises have reported variable results. Siebert et al. (2006) reported that most bycaught harbour porpoises from Norwegian and Icelandic waters displayed mild to moderate bronchopneumonia, with severe lesions being rare. In contrast, other European studies reported a predominance of moderate to severe pneumonia as the primary cause of strandings and mortalities (di Guardo et al., 1995; Domingo et al., 1992; Jauniaux et al., 2002; Jepson et al., 2000; Neimanis et al., 2022; Siebert et al., 2020; Siebert et al., 2001; van Elk et al., 2019). These contrasts likely reflect differences in environmental stressors and host susceptibility, since disease patterns in sentinel species often reflect the health of the environment (Domiciano et al., 2016; Neimanis et al., 2022). For example, harbour porpoises from the relatively pristine waters off Greenland showed mostly mild pneumonia cases, whereas their counterparts in the heavily polluted German North Sea exhibited much more severe cases of pneumonia (Wunschmann et al., 2001).

Environmental contamination is a plausible driver of regional differences in disease expression and prevalence. Blubber concentrations of PCBs in New Zealand delphinids are markedly lower than those recorded in Northern Hemisphere populations. Mean blubber concentrations were 19.41 µg/g lw in male and 13.49 µg/g lw in female UK harbour porpoises (Jepson et al., 2016) and 17.55 µg/g lw in Indo-Pacific humpback dolphins (*Sousa chinensis*) in the Pearl River estuary, China (Xie et al., 2021), compared with 1.11 µg/g lw in New Zealand Hector's dolphins (*Cephalorhynchus hectori*) (Casano-Bally, 2023) and up to 1.63 µg/g wet weight in common dolphins (Stockin et al., 2007). This probably reflects New Zealand’s relative

remoteness from major industrial PCB sources (Casano-Bally, 2023; Jones et al., 1999; Stockin et al., 2007).

Numerous studies have shown that chronic exposure to PCBs disrupts immune function in marine mammals, resulting in immunosuppression and increased susceptibility to infectious diseases (de Swart et al., 1996; Desforges et al., 2016; Jepson et al., 2016; Kannan et al., 1993; Lahvis et al., 1995; Ross, 2002; Schwacke et al., 2012). In a controlled feeding experiment, captive harbour seals (*Phoca vitulina*) fed with PCB-contaminated herring (*Clupea harengus*) showed marked immunosuppression compared to controls fed with uncontaminated herring (de Swart et al., 1996; Ross, 2002; Van Loveren et al., 2000). In free ranging cetaceans, long-term pathological investigations of the St. Lawrence Estuary belugas have directly linked high environmental organochlorine contaminants to opportunistic systemic infections and increase in disease related mortality (Lair et al., 2015). Similarly, a UK study of harbour porpoises and common dolphins showed a statistically significant association between high blubber PCB concentrations and mortality due to infectious diseases (Jepson et al., 1999; Williams et al., 2025). Physiological and immunotoxicological effects of PCBs in marine mammals are reported to begin at 9μg/g lw (Kannan et al., 1993), with a threshold of 17-22μg/g lw associated with increased risk of mortality from infectious disease (Jepson et al., 2016; Megson et al., 2021; Williams et al., 2025). Because blubber PCB concentrations in New Zealand dolphins appear to be much lower than the reported immunotoxicological limit, contaminant-induced immunosuppression may have limited influence on the immune competence in this population, although contaminant burdens were not measures in this study.

Aetiology

Parasitism accounted for 70% (44/63) of pneumonia cases reviewed in the current study. This high proportion of verminous aetiology contrasts with several regional reports where bacterial or multifactorial aetiologies predominated (Durden et al., 2023; Gonzales-Viera et al., 2011; Lair et al., 2015; McFee & Lipscomb, 2009; Venn-Watson et al., 2015). However, it is consistent with the broader observation that pulmonary nematodiasis is a major cause of pulmonary pathology in many cetacean populations (Conlogue et al., 1985; Fauquier et al.,

2009; Fenton et al., 2017; Fischbach & Seguel, 2023; Measures, 2001; Suárez-González et al., 2024; Tomo & Kemper, 2022). Siebert et al. (2001) reported that two-thirds of the harbour porpoises with pneumonia in the North and Baltic Seas were associated with lungworm infections. In contrast, in Amazon River dolphins (*Inia geoffrensis*) and Tucuxi (*Sotalia fluviatilis*), over half of the examined animals had pneumonia, with lung parasites and bacteria implicated in only 25% of cases of each (Rodrigues et al., 2018).

Despite effective control of parasitism in the managed population, pneumonia remains a frequent postmortem finding among cetaceans under human care. In long-term managed bottlenose dolphins, pneumonia was diagnosed in 50% of the necropsied animals. However, the primary pathogens were bacterial (42.9%) and fungal (28.6%), with no recorded lungworm infections (Venn-Watson et al., 2012). These findings indicate that although preventive health management and routine anthelmintic prophylaxis effectively eliminate lungworms, they do not remove the overall risk of respiratory disease. Instead, these management practices only shift the etiologic profile toward bacterial and fungal pathogens (Meegan et al., 2012; Venn-Watson et al., 2012).

In wild cetaceans, parasitic lung damage can predispose hosts to secondary bacterial infections, compounding tissue destruction and severe suppurative pneumonia or fatal septicaemia (Fischbach & Seguel, 2023; Giorda et al., 2017; Measures, 2001; Siebert et al., 2001). In the current study, three animals (4.8% of pneumonia cases) displayed mixed parasitic and bacterial pneumonia. This low rate of co-infection indicates that while secondary bacterial infection occurs in New Zealand common dolphins, it is less common than in other, more polluted Baltic and Black seas (Birkun Jr, 2002; Siebert et al., 2020). This finding emphasizes the importance of incorporating routine microbiological diagnostics into necropsy protocols to clarify proximate causes of disease and distinguish primary parasitic pathology from secondary bacterial involvement.

The prevalence and severity of parasitic pneumonia reported across published studies vary because host, parasite, environmental, and methodological factors interact to determine exposure, susceptibility, and clinical outcomes. Host factors such as species and age influence

disease expression, while parasite biology – particularly differences in pathogenicity and life-cycle dynamics – means that some host-parasite combinations may result in more severe disease outcomes (Measures, 2001; Pool et al., 2024). Ecological context and prey communities affect exposure risk, and seasonal and anthropogenic stressors (e.g., organochlorine pollution, prey depletion) can further affect host resistance (Pool et al., 2021; Saldaña et al., 2022). Furthermore, methodological differences between studies, such as sampling (e.g., stranded only vs stranded and bycaught), diagnostic effort, and sample size, can also result in apparent heterogeneity. For example, studies in Guiana dolphins from three different regions in Brazil reported a very high infection rate with *H. brasiliensis,* ranging from 88% (15/17) to 100% (7/7), with 40% of the infections associated with pneumonia (Demarque et al., 2020; Marigo et al., 2010). In contrast, a study in the North Sea reported only 59% (36/61) of the examined stranded harbour porpoises were infected with pseudaliid nematodes, but a higher proportion of the infected animals (52%, 19/36) displayed pneumonia (van Elk et al., 2019).

Although lungworm infection was graded in only 20.6% of dolphins (Section 4.3), 25 of these cases were diagnosed from characteristic histological lesions in which no parasite was seen (Section 2.4), so lungworm prevalence as graded may underestimate parasite-associated lung disease.

4.3 Lung Parasitism

The lungworm prevalence (20.6%) observed in the current study lies at the lower end of the global range. For example, some coastal cetacean populations show very high prevalence (60-100%) in parts of Brazil, Germany, and Florida (Demarque et al., 2020; Fauquier et al., 2009; Marigo et al., 2010; Siebert et al., 2001). In contrast, other regional surveys documented lower or comparable rates, such as 12% in common bottlenose dolphins and 26% in common dolphins reported in South Australia (Tomo et al., 2010), as well as a 25% prevalence in Amazon River dolphins (Rodrigues et al., 2018). A multi-region study of harbour porpoises in the Baltic Sea reported intermediate values ranging from 25-58% (Siebert et al., 2020).

The distribution of parasitism severity in the present study, mostly mild (10.6%) and moderate (6.4%) infections, with severe cases comprising only 3.5% of the total sample, is consistent with earlier findings in New Zealand common dolphins (Stockin et al., 2009) and with the broader pattern identified in other healthy cetacean populations, where lungworm burdens are frequently incidental findings (Lair et al., 2015; McFee & Lipscomb, 2009). This distribution also reflects contrasts between cetacean populations inhabiting pristine versus polluted environments. For example, Greenlandic harbour porpoises exhibited high lungworm prevalence (85%), but all were mild infections. In contrast, their German North Sea counterpart, which is exposed to more environmental pollution, shows a higher proportion of moderate infections (Wunschmann et al., 2001). Such variation among studies likely reflects differences in parasite species involved, host and age structure, trophic ecology, and exposure to infected fish hosts, local environmental and anthropogenic stressors, and methodological differences in detection (e.g., gross examination vs. combined gross and histological evaluation) (Fauquier et al., 2009; Measures, 2001; Pool et al., 2021).

The pulmonary parasites identified in this study – *H. lagenorhynchi, S. ovatus* and *Halocercus* sp. – are pseudaliid parasites known to occur in multiple cetacean hosts, including common dolphins (Measures, 2001; Saldaña et al., 2022; Sweeney & Ridgway, 1975; Tomo et al., 2010). These nematodes typically inhabit the pulmonary parenchyma, bronchioles, and bronchi (Measures, 2001; Sweeney & Ridgway, 1975)

Sex and Age Class

No statistically significant association between lungworm infection and sex was identified, a finding consistent with other studies (Rodrigues et al., 2018; Saldaña et al., 2022). For example, surveys of harbour porpoises in the Mediterranean and Black Sea (Birkun Jr, 2002), harbour porpoises and beluga in the Gulf of St. Lawrence and St. Lawrence Estuary, respectively, in the North Atlantic Ocean (Faulkner et al., 1998; Lair et al., 2015), and a multi-species survey of dolphins in northwest Spain (Saldaña et al., 2022), reported no difference in the parasite prevalence and severity between males and females.

In contrast, lungworm infection and burden are strongly associated with age class ($p < 0.001$). Specifically, perinates showed substantially higher parasite prevalence and greater proportions of moderate and severe infections than the immature and sexually mature animals, while sexually mature animals were predominantly parasite-free. This age-class pattern is consistent with the earlier findings reported by Stockin et al. (2009) for common dolphins in New Zealand, as well as studies on Indo-Pacific bottlenose dolphins, common bottlenose dolphins, harbour porpoises, and Dall's porpoises (*Phocoenoides dalli*) (Conlogue et al., 1985; Fauquier et al., 2009; Measures, 2001; Tomo et al., 2010). These findings corroborate the conclusion that lungworm burden in cetaceans is strongly age-dependent and likely linked to susceptibility and exposure.

Foetuses and neonates are significantly more susceptible to pathogens because their immune system is functionally immature and lacks prior exposure to antigens (Basha et al., 2014; Colegrove et al., 2016; Measures, 2001). Although foetuses and neonates can mount immune responses, these are generally weaker and slower than adult responses and provide limited immunological memory, making primary infections more likely to establish and progress (Basha et al., 2014; Cortese, 2009). Empirical data from both wild and captive animals show higher morbidity and mortality from infectious diseases among the youngest age classes. For example, the mortalities of beluga whales in the St. Lawrence Estuary were 72% among juveniles and only 29% among adults (Lair et al., 2015).

The high prevalence of *Halocercus* sp. infections in younger individuals in this study suggests that vertical transmission (transplacental, transmammary, or both) is likely (Measures, 2001). Dailey et al. (1991) reported *H. lagenorhynchi* in bottlenose dolphin calves aged newborn to three weeks, while Pool et al. (2021) documented *H. delphini* and *Stenurus globicephalae* in neonate striped and Risso's dolphins, respectively. Perhaps the most convincing evidence is the study by Fauquier et al. (2009) that described histological evidence of lungworm infection in a stillborn bottlenose dolphin, for which transplacental transmission is the only plausible route.

Body Condition Score

Contrary to the expectation that animals in poorer body condition would carry heavier parasite burdens, dolphins in good body condition (BCS 1) displayed the highest prevalence and severity of lungworm infection. Those with moderate or poor condition (BCS 2–3) showed comparatively lower burdens, although this pattern was not statistically significant ($p = 0.842$). This finding is consistent with several studies that have shown robust, free-ranging cetaceans frequently carry heavy lungworm burdens without obvious loss of condition (Cowan, 1966; Faulkner et al., 1998; Measures, 2001). These findings indicate that the presence of parasites alone does not equate to emaciation or impaired feeding (Faulkner et al., 1998; Measures, 2001; Tomo et al., 2010).

Several plausible explanations exist to explain this seemingly contradictory phenomenon. First, pseudaliid lungworms exhibit heteroxenous life cycles and use fish as intermediate hosts (Fischbach & Seguel, 2023; Hansen et al., 2017). This means that actively foraging animals are more exposed, and thus more likely to acquire the parasite post-weaning (Lehnert et al., 2010; Measures, 2001; Siebert et al., 2020). Second, hosts also commonly tolerate significant parasite burdens without immediate energy compromise, and lungworms often cause severe disease only when they lead to secondary bacterial infections, cardiovascular complications, or hypersensitivity reactions (Cowan, 1968; Fahlman et al., 2017; Measures, 2001). Lungworm infections typically progress to life-threatening disease only when larval migration or bronchial occlusion causes secondary bacterial infections, severe cardiovascular complications (e.g. vasculitis or thrombosis), or other systemic stressors (Fischbach & Seguel, 2023; Giorda et al., 2017; Neimanis et al., 2022; Siebert et al., 2001; van Elk et al., 2019). These lines of evidence suggest that lungworms are unlikely to be the principal driver of poor body condition in New Zealand common dolphins, although they can contribute to morbidity or mortality in specific cases. This is particularly pertinent when parasitism is accompanied by secondary infections or other stressors (Giorda et al., 2017; Mazzariol et al., 2007; van Elk et al., 2019).

Animal Source

Animal source did not significantly affect parasitism severity, and the pattern did not follow the expected gradient. Bycaught dolphins are actively foraging animals and might be expected to carry lighter lungworm burdens than animals that stranded, yet infection was proportionally most frequent in bycaught dolphins. All of these infections were mild. This is compatible with lungworm infection being common and often tolerated in otherwise healthy dolphins (Geraci & St. Aubin, 1987; Ijsseldijk et al., 2021; McFee & Lipscomb, 2009). In contrast, moderate and severe infections were found only in stranded animals, which fits the idea that heavy burdens are associated with debilitation and stranding (Baker & Martin, 1992; Guimarães et al., 2015; Marigo et al., 2010), although the numbers are too small to explore this hypothesis. However, only 14 bycaught dolphins were examined and the bycaught result rests on just four infected animals, so a few animals could change these proportions considerably and the power to detect differences was limited. Overall parasite prevalence should therefore be interpreted cautiously, as sampling bias may influence apparent population-level patterns (Section 4.5).

4.4 Summary

This thesis assessed lung health in common dolphins by examining the prevalence and aetiology of pneumonia, the prevalence of lung parasitism and evidence of irreparable tissue damage. Using gross findings, histopathology, and parasitology data in the context of sex, age class, body condition and animal source, I assessed which factors were associated with lung health. The prevalence of lungs without any evidence of parasitism, inflammation, or irreparable tissue damage provides important epidemiological baseline data for the common dolphin population in New Zealand. Pulmonary lesions identified were mostly mild and unlikely to have caused clinical signs of disease. While no association was found between lung health and sex, body condition or animal source, lung health was significantly associated with age class. The perinates were the most affected, while immature and sexually mature animals had similar and generally good lung health. This profile suggests that the perinates are more susceptible to acute and severe pulmonary diseases due to physiological immaturity, and that lung health after weaning is similar across age classes.

Approximately half of the dolphins had pneumonia, and most cases were mild. Parasitism was the most frequently identified aetiology (70% of pneumonia cases), while mixed parasitic-bacterial infections were uncommon (4.8%). Differences between this and international studies likely reflect variation in host susceptibility, aetiology, ecological context, anthropogenic stressors, and sampling methodology.

The prevalence of lungworm infection in this study (20.6%) was comparable to that of common dolphins examined in wider Oceania (26%; Tomo et al., 2010), and the nematodes identified (*H. lagenorhynchi, S. ovatus,* and *Halocercus* sp.) were the expected pseudaliids. No association between lungworm infection and sex, body condition or animal source was evident in this study, consistent with other surveys and with pseudaliid transmission ecology. In contrast, age class was significantly associated with lungworm infection, with perinates showing a higher prevalence and a greater proportion of moderate and severe infections than immature and sexually mature animals. This age-related pattern likely reflects a combination of vertical transmission of parasites and the functional immaturity of the perinatal immune system, which increases susceptibility to primary infection and limits the capacity to control it.

4.5 Study Limitations

Undertaking epidemiological studies on wild animal populations is challenging because obtaining a representative sample is inherently difficult (Lair et al., 2015; Saavedra et al., 2017). This is particularly true for cetaceans that live underwater in the vast and largely inaccessible marine environment. As such, this thesis is limited to studying those that have stranded alive and died, were found beachcast or recovered as bycatch. The dataset was unevenly distributed across animal sources, with single strandings making up most of the animals examined. Because prior health status may differ among animal sources, the combined dataset may not represent the health of the wider population, and the small groups limited the power of the comparison among sources for both lung health and parasitism. The 30 mass stranded dolphins came from six events, and animals from the same event are not independent observations, as they may share exposure to pathogens and parasites. This may reduce the effective sample size of the comparison among animal sources. Although mass

stranded and bycaught animals typically include apparently healthy individuals (Karns et al., 2019; Mazzariol et al., 2018), the dataset remained subject to recovery bias. Further, despite efforts to collect carcasses nationwide, most samples were obtained from the North Island of New Zealand, where the vast majority of strandings occur. These biases may distort estimates of lung health grade, pneumonia prevalence, and lung parasitism and therefore, limit direct extrapolation to the entire wider common dolphin population in New Zealand.

Although the age distribution in my study mirrors that of the wider population, the dataset is nevertheless skewed toward younger animals. Young animals differ from older animals in maternal dependence, exposure to pathogens, and immune competence, and therefore carry different risks of parasitism and respiratory disease. Because of this demographic bias, the prevalence estimates observed in this study may not be representative of older and sexually mature members of the population. Additionally, body condition score was used as a proxy for overall health. However, the sample was dominated by animals with good BCS, with very few with poor BCS (7.6%, n=9). This skew towards supposedly “healthier” individuals may underestimate the prevalence or severity of lung pathologies in the population, as the “debilitated” animals are systematically excluded from the dataset. Furthermore, prevalence and severity of lung parasitism may also be underestimated because lungworms inhabiting the pulmonary vessels were not included in the severity grading.

Practical constraints also affected lesion classification. The distribution of consolidation (focal, multifocal or diffuse) was mostly classified from postmortem photographs rather than from direct, hands-on examination of the lungs, with necropsy descriptions taking precedence where they existed. Classification from photographs precludes palpation, which is particularly valuable for detecting consolidation in cetacean lungs. Photograph quality, lighting, angle and the views available may also have influenced classification. Congestion, atelectasis, or postmortem colour change can resemble consolidation, so the extent of consolidation may have been overestimated, and distribution scores may be inflated. The scoring also has design limitations. Pattern and extent are combined in a single distribution variable, and multifocal disease spans a wide range of lung involvement (>10% to ≤75%). Because the distribution

scores and grade bands differ from those of Schick et al. (2024), lung health scores here are not directly comparable with theirs.

Histological findings were taken from pathology reports and not from a standardised re-examination of the slides. Pneumonia severity was graded by the pathologist who examined each case, and no formal inter-observer agreement was assessed, so observer variability is inevitable. Lung samples were submitted with those from other organs and the number of slides per animal was not recorded, so the amount of tissue examined per animal is unknown. Because pneumonia was diagnosed histologically, gross changes in animals without histological pneumonia were not scored as consolidation.

Pneumonia was classified as verminous in 25 animals from histological lesions in which no parasite was identified. Granulomatous, eosinophilic inflammation and mineralisation are not specific to parasites, so some misclassification is possible. Lungworm identification was possible for only 9 animals, because specimens were collected opportunistically. Finally, few animals fell into the severe lung health grade (n=3), which limits the power of the statistical tests for that category.

4.6 Future Research

The interconnection of animal, environmental, and human health within the “One Health” framework highlights the value of epidemiological studies on wild animals. Such studies can reflect environmental health and signal risks that ultimately affect people. Cetaceans occupy high trophic positions and experience multiple environmental stressors (Bossart, 2010). Therefore, systematic necropsy surveillance documenting respiratory pathogens yields information relevant to animal, ecosystem, and human health. This study establishes an important baseline for lung health in common dolphins and, by extension, an indicator of the condition of the marine ecosystem in New Zealand. Embedding future work within the One Health framework will maximise the value of stranded and bycaught carcasses. It is recommended that necropsy findings be linked to environmental contaminants, pathogen surveillance, anthropogenic noise, and any evidence of exposure to hazards shared with humans (e.g., HAB toxins or zoonotic agents). Continued monitoring of dolphin lung health

becomes not only a conservation tool, but also a practical tool for early detection and risk assessment for the environment and humans.

To increase the utility of the baseline established here, the current standardised necropsy protocol should be expanded to include lung health scoring for all dolphin species recovered in New Zealand waters. Future focus should be to broaden molecular screening to include viruses known to cause cetacean respiratory disease and mass mortalities (e.g., CeMV and herpes virus) in addition to the existing bacterial, fungal, and parasitic panels. A parallel profiling of blubber contaminants (e.g., PCBs and heavy metals) would further help test the hypotheses that contaminants may influence the susceptibility of the animals to respiratory pathogens. Routine reporting of lung health scores, accompanied by a formal synthesis and analysis of results every two to three years, would allow timely detection of changes in prevalence or severity and enable a rapid investigation when an alarm is raised.

The limitations of this study point to specific improvements in data collection. Recording palpation findings at necropsy and photographing both lungs in standard views with a scale would allow consolidation to be classified more reliably than from photographs alone. Standardising lung sampling (predefined regions plus lesions) and recording the number of slides per animal would make the histology reproducible. Ancillary diagnostics such as microbiology and molecular detection of parasites and viruses would reduce the proportion of pneumonia that cannot be assigned an aetiology and would allow parasitic pneumonia without a visible parasite to be confirmed. Assessing inter-observer agreement among pathologists and validating the lung health score against cause of death, would test whether the grades reflect clinical significance. Scoring the pattern and extent of consolidation as separate variables would remove the ambiguity of combining them in one distribution score.

Although animal source was compared here, the small numbers of mass-stranded and bycaught dolphins limited the power of that comparison. Larger samples for these groups would allow more robust analysis and clarify whether observed differences reflect genuine biological variation or differences in carcass recovery pathways. Integrating fisheries observer data or live health assessments would further strengthen representativeness.

Lastly, the accuracy and spatial representation of surveillance depend on improving carcass recovery and diagnostic capacity nationally. Efforts to maximise retrieval (e.g., community engagement, fisheries partnership, rapid-response protocols) will greatly assist so that wider parts of New Zealand can be represented. Building regional capacity for necropsy, standardised sample collection, and short-term storage will improve the quality of data collected and reduce geographic bias. This will enable spatial analyses (e.g., North vs South Island or east vs west coast comparisons), trend detection, and improve risk assessment, resulting in a better response to the problem.

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Appendix

Table A.1. Sample details and lung health scores of the 122 common dolphins (*Delphinus delphis*) examined between 1999 and 2025 in New Zealand. Note: Animal source legend (S = single stranding, M = mass stranding, B = bycatch), Sex (M = male, F = female), Age class (S = sexually mature, I = immature, P = perinate), ND = no data.

Animal Code	Animal Source	DCC	BCS	Sex	Age	Age Class	Parasitism		Pneumonia					Tissue Damage				Lung Health Score	Other Observations			
							Severity	Score	Type	Severity	Distribution	Points	Score	Fibrosis	Mineralisation	Necrosis	Score		Oedema	Congestion	Haemorrhage	Emphysema
WS99-25Dd	S	2	ND	F	ND	S	none	0	n/a	none	n/a	0	**0**				**0**	**0**	X		X	
WS00-01Dd	S	2	ND	M	ND	S	none	0	n/a	none	n/a	0	**0**				**0**	**0**	X	X	X	
WS00-01DdA	S	2	ND	M	ND	S	none	0	n/a	none	n/a	0	**0**				**0**	**0**	X	X	X	
WS02-03Dd	S	2	2	F	8	S	none	0	non-specific	mild	focal	1	**1**	X			**1**	**2**	X	X		
WS02-04Dd	S	2	1	F	>8	S	none	0	n/a	none	n/a	0	**0**				**0**	**0**	X	X	X	
WS02-05Dd	S	2	1	F	0	I	moderate	2	Parasite	mild	multifocal	2	**2**				**0**	**4**	X	X		X
WS02-06Dd	S	3	1	F	0	P	moderate	2	Multifactorial	severe	diffuse	3	**9**				**0**	**11**	X	X		
WS02-07Dd	S	2	1	M	0	I	moderate	2	Parasite	severe	multifocal	2	**6**	X			**1**	**9**	X	X		
WS02-08Dd	S	2	ND	M	0	P	moderate	2	Parasite	mild	multifocal	2	**2**				**0**	**4**	X	X		
KS06-03Dd	S	3	2	F	0	P	severe	3	Parasite	severe	diffuse	3	**9**				**0**	**12**				
WS06-14Dd	S	3	1	F	3	I	none	0	non-specific	mild	diffuse	3	**3**	X	X		**2**	**5**	X	X		
WS06-15Dd	S	2	1	M	3	I	none	0	n/a	none	n/a	0	**0**				**0**	**0**				
KS08-09Dd	S	2	1	F	13	S	none	0	non-specific	mild	diffuse	3	**3**				**0**	**3**		X		
KS09-11Dd	S	2	1	F	3	I	none	0	non-specific	mild	diffuse	3	**3**				**0**	**3**		X		
KS09-13Dd	S	1	1	M	8	S	none	0	non-specific	mild	diffuse	3	**3**				**0**	**3**		X		
KS09-18Dd	S	2	1	F	11	S	none	0	non-specific	mild	diffuse	3	**3**				**0**	**3**		X	X	
KS10-01Dd	S	2	2	F	2	I	mild	1	Parasite	mild	diffuse	3	**3**				**0**	**4**	X	X		
KS10-16Dd	S	2	1	M	16	S	none	0	n/a	none	n/a	0	**0**				**0**	**0**		X		
KS10-29Dd	S	2	2	M	1	I	none	0	n/a	none	n/a	0	**0**				**0**	**0**		X		
KS11-08Dd	S	1-2	2	M	6	I	none	0	non-specific	mild	diffuse	3	**3**				**0**	**3**				

KS11-12Dd	B	3	2	M	6	I	none	0	Bacteria	mild	diffuse	3	**3**		**0**	**3**		X	
KS11-13Dd	S	2	3	M	17	S	none	0	non-specific	mild	diffuse	3	**3**		**0**	**3**			X
KS11-14Dd	S	3	3	F	12	S	none	0	n/a	none	n/a	0	**0**		**0**	**0**		X	
KS11-27Dd	S	1	2	M	2	I	none	0	n/a	none	n/a	0	**0**		**0**	**0**		X	
KS11-39Dd	S	2	2	F	2	I	none	0	n/a	none	n/a	0	**0**		**0**	**0**	X	X	X
KS11-50Dd	S	2	2	F	≥14	S	none	0	n/a	none	n/a	0	**0**		**0**	**0**		X	
KS11-50ADd	S	2	1	F	0	P	none	0	non-specific	severe	diffuse	3	**9**		**0**	**9**			
KS12-13Dd	S	3	1	F	8	S	none	0	non-specific	severe	diffuse	3	**9**		**0**	**9**			
KS12-14Dd	S	2	2	M	11	I	none	0	n/a	none	n/a	0	**0**		**0**	**0**		X	
KS12-15Dd	S	2	1	F	≥20	S	none	0	non-specific	moderate	focal	1	**2**		**0**	**2**			X
KS12-17Dd	S	2	1	M	1.5	I	none	0	n/a	none	n/a	0	**0**		**0**	**0**		X	
KS12-18Dd	S	2-3	1	F	1	I	mild	1	n/a	none	n/a	0	**0**	X	**1**	**2**			
KS12-23Dd	S	2	1	M	2.5	I	none	0	non-specific	mild	focal	1	**1**		**0**	**1**			
KS13-02Dd	B	2	1	M	ND	P	none	0	n/a	none	n/a	0	**0**		**0**	**0**		X	X
KS13-07Dd	S	3	1	M	0.5	I	none	0	n/a	none	n/a	0	**0**		**0**	**0**		X	
KS13-09Dd	B	2	1	M	5.5	I	mild	1	n/a	none	n/a	0	**0**		**0**	**1**	X	X	
KS13-10Dd	S	3-4	1	F	18	S	none	0	non-specific	mild	diffuse	3	**3**		**0**	**3**		X	X
KS13-12Dd	S	3-4	1	M	3	I	none	0	n/a	none	n/a	0	**0**		**0**	**0**		X	
KS14-34Dd	S	3	2	F	11	S	none	0	n/a	none	n/a	0	**0**		**0**	**0**	X	X	
KS14-38Dd	S	2	1	M	16	S	none	0	non-specific	mild	multifocal	2	**2**		**0**	**2**	X	X	
KS14-39Dd	S	3	3	F	2	I	none	0	n/a	none	n/a	0	**0**		**0**	**0**	X	X	
KS14-42Dd	B	2	1	F	1	I	none	0	n/a	none	n/a	0	**0**		**0**	**0**			
KS14-62 Dd	S	2	1	F	ND	P	moderate	2	Parasite	moderate	diffuse	3	**6**		**0**	**8**			
KS14-63Dd	S	2-3	3	M	ND	P	none	0	n/a	none	n/a	0	**0**		**0**	**0**			
KS15-16Dd	S	3	2	M	26	S	none	0	n/a	none	n/a	0	**0**		**0**	**0**		X	
KS15-19Dd	S	2	1	F	11	S	none	0	non-specific	moderate	diffuse	3	**6**		**0**	**6**		X	
KS15-20Dd	B	2	1	F	0.5	I	none	0	n/a	none	n/a	0	**0**	X	**1**	**1**			
KS15-21Dd	S	2	2	F	4	I	none	0	n/a	none	n/a	0	**0**		**0**	**0**		X	X
KS16-06Dd	S	2-3	1	F	0.5	I	none	0	n/a	none	n/a	0	**0**		**0**	**0**			
KS17-01Dd	M	2	1	M	12	S	none	0	Bacteria	severe	diffuse	3	**9**		**0**	**9**		X	X

KS17-02Dd	M	2	1	M	11.5	S	none	0	n/a	none	n/a	0	**0**				**0**	**0**		X	X	
KS17-03Dd	M	2	1	M	16	S	none	0	n/a	none	n/a	0	**0**				**0**	**0**				
KS17-08Dd	S	2	1	M	4	I	none	0	n/a	none	n/a	0	**0**				**0**	**0**				
KS18-01Dd	S	3	2	M	≥11	S	none	0	n/a	none	n/a	0	**0**				**0**	**0**		X		
KS19-03Dd	S	2	1	F	0.5	I	none	0	n/a	none	n/a	0	**0**				**0**	**0**		X		
KS19-10Dd	M	2	1	F	7	I	none	0	non-specific	mild	multifocal	2	**2**	X			**1**	**3**				
KS19-11Dd	M	3	2	F	4	I	none	0	n/a	none	n/a	0	**0**				**0**	**0**		X		
KS19-12Dd	M	2-3	1	M	9	I	none	0	n/a	none	n/a	0	**0**				**0**	**0**		X		
KS19-13Dd	M	2-3	1	F	5.5	I	moderate	2	Parasite	mild	diffuse	3	**3**	X	X		**2**	**7**		X		
KS19-14Dd	M	3	1	F	10	S	none	0	n/a	none	n/a	0	**0**				**0**	**0**		X		
KS19-16Dd	S	2	1	F	9	I	none	0	n/a	none	n/a	0	**0**				**0**	**0**	X	X	X	
KS19-17Dd	M	2-3	1	F	12	S	none	0	n/a	none	n/a	0	**0**				**0**	**0**				
KS19-18Dd	M	2	1	M	0.25	I	moderate	2	Parasite	severe	diffuse	3	**9**		X	X	**2**	**13**				
KS19-19Dd	M	2	2	F	11	S	none	0	n/a	none	n/a	0	**0**				**0**	**0**		X		
KS19-20Dd	M	2	2	F	12	S	none	0	non-specific	moderate	diffuse	3	**6**				**0**	**6**				
KS19-21Dd	M	2-3	2	F	15	S	none	0	n/a	none	n/a	0	**0**				**0**	**0**		X		
KS19-22Dd	M	2	2	F	18	S	none	0	non-specific	mild	multifocal	2	**2**				**0**	**2**		X		
KS19-23Dd	M	2-3	2	F	ND	S	none	0	n/a	none	n/a	0	**0**				**0**	**0**		X		
KS19-26Dd	S	2-3	1	F	15	S	none	0	n/a	none	n/a	0	**0**				**0**	**0**	X	X		
KS19-38Dd	B	2-3	1	F	0	P	none	0	n/a	none	n/a	0	**0**				**0**	**0**		X		
KS19-39Dd	S	2-3	3	F	8	S	none	0	n/a	none	n/a	0	**0**				**0**	**0**		X	X	
KS20-05Dd	S	3	1	M	9.5	S	none	0	non-specific	mild	diffuse	3	**3**				**0**	**3**				
KS20-30Dd	B	2-3	2	M	2	I	none	0	n/a	none	n/a	0	**0**				**0**	**0**		X		
KS21-31Dd	S	2	1	F	15	S	none	0	n/a	none	n/a	0	**0**				**0**	**0**	X	X		
KS21-41Dd	S	1-2	2	F	12	S	none	0	n/a	none	n/a	0	**0**				**0**	**0**	X	X		
KS21-42Dd	S	2-3	1	F	2	I	none	0	n/a	none	n/a	0	**0**				**0**	**0**		X		
KS22-03Dd	S	2	2	F	9	S	none	0	non-specific	mild	diffuse	3	**3**				**0**	**3**	X	X		
KS22-08Dd	S	2	1	F	2.25	I	mild	1	Parasite	moderate	diffuse	3	**6**	X	X		**2**	**9**				
KS22-48Dd	M	2	1	F	> 25	S	none	0	non-specific	mild	diffuse	3	**3**	X			**1**	**4**				X
KS22-49Dd	M	2	1	F	2	I	none	0	non-specific	mild	diffuse	3	**3**				**0**	**3**				

KS22-53Dd	S	2-3	3	M	1	I	moderate	2	Parasite	moderate	diffuse	3	**6**	X	X		**2**	**10**					
KS22-59Dd	S	2	3	M	1	I	none	0	n/a	none	n/a	0	**0**				**0**	**0**					
KS22-77Dd	S	2-3	2	M	0	P	moderate	2	Parasite	moderate	diffuse	3	**6**				**0**	**8**					
KS23-03Dd	S	3	2	F	1.5	I	none	0	non-specific	moderate	multifocal	2	**4**				**0**	**4**		X			
KS23-04Dd	S	2	1	F	0.5	I	severe	3	n/a	none	n/a	0	**0**				**0**	**3**					
KS23-05Dd	S	1	1	F	20	S	none	0	n/a	none	n/a	0	**0**	X			**1**	**1**		X			
KS23-06Dd	M	1-2	1	F	0.25	P	severe	3	Multifactorial	severe	multifocal	2	**6**				**0**	**9**		X			
KS23-07Dd	M	1	1	F	13	S	mild	1	Multifactorial	mild	diffuse	3	**3**				**0**	**4**		X			
KS23-08Dd	M	2	2	M	2.25	I	mild	1	Parasite	mild	diffuse	3	**3**	X	X		**2**	**6**	X	X	X		
KS23-09Dd	M	2	1	F	8	I	none	0	non-specific	moderate	multifocal	2	**4**				**0**	**4**		X			
KS23-10Dd	M	2	1	F	14	S	none	0	non-specific	moderate	diffuse	3	**6**				**0**	**6**					
KS23-11Dd	M	2	1	F	17	S	none	0	non-specific	moderate	diffuse	3	**6**				**0**	**6**					
KS23-12Dd	M	2	1	M	0	P	severe	3	Parasite	severe	multifocal	2	**6**				**0**	**9**					
KS23-13Dd	S	3	2	F	3	I	none	0	non-specific	mild	diffuse	3	**3**				**0**	**3**		X			X
KS23-42Dd	M	1	1	M	21	S	none	0	non-specific	mild	diffuse	3	**3**				**0**	**3**					
KS23-43Dd	M	1	1	M	16.5	S	none	0	non-specific	mild	diffuse	3	**3**				**0**	**3**	X		X		
KS23-44Dd	M	2	1	M	9.5	S	none	0	non-specific	mild	diffuse	3	**3**	X			**1**	**4**		X	X		
KS23-45Dd	M	2	1	M	31	S	none	0	non-specific	mild	diffuse	3	**3**				**0**	**3**		X	X		
KS23-46Dd	M	1	1	M	24	S	none	0	non-specific	mild	diffuse	3	**3**				**0**	**3**		X			
KS23-47Dd	M	2	1	M	11.5	S	none	0	non-specific	mild	diffuse	3	**3**	X			**1**	**4**		X			
KS23-50Dd	S	2	1	M	12	S	none	0	non-specific	mild	diffuse	3	**3**	X			**1**	**4**					
KS23-57Dd	S	2-3	1	M	4	I	mild	1	Parasite	moderate	multifocal	2	**4**	X	X	X	**3**	**8**					
KS23-58Dd	S	2-3	1	M	7	I	mild	1	Parasite	mild	multifocal	2	**2**				**0**	**3**					
KS24-48Dd	S	2-3	2	M	ND	P	none	0	Bacteria	severe	diffuse	3	**9**				**0**	**9**					
KS24-54Dd	S	3	2	M	0	P	mild	1	Parasite	severe	multifocal	2	**6**				**0**	**7**					
KS24-59Dd	S	3	3	F	10	I	none	0	n/a	none	n/a	0	**0**				**0**	**0**	X	X			
KS24-81Dd	S	2	2	M	17.5	S	none	0	Bacteria	severe	diffuse	3	**9**				**0**	**9**		X			
KS24-86Dd	S	2	1	M	2	I	none	0	non-specific	mild	diffuse	3	**3**				**0**	**3**	X	X	X		
KS24-88Dd	S	2	2	F	1.5	I	none	0	n/a	none	n/a	0	**0**				**0**	**0**					
KS24-96Dd	S	2	2	M	0	P	none	0	n/a	none	n/a	0	**0**				**0**	**0**		X			

KS24-97Dd	B	2-3	1	M	6	I	none	0	n/a	none	n/a	0	**0**			**0**	**0**		X	
KS25-07Dd	B	1	2	M	12	S	none	0	n/a	none	n/a	0	**0**			**0**	**0**	X	X	X
KS25-08Dd	B	1	2	M	11	S	mild	1	Parasite	mild	diffuse	3	**3**		X	**1**	**5**	X	X	X
KS25-09Dd	B	1-2	1	F	11.5	S	none	0	non-specific	moderate	diffuse	3	**6**			**0**	**6**		X	X
KS25-10Dd	B	1-2	1	F	7	I	none	0	n/a	none	n/a	0	**0**			**0**	**0**	X	X	X
KS25-11Dd	B	1	1	M	ND	P	mild	1	Parasite	moderate	multifocal	2	**4**			**0**	**5**			
KS25-61Dd	B	2-3	2	F	2.25	I	mild	1	Parasite	mild	multifocal	2	**2**		X	X	**2**	**5**			
KS25-67Dd	S	2	3	F	19	S	none	0	Bacteria	mild	diffuse	3	**3**			**0**	**3**			
KS25-79Dd	S	ND	1	M	ND	S	none	0	n/a	none	n/a	0	**0**			**0**	**0**		X	X
KS25-80Dd	S	ND	1	F	ND	S	none	0	n/a	none	n/a	0	**0**			**0**	**0**		X	X
KS25-80DdA	S	ND	1	M	0	P	none	0	n/a	none	n/a	0	**0**			**0**	**0**			
KS25-80DdB	S	ND	2	F	0	P	none	0	n/a	none	n/a	0	**0**			**0**	**0**			

Table A.2. Sample details and parasite severity of the 141 common dolphins (*Delphinus delphis*) examined between 1999 and 2025 in New Zealand. Note: Animal source legend (S = single stranding, M = mass stranding, B = bycatch), Sex (M = male, F = female), Age class (S = sexually mature, I = immature, P = perinate), ND = no data.

Animal Code	Animal Source	DCC	BCS	Sex	Age	Age Class	Parasite Severity
WS99-25Dd	S	2	ND	F	ND	S	none
WS00-01Dd	S	2	ND	M	ND	S	none
WS00-01Dd(a)	S	2	ND	M	ND	S	none
WS02-03Dd	S	2	2	F	8	S	none
WS02-04Dd	S	2	1	F	>8	S	none
WS02-05Dd	S	2	1	F	0	I	moderate
WS02-06Dd	S	3	1	F	0	P	moderate
WS02-07Dd	S	2	1	M	0	I	moderate
WS02-08Dd	S	2	ND	M	0	P	moderate
KS06-03Dd	S	3	2	F	0	P	severe
WS06-14Dd	S	3	1	F	3	I	none
WS06-15Dd	S	2	1	M	3	I	none
KS07-12DdA	S	4	ND	M	0	P	none
KS08-08Dd	S	2	2	M	1	I	none
KS08-09Dd	S	2	1	F	13	S	none
KS08-11Dd	S	4	1	M	≥11	S	none
KS09-11Dd	S	2	1	F	3	I	none
KS09-13Dd	S	1	1	M	8	S	none
KS09-14Dd	S	3	2	F	18	S	none
KS09-18Dd	S	2	1	F	11	S	none
KS09-19Dd	S	4	1	F	7	I	none
KS10-01Dd	S	2	2	F	2	I	mild
KS10-16Dd	S	2	1	M	16	S	none
KS10-26Dd	S	3-4	1	M	9.5	S	mild
KS10-27Dd	S	3	3	M	4	I	mild
KS10-29Dd	S	2	2	M	1	I	none
KS11-08Dd	S	1-2	2	M	6	I	none
KS11-12Dd	B	3	2	M	6	I	none
KS11-13Dd	S	2	3	M	17	S	none
KS11-14Dd	S	3	3	F	12	S	none
KS11-27Dd	S	1	2	M	2	I	none
KS11-39Dd	S	2	2	F	2	I	none
KS11-50Dd	S	2	2	F	≥14	S	none
KS11-50ADd	S	2	1	F	0	P	none
KS11-52Dd	S	3-4	2	M	8	I	none
KS12-13Dd	S	3	1	F	8	S	none
KS12-14Dd	S	2	2	M	11	I	none
KS12-15Dd	S	2	1	F	≥20	S	none
KS12-17Dd	S	2	1	M	1.5	I	none
KS12-18Dd	S	2-3	1	F	1	I	mild

KS12-23Dd	S	2	1	M	2.5	I	none
KS13-02Dd	B	2	1	M	ND	P	none
KS13-07Dd	S	3	1	M	0.5	I	none
KS13-08Dd	S	3-4	1	M	2.5	I	none
KS13-09Dd	B	2	1	M	5.5	I	mild
KS13-10Dd	S	3-4	1	F	18	S	none
KS13-12Dd	S	3-4	1	M	3	I	none
KS14-34Dd	S	3	2	F	11	S	none
KS14-38Dd	S	2	1	M	16	S	none
KS14-39Dd	S	3	3	F	2	I	none
KS14-42Dd	B	2	1	F	1	I	none
KS14-62 Dd	S	2	1	F	ND	P	moderate
KS14-63Dd	S	2-3	3	M	ND	P	none
KS15-16Dd	S	3	2	M	26	S	none
KS15-19Dd	S	2	1	F	11	S	none
KS15-20Dd	B	2	1	F	0.5	I	none
KS15-21Dd	S	2	2	F	4	I	none
KS16-06Dd	S	2-3	1	F	0.5	I	none
KS17-01Dd	M	2	1	M	12	S	none
KS17-02Dd	M	2	1	M	11.5	S	none
KS17-03Dd	M	2	1	M	16	S	none
KS17-08Dd	S	2	1	M	4	I	none
KS18-01Dd	S	3	2	M	≥11	S	none
KS19-03Dd	S	2	1	F	0.5	I	none
KS19-10Dd	M	2	1	F	7	I	none
KS19-11Dd	M	3	2	F	4	I	none
KS19-12Dd	M	2-3	1	M	9	I	none
KS19-13Dd	M	2-3	1	F	5.5	I	moderate
KS19-14Dd	M	3	1	F	10	S	none
KS19-16Dd	S	2	1	F	9	I	none
KS19-17Dd	M	2-3	1	F	12	S	none
KS19-18Dd	M	2	1	M	0.25	I	moderate
KS19-19Dd	M	2	2	F	11	S	none
KS19-20Dd	M	2	2	F	12	S	none
KS19-21Dd	M	2-3	2	F	15	S	none
KS19-22Dd	M	2	2	F	18	S	none
KS19-23Dd	M	2-3	2	F	ND	S	none
KS19-26Dd	S	2-3	1	F	15	S	none
KS19-38Dd	B	2-3	1	F	0	P	none
KS19-39Dd	S	2-3	3	F	8	S	none
KS20-05Dd	S	3	1	M	9.5	S	none
KS20-30Dd	B	2-3	2	M	2	I	none
KS21-01Dd	S	3	2	F	12	S	none
KS21-01ADd	S	3	2	M	0	P	none
KS21-31Dd	S	2	1	F	15	S	none
KS21-41Dd	S	1-2	2	F	12	S	none
KS21-42Dd	S	2-3	1	F	2	I	none

KS22-03Dd	S	2	2	F	9	S	none
KS22-07Dd	S	3-4	3	F	2	I	none
KS22-08Dd	S	2	1	F	2.25	I	mild
KS22-48Dd	M	2	1	F	> 25	S	none
KS22-49Dd	M	2	1	F	2	I	none
KS22-50Dd	S	3	2	M	0	P	none
KS22-52Dd	S	3	1	M	11.5	S	mild
KS22-53Dd	S	2-3	3	M	1	I	moderate
KS22-59Dd	S	2	3	M	1	I	none
KS22-73Dd	S	3	2	M	ND	P	none
KS22-77Dd	S	2-3	2	M	0	P	moderate
KS23-03Dd	S	3	2	F	1.5	I	none
KS23-04Dd	S	2	1	F	0.5	I	severe
KS23-05Dd	S	1	1	F	20	S	none
KS23-06Dd	M	1-2	1	F	0.25	P	severe
KS23-07Dd	M	1	1	F	13	S	mild
KS23-08Dd	M	2	2	M	2.25	I	mild
KS23-09Dd	M	2	1	F	8	I	none
KS23-10Dd	M	2	1	F	14	S	none
KS23-11Dd	M	2	1	F	17	S	none
KS23-12Dd	M	2	1	M	0	P	severe
KS23-13Dd	S	3	2	F	3	I	none
KS23-42Dd	M	1	1	M	21	S	none
KS23-43Dd	M	1	1	M	16.5	S	none
KS23-44Dd	M	2	1	M	9.5	S	none
KS23-45Dd	M	2	1	M	31	S	none
KS23-46Dd	M	1	1	M	24	S	none
KS23-47Dd	M	2	1	M	11.5	S	none
KS23-50Dd	S	2	1	M	12	S	none
KS23-54Dd	S	3	1	F	0.5	I	severe
KS23-57Dd	S	2-3	1	M	4	I	mild
KS23-58Dd	S	2-3	1	M	7	I	mild
KS24-48Dd	S	2-3	2	M	ND	P	none
KS24-54Dd	S	3	2	M	0	P	mild
KS24-59Dd	S	3	3	F	10	I	none
KS24-68Dd	S	3	2	F	ND	P	none
KS24-81Dd	S	2	2	M	17.5	S	none
KS24-86Dd	S	2	1	M	2	I	none
KS24-88Dd	S	2	2	F	1.5	I	none
KS24-96Dd	S	2	2	M	0	P	none
KS24-97Dd	B	2-3	1	M	6	I	none
KS25-07Dd	B	1	2	M	12	S	none
KS25-08Dd	B	1	2	M	11	S	mild
KS25-09Dd	B	1-2	1	F	11.5	S	none
KS25-10Dd	B	1-2	1	F	7	I	none
KS25-11Dd	B	1	1	M	ND	P	mild
KS25-61Dd	B	2-3	2	F	2.25	I	mild

KS25-62Dd	S	3	2	M	ND	I	none
KS25-67Dd	S	2	3	F	19	S	none
KS25-68Dd	S	3	3	F	≥18	S	none
KS25-79Dd	S	ND	1	M	ND	S	none
KS25-80Dd	S	ND	1	F	ND	S	none
KS25-80DdA	S	ND	1	M	0	P	none
KS25-80DdB	S	ND	2	F	0	P	none

Table A.3. Histopathological findings recorded for each animal in the pathology reports, with how each was recorded and how it was used in this study.

Finding	Recorded as	Source	Use
Lungworm infection	None, mild, moderate or severe (Table 2.2)	Necropsy record and pathology report	Scored
Pneumonia type	Parasitic, bacterial, multifactorial, non-specific	Pathology report	Descriptive
Pneumonia severity	Mild, moderate, severe (Table 2.3)	Pathology report	Scored
Pneumonia distribution	Focal, multifocal, diffuse (Table 2.4)	Necropsy photographs and report	Scored
Fibrosis, mineralisation, necrosis	Present or absent	Pathology report	Scored
Oedema, congestion, haemorrhage, emphysema	Present or absent	Necropsy record and pathology report	Recorded, not scored